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DISEASES

OF THE

NERVOUS SYSTEM

A TEXT-BOOK OF

NEUROLOGY AND PSYCHIATRY

BY

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FIFTH EDITION, REVISED, REWRITTEN AND ENLARGED

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TO

HELENA LEEMING JELLIFFE

WHOSE LOFTY PURPOSE,

IDEAL STRIVING, AND NEVER-FAILING COÖPERATION,

HAVE BEEN A CONSTANT STIMULUS TO PROGRESSIVE ENDEAVOR,

THIS BOOK IS DEDICATED

AS A TOKEN OF LOVE AND ESTEEM

PREFACE TO THE FIFTH EDITION.

It is highly gratifying to the authors that their effort in the production of this Textbook has met with such a generous response as to justify a new edition. For this approval we here express our sincere thanks to our colleagues who have found it useful in their teaching.

The present edition has been rigorously revised. Hardly a chapter remains entirely as it was in the previous edition since newer researches have made it necessary to reflect the advances in knowledge of the structure and disorders of the nervous system, at all of its various levels of integration.

In this revision the more recent outlooks concerning the vegetative nervous system have received special attention, likewise the progress made in the endocrine disorders.

The concept of the nervous system as a coördinator of experience with the outside world is still further advanced. The dissolution of function that results from conflict with extraneous energy systems, be they traumatic, bacterial, toxic, or psychological are carefully reviewed and their results simplified and made available for the student, the practitioner, and even, may we hope, for the specialist. No effort has been spared to make the work simple, complete, and adequate to meet the needs of these three classes of students.

In the past few years a veritable flood of research material has appeared in the periodical literature—this has received the authors' earnest consideration and it is hoped that the present work adequately and judiciously portrays the front line advances of this knowledge.

If it may seem that the authors have inclined a bit toward a radical approval of these newer researches, both in the neurological and psychiatric fields, it is because we have felt justified in the belief that a healthy optimism is to be preferred to a studied conservatism, holding that the future contains infinitely more promise than the past. Hence a certain preferance for things hoped for, rather than for a repetition of that which is already proven.

The accent therefore in this, as in previous editions, is toward a scientific curiosity in solving riddles heretofore insoluble. We have preferred to tell the student, the practitioner, and the specialist as well, what little is definitely known, and how great is the territory still to be explored and conquered, rather than to be satisfied with a simple recital of the static, the known, and the dead past.

In such a spirit of portrayal of the problems of the diseases of the nervous system we offer this present edition to the toast—"to the future."

S. E. J.
W. A. W.

New York and Washington.

PREFACE TO THE FIRST EDITION.

THE diseases of the nervous system are no longer compassed by a description of the gross lesions of the brain, spinal cord, cranial and peripheral nerves. The more limited symptomatology of disorders of these structures, which in this work has been called sensori-motor neurology, has been expanded in two directions—in one by the increase in our knowledge of the historically oldest portion of the nervous system, namely, the sympathetic and autonomic (vegetative) nervous system and in the other by the increase in our knowledge of the mechanisms that operate at the psychic or mental levels.

The vegetative nervous system is in close functional relations with the endocrinous glands, and, although some of the endocrinopathies may ultimately turn out not to be exclusively nervous affections, still these organs of internal secretion are so closely related from all points of view, embryological, anatomical, physiological, pathological, and pharmacodynamic, with the vegetative nervous system that their disordered functions must needs be considered in a work dealing with the diseases of the nervous system. The symptomatology of this region constitutes a borderland of neurology and internal medicine.

At the highest level stand the mental mechanisms in which action receives a symbolic representation. Here the nervous system is also the medium through which that form of physiological or pathological activity called conduct is brought about. These mechanisms, while operating consciously, largely through the sensori-motor channels of adjustment, are also intimately related to the vegetative levels where through the emotions they act unconsciously.

The authors have kept in mind the concept of the individual as a biological unit tending by development and conduct toward certain broadly defined goals and have considered the nervous system as only a part of that larger whole. The part, however, partakes of the unity of the whole and, so far as possible, the attempt has been made to arrange the diseases of the nervous system in accord with this evolutionary concept.

For practical purposes and for the reasons stated the work has therefore been divided into three parts dealing respectively with the vegetative, the sensori-motor, and the psychic levels, the reactions in all of which come to pass through the medium of the nervous system.

Man is not only a metabolic apparatus, accurately adjusted to a marvelous efficiency through the intricacies of the vegetative neurological mechanisms, nor do his sensori-motor functions make him solely a feeling, moving animal, seeking pleasure and avoiding pain, conquering time and space by the enhancement of his sensory possibilities and the magnification of his motor powers; nor yet is he exclusively a psychical machine, which by means of a masterly symbolic handling of the vast horde of realities about him has given him almost unlimited powers. He is all three, and a neurology of today that fails to interpret nervous disturbances in terms of all three of these levels, takes too narrow a view of the function of that master spirit in evolution, the nervous system.

For these reasons the treatise has been called primarily a work on the diseases of the nervous system rather than two books, one on neurology and one on psychiatry, which would perpetuate a distinction which the authors believe to be wholly artificial.

S. E. J.
W. A. W.

New York and Washington, 1915.

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DISEASES OF THE NERVOUS SYSTEM.

INTRODUCTION.

ON SOME PRINCIPLES UNDERLYING DISEASE OF THE BODY AND MODIFICATIONS OF HUMAN BEHAVIOR.

CERTAIN fundamental considerations are attempted in this book. It is not only a work on diseases of the nervous system but it would offer a general viewpoint from which to regard all types of disease, even though it makes its main object to discuss such modification of function of certain organs as are comprised within the very broad confines of neuropsychiatry.

The nervous system is the organizer of all experience, racial as well as individual, and is the coördinator of that experience. Through it the human animal is put in touch with the entire past of the race and by it can make that past of value for each moment of living. The future can only be reached through that racial accumulation of experience.

The human being is here regarded in the light of an open energy system, by which energy is captured, transformed and delivered. Neither as a whole, nor in its parts does it make energy, it simply captures, transforms and delivers it.

The energy comes from the cosmos. It is caught by specific energy receptors (Johannes Müller), located upon the surface of and throughout the interior of the body. These specific energy receptors are numerous. The "five senses" is a naïve concept of the truth, which has its place in the history of the development of our present ideas, and the chimerical sixth sense is but a little better notion of the marvellous reality concerning the multitude of receptors or other types of apparatus through which and by means of which the human organism captures energy from the environment.

The histological details of the receptor structures are being amplified daily and the means by which the transformations and deliveries take place are also slowly delivering up their structural as well as dynamic secrets.

Since life appeared on the face of the earth (estimated as being from one thousand to one hundred million years), it has unceasingly increased its capacity to continue itself through the mounting complexities by which it has captured, transformed and delivered the available energy.

As this work is not a histology it cannot go into all of the details of the structures serving as receptors, connectors and effectors.[1] Likewise it must leave to anatomy the details of the aggregates of these structuralized functions, the organs, only lightly touching upon such intimate details of the connecting mechanisms of the nervous system as are needed for the general formulations here advanced.[2]

As one surveys the progress of life with a biological vision two large fundamental functions become manifest. They are the preservation of the individual and the maintenance of the race. Instead of saying that self-preservation is Nature's first law the viewpoint here formulated would emphasize the fact that the two functions are correlates, and in a sense opposites. The mystery of their opposing aspects has been the subject of much philosophical speculation from the days of Empedocles to the present time. Even in possibly the earlier days of Indo-Germanic beginnings is found this dual aspect of the individual and the race, and even more interestingly the warning lest the polarity be lost and the inertia of death attained by an equalization of the opposing forces. "Beware of being caught in the pathway of opposites" purports to be a quotation of Hindu antiquity, which in modern mechanics means "being caught on dead center." The formulations followed in this volume assume that whatever is called "Nature" has provided for this contingency and has accumulated such racial memories (Semon's engrammes) as to push forward with greater dynamic potential one of these pairs of opposites, namely the racial maintenance. Hence *race propagation* is the loaded side of life's revolving wheel, and *self-preservation* its obedient opposite.

The biological program that made evolution possible was that of reproduction by a sex mechanism rather than of self-perpetuation by the non-sexual mechanism of fission. Hence it may be stated that the oldest biological pattern that successfully maintained and evolved the phylum is the sex pattern. It has been incorporated into every activity of the animal organism for at least a billion years.

It must be very strongly emphasized that these statements apply to the instinctive, unconscious and automatic mechanisms. The widely prevalent, shallow and unintelligent idea of sex as the equivalent of genital activity is not here under consideration. The biological conception of sex is parallelled by the theologian's God. Furthermore it is not altogether a coincidence that the theological formulation of the Devil should so closely conform to this same shallow unintelligent attitude toward Love.

The formulation in psycho-biological terms then states that any deviation from the inner, or instinctive action, or good, or God-like-

[1] See Kappers, C. A.: Die vergleichende Anatomie des Nervensystems der Wirbelthiere und des Menschen, 1920–1922; for details of the anatomical structures which make this capture, transformation and delivery possible, also Herrick, C. J., Introduction to Neurology.

[2] Consult Winkler C.: Manual de Neurologie, 1919–1922, 1927, for a detailed consideration of the human nervous system from this energic point of view.

sex-pattern—will result in faulty adaptation, ergo "disease." In what sense then is the inner, the instinctive, the unconscious pattern to be understood? Is it different from the outer, the habitual, the conscious pattern? Here again one meets with another possible series of opposites, the reconciliation of which, it is here assumed is the chief function of the nervous system, for health and happiness by adaptation to the environment. To choose the good and flee the Devil is the theological formula, which unfortunately, however, has so many dogmatic statements about what is Good and what is the Devil that the true Delphic formula "Know Thyself" is lost in the confusion.

It is then toward a more psychological conception that medicine must look if its understanding of dynamic processes is to better founded, and the mysteries of health and disease, not alone of the nervous system, but of all of the bodily organs, laid bare. When Claude Bernard wrote, "some time, he hoped, the day would arrive when the physiologist, the philosopher, and the poet would talk the same language and understand each other. Then a dynamic medicine would arise."[1] Then the descriptive and static concepts of structural alterations would be infused with a new life and the *how* and the *why* be added to the purely descriptive *what.*

For a long time it has been asserted that the nervous system is the means by which all of the several parts of the human unit are integrated by a species of complicated adjustments to given ends. It can be perceived how this integration is actually brought to pass by means of the vegetative nervous system and the chemical regulators of metabolism, at a physicochemical level, and how by the successive compounding of reflexes at the sensorimotor level, the human unit is further integrated, so that it can as a whole work more consistently toward broadly defined goals, the integration manifesting itself at successively higher and higher levels in the history of the individual and of the species.

Viewed in this way the individual is seen struggling along the path of evolution in constant conflict with an inherent inertia that would keep it at a given level, but gradually advancing by a series of give-and-take compromises that finally bring it to better adjustment with its environment at ever higher levels of integration.

Sherrington has beautifully illustrated this integrative action of the nervous system in the simple reflex with its innervation of agonists on the one hand and antagonists on the other, and the channelling of final common pathways for nervous discharge. This law of conflicting tendencies, pathways of opposites—ambivalence—where the final issue for higher integrations is made possible at the sensorimotor level by the tension of reciprocal innervations, is found also to be the rule in the vegetative nervous system, with its double set of pharmacodynamically demonstrated balancing elements, mediated, at least in

[1] See Kraus, F.: Die allgemeine and spezielle Pathologie der Person, Leipzig, 1919, vol. 1.

part, by equally opposed, stimulogenic chemical substances secreted by the endocrinous glands, the hormones.

Finally an analogous ambivalent mechanism is seen working at the highest, the most complex level, the psychic, which determines certain aspects of behavior with the assistance of the phenomena called consciousness, in which a psychological symbolism is found replacing sensory and motor neurons, and exciting and inhibiting hormones.

For practical purposes, then, the nervous system may be divided into these three levels of activity, the vegetative, the sensorimotor and the psychic.[1]

This threefold division of the reactions of the nervous system is the fundamental basis on which a classification may be founded. The biological processes which maintain life at the lowest level are physical and chemical, and thus that portion of the nervous system which has direct controlling influences over these activities is properly designated as the vegetative nervous system, and that part of neurology which has to do with a consideration of these physico-chemical systems because it deals with the nervous control of the viscera and of metabolism, is properly designated as vegetative neurology.

In this region of vegetative neurology a rich variety of disturbances is found, involving the glandular, gastro-intestinal, genito-urinary, vascular, respiratory, muscular, cutaneous and bony systems. In addition there are certain complex clinical groups involving, for the most part, the glands of internal secretion, the endocrinopathies. Here is a rich field which has been unequally cultivated since Brown-Séquard first tried to discover the fountain of youth in the testicular extracts.

While the symptomatology of the neurological disturbances of the tear, mucous and salivary glands is a comparatively limited one, a very rich symptomatology has grown up about the vascular system in the group of vasomotor neuroses. There are many strictly neurological problems among the gastro-intestinal and in the cutaneous disorders, which, however, are for the most part taken over by the specialties dealing with these respective systems, but nevertheless many of these disorders will receive an adequate explanation only through the understanding of visceral neurology. Some portions of the field are as yet too little known to offer much that is of value, as, for example, the neurology of the bony system and the nervous mechanisms underlying the regulation of the blood cells and the relations of the vegetative nervous system to immunity and anaphylaxis, while in other systems the disturbances are known only as contributing symptoms in fairly well-defined clinical groups, as, for example, myasthenia gravis as a disturbance of the muscle vegetative mechanisms.

The endocrinopathies naturally form a considerable part of visceral neurology, and many of the disturbances of the several systems are still best included in the various clinical groups that are considered

[1] These are not the levels of Hughlings Jackson, but the concept is the same.

as due to disturbances in one or more of the endocrine glands. This field today occupies the main focus of attention of the medical practitioner, and much that is false is being taught; but out of the divergent trends sound harmonies will be evolved.

If the vegetative nervous system has for its function, in the main, the maintenance of the vegetative, that is, the metabolic processes of life, such as nutrition, growth, development and involution, the next higher level, the sensorimotor, has as its function, in the main, further integration by providing the means for the balanced interrelations of the various motor organs of the body. It has to provide that all the various parts of the machine work harmoniously together, that the functions of the various organs are not only properly timed in relation, one to the others, but also adequately related on the basis of the functional demands made on them to carry out the necessary motor activities to satisfy the vegetative system needs.

This field of sensorimotor neurology, including the disorders of the cranial and peripheral nerves, the spinal cord, medulla, pons, brainstem, midbrain, cerebellum and cerebrum, is that portion of neurology to which the term "neurology" is generally thought of as applying, to all intents and purposes, exclusively. Here the field is pretty well cultivated, and classifications based on anatomical and on etiological criteria are fairly well organized.

The third, the highest, the psychic level is the most complex. Its function is no longer simply one of integration of the various parts of the individual but at its highest, conscious level it has to do not only with the relation of the individual as a whole to his environment but more especially to his social environment.

At this level it has been the prevalent custom to think only of consciousness, and of behavior consciously regulated by intelligence. Ideas are symbols; they are symbols of the contemplated action on things, through which the individual comes to an efficient adjustment with his environment by controlling them. The symbol therefore becomes a carrier of energy which is translated into conduct.

The ways in which these psychic symbolizations work at the highest conscious levels are pretty well formulated in current psychology, and these ways work very well so long as there is nothing unusual the matter with the whole machine. The great error of the academic psychologist, however, has been to suppose that the matter stopped here. The lower animals exhibit most complex forms of behavior without its being thought necessary to ascribe conscious motives (intelligence) to them in explanation. Very complicated activities low down in the biological scale are ascribed to tropisms, while for man it has been supposed that what he did he consciously intended. Recent studies in psychopathology have shown the inadequacy of this conception, and it is thoroughly well established that lying back of consciousness is a much larger, a much more important territory which furnishes a psychic motivation of conduct, and, in fact, that conscious processes

as they are known to the individual are largely, if not altogether determined by what lies in this region—the unconscious.[1]

Psychic symbols—ideas, feelings—must therefore be traced farther back than the conscious level at which the individual is acquainted with them in order to understand their real meanings. Psychoanalysis is as important for the understanding of the construction of the psyche as dissection is for the understanding of the structure of the body, or chemical analysis for the understanding of the constitution of the molecule.

The greatest deficiency in the psychology of the nineteenth century relative to the understanding of human conduct has been the neglect of the unconscious.

For centuries man has marvelled and speculated and gathered observations concerning the exquisite subtleties of adaptation of plant structures to their environment. Students of nature have recorded in encyclopedic proportions the intricacies of Nature's story of the conduct of the lower animals from protozoa to highest ape. These activities have been relegated to tropisms and to instincts. Man alone has supposed that he could explain his own conduct by reference to that which appears in his consciousness, unmindful of the millions of years of evolution preceding that which he has designated as his conscious activities.

With the help of the hypothesis of the unconscious, however, it has come to be recognized that the psyche has its embryology and its comparative anatomy—in short, its history—just as the body has and in precisely the same way as in the case of the body this history has to be utilized before it can be understood.

So long as the unconscious failed to be recognized, just so long was the gap between so-called body and so-called mind too wide to be bridged, and so there arose the two concepts, body and mind, which gave origin to the necessity of defining their relations. Consciousness covered over and obscured the inner organs of the psyche just as the skin hides the inner organs of the body from vision. But just as a knowledge of the body first became possible by the removal of the skin and the revealing of the structures that lay beneath, so a knowledge of the psyche first became possible when the outer covering of consciousness was penetrated and what lay at greater depth was revealed. As soon as this was done, the wonderful history of the psyche began to give up its secrets, and the distinction between body and mind began to dissolve, until now it has come to be considered that the psyche is the end-result in an orderly series of progressions in which the body has used successively more complex tools to deal with the problems of integration and adjustment.

The hormone is the type of tool at the physicochemical level, the reflex at the sensorimotor level, and finally, the symbol at the psychic level.

[1] Jelliffe, S. E.: Paleopsychology, a Tentative Sketch of the Evolution of Symbolic Functions, Psychoanalytic Review, January, 1923. See Hughlings Jackson on Evolution and Dissolution of Function for conception of levels of the nervous system.

In the phyletic history of that development which culminates in man, the symbol has been developed after trying and laying aside a great variety of tools, because it offered the means of unlimited development of man's control over nature. The hormone, the reflex, are confined in their capacities for reaction within relatively narrow limits of possibilities. The symbol is capable of infinite change and adjustment, and so has grown out of the necessity created by ever increasing demands. The growth from the lowest to the highest, from the youngest to the oldest, from the simplest to the most complex has been here, as everywhere in nature, without gaps.

CHAPTER I.

METHODS OF EXAMINATION OF THE NERVOUS SYSTEM.[1]

THE student of human disease, whether manifesting itself through faulty integration of the vegetative mechanisms, through disturbance in somatic sensorimotor pathways, or in faulty social conduct, should first learn a practical, systematic series of methods for examining his patients. Such a scheme of case-examination should be orderly, and sufficiently extensive to enable him first to make a rapid sizing-up of the chief features of the disturbance under investigation. The first approach should be general and not detailed. A broad perspective is the primary requisite.

A *minimum requirement* case—history should include a study of the

I. Family History.
II. History of Patient's Previous Life.
III. History of the Present Illness.
IV. General Observations.
V. General Examination of Bodies—Major Organs.
VI. Examination of Vegetative Nervous System.
VII. Examination of Somatic Sensorimotor Pathways.
VIII. Examination of Mental Functionings.

For the sake of convenience the main factors to be covered are here given in the form of printed questionnaires. Such printed schemes have advantages as well as disadvantages. If carefully and thinkingly used the former outweigh the latter. For the beginner to whom this book is addressed chiefly, such routine examinations well systematized are the first steps in the development of a technic of case-examination which will prove invaluable. It is not thought that other forms may not prove as satisfactory, but some definite scheme is indispensable.

Questionnaires.—The heading, size, form of binding, of ruling, etc., will be determined by local conditions and by special needs. The definite data which are noted in the questionnaires are not by any means all of the observations which may be collected, and such printed question blanks are given here simply as guides, rather than as finished products. As practical blanks for out-patient dispensary work they are valuable and also for note-taking in private work. In hospital work where it is hoped that autopsy material may afford opportunities for research, the main data are essential, but they are insufficient for detailed studies.

A general-status blank is advisable. Such a blank is here given in short form: Its details are discussed later.

[1] This chapter cannot claim to be a semiologie. Advanced students should consult special works. The most complete is that of Dejerine, Semiologie des Affections Nerveuse. The work of Lévy-Valensi, Diagnostic Neurologique is very practical. In English, Purves Stewart, Diagnosis of Nervous Diseases, Barker, Monographic Medicine, vol. 4. Tilney and Riley, Forms and Function of the Nervous System and Oppenheim Textbook, Tr. by Bruce, are recommended. Frequent footnotes refer to special studies of significance and further tests of function are found in later chapters.

NAME: Age:

Birthplace: Date: In U. S.: Relig.:

Occupations: $: M.S.W.D.: Enlist: Draft:

Home Address: Nearest Relative: Rank: Co: Corps:

Admitted: Referred: Transfer: Examiner:

Heredity:

Parents related: Birthplace:

F. Mental:

M. Nervous:

Children: Epilepsy:

TBC: Diabetes:

Alcohol: Goitre:

Syphilis: Endocrinopathy:

Birth: Early History:

Walk: Talk: Read:

Children's Dis.:

Enuresis: Nail bite:

Sleep Walk: Stammer:

Other neurotic traits:

Education:

Adult Status:

Syphilis: Treatment:

Shocks: Tropics:

Habits: Alc.: Tob.:

Mast: Sex Experience:

Ardor: Frigidity:

Accident: Gunshot:

Occupation Toxemias:

Convulsions (injury—tongue—urine):

Constitution: Wt.: Ht.:

Marriage:

Menses:

Children:

Mis.: Dead:

Military History:

Present Illness:

[1] Personal Experience has shown that a blank printed on paper 8 x 10½ is a practical size.

I. **Family History.**—*Informant* (name, relationship to patient, address.)

Grandparents; *parents* (uncles and aunts); *Siblings.*

Children (with abortions and miscarriages).

NOTE.—In securing the family history it must be remembered that it is equally important to get a record of all the well members of the family as well as the sick ones and not stop with securing the latter as is often done. The relation to possible hereditary factors can only be determined by securing the fullest information about his ancestors.

It is desirable to get a family history as well as an objective history both from the patient and from other members of the family.

The first factor to be investigated is heredity. The usual facts gathered concerning heredity are frequently worthless. Better no facts concerning the extremely complicated question of heredity than false ones. A complete consideration of heredity is best left to special eugenic students whose business it has become to properly collate and weigh the mass of material needed to bring out salient factors in the heredity problem. The main facts to be noted in the questionnaire concern the following:

Hereditary nervous diseases, such as Friedreich's ataxia, Huntington's chorea, Thomsen's myotonia, aplasia axialis, cerebellar disease, spinal paraplegia, muscular dystrophies, myatonia congenita, myoclonus epilepsies, stuttering. *Hereditary metabolic factors*, such as, diabetes, icterus, splenomegaly, juvenile arteriosclerosis, hemophilia, obesity, cystinuria, alkaptonuria, achylia gastrica. *Hereditary structural anomalies*, such as, cervical rib, polydactyly, bradydactyly, tower skull, multiple exostoses, chondrostrophia, tooth anomalies, split hand, split foot, ankylosed fingers, wolf palate. *Hereditary sense organ anomalies*, such as, hemeralopia, lack of iris, hydrophthalmos, coloboma, distichiasis, glaucoma, myopia, ptosis, nystagmus, epicanthus, color blindness, retinitis pigmentosa, albinism, deaf-dumbness, *Hereditary skin anomalies*, such as, keratoma palmara, epidermolysis bullosa, alopecia, xerodermia, hypo- and hypertrichosis, neurofibroma, prurigo, ichthyosis, ephelides, telangiectoma. *Hereditary genital anomalies*, such as, epispadias, hypospadias, phimosis, undescended testicles, atresia vaginæ, uterus bicornis, polymastia, gynäkomastia, aplasia of mammæ. Photographs should be taken, or sketches to aid description.

Consanguinity in the parents is first to be inquired into; the degree of relationship should be explicitly expressed. Rough charts constructed on the plan of Fig. 1 should be used to show the relationships. Crzellitzer's chart is also a practical one.

Consanguinity may or may not have any relation to nervous disorders; it much depends on the stock. Constitutional factors present in one or another may become recessive in the descendants. Mendelian laws are well established in certain disorders. With dominant neuropathic traits of the same kind in both ascendants, the chances are less hopeful in the descendants. In human fecundity but few of the ova are impregnated, and each ovum undoubtedly contains its own

individualistic grouping of hereditary factors. The chance element seems too great to estimate. One feature of consanguinity not to be overlooked is that many relatives (first cousins, second cousins) marry each other because of a neuropathic tendency.[1] It is not impossible that the evil results sometimes seen is a transmission, are imitations of the neuropathic traits that were primarily responsible for the marriage. The studies of Woods,[2] Cox,[3] Lundborg,[4] Punnett,[5] Bateson,[6] Karplus,[7] Karl Pearson,[8] Rudin,[9] Johannsen[10] and others[11] show that the problems are extraordinarily complex, and that general statements are usually wrong. Johannsens new conceptions are specially to be recommended to the student of hereditary problems. These must be sought for in special studies in footnotes.

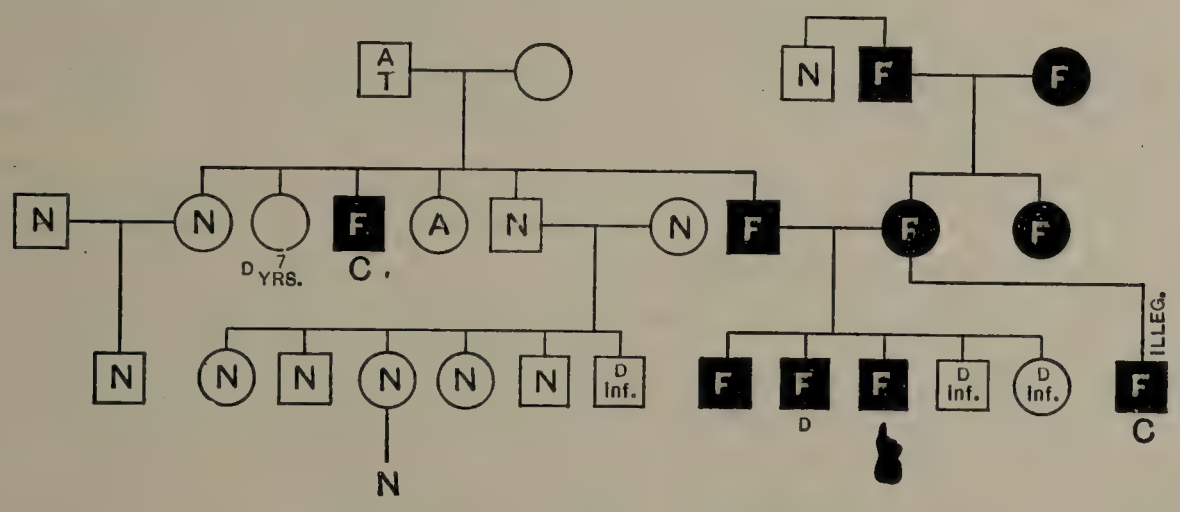


FIG. 1.—Pedigree chart illustrating how two defective parents may have only defective children. *A*, alcoholic; *C*, criminalistic; *D*, infant, died in infancy; *F*, feeble-minded; *N*, normal; *T*, tuberculous □ = Males. ○ = Females. (Goddard, 1910.)

The concept of inferior organs (Aristole, Adler) should be considered in the matter of heredity in a much broader sense than heretofore. Inferior organs, in their phylogenetic relationships have to be considered and the indirect results noted rather than attempting to follow out heredity along the concrete lines heretofore in evidence. For example, it has recently been shown that in the ascendants of pellagrines there was a noticeable tendency to diseases of the skin and of the gastro-enteric tract. In the interpretation of such a fact the evident relationship of the skin and mucous membrane of the stomach and intestines has to be correlated with the presumed etiological factor of

[1] Die Stellung der Verwandtenehe in der Psychologie der Neurosen, Jahrbuch f. Psychoan. u. Psychopath. Forschungen, 1909.

[2] Heredity in Royalty, 1906.

[3] Psychiatrische Neurologische Bladen, 1907.

[4] Ueber Degeneration, 1901.

[5] Mendelism in Relation to Disease.

[6] Brain, 1906.

[7] Zur Kenntniss der Variabilität und Vererbung am Zentralnervensystem, 1907.

[8] Biometrica.

[9] Rüdin: Studien über Vererbung und Entstehung geistiger Störungen, 1916, and a series of studies from the Research Institute in Mental Disease of Munich, chiefly to be found in Foerster and Willmann's Monograph Series, Julius Springer, Berlin.

[10] Johannsen: Erblichkeitsforschungen, 1913 and later editions.

[11] Siemens: Heredity, tr. by Barker: Also see journal literature. Eugenics, Biometrica, and others. See specially vol. 1 of Brugsch and Levy, Der Biologie der Person for important literature to 1926.

the disease. Thus the work of Goldberger would point to pellagra as a metabolism disorder belonging in the group with beriberi and scurvy and dependent upon a deficiency factor in the diet. The toxemia resulting produces the symptomatology which largely manifests itself as a peculiar form of dermatitis associated with a gastro-enteritis. The lack of some phosphorus compound is a necessary and specific factor, while the toxin which results produces its effect upon the inferior organs, the skin and mucous membrane. Viewing the facts of heredity in this larger way will undoubtedly broaden our concept of various diseases.[1]

Mental Disorder.—The presence of mental disorder in the direct ancestry is of moment,[2] not as indicating that mental disorder as such is inherited but as an aid to an understanding of the characteristics of the stock with which the individual comes to take up his life problems. The evidence drawn from aunts and uncles is of value, while

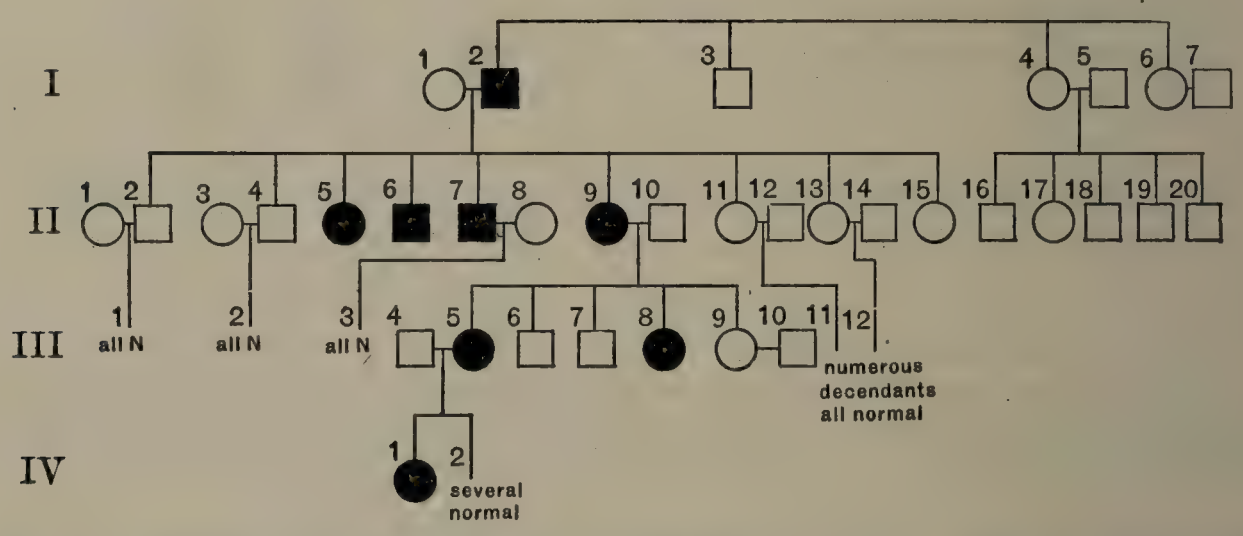


FIG. 2.—Pedigree of a family showing Huntington's chorea. Affected persons (indicated by black symbols) are always derived from affected parents. From original data furnished by Dr. S. E. Jelliffe; Smi family. (Davenport.)

that from cousins is open to the influence of another stock. It is useless to record the fact "insane" without any definite knowledge of the particular character of the mental disturbance. "Insanity" as a disease entity has no existence, the term is a legal one and has no place in medicine. Mental peculiarities should be noted: great avarice, originality, queerness, eccentricities, great capability, marked incapacities, temperamental peculiarities, inability of husband and wife to get along, suicides (valuable as evidence only if cause be known), all these may be of considerable service in offering a clue to important considerations in sizing up the sick individual's difficulties. The occurrence of the hysterical type of reaction in the parents or in the brothers or sisters

[1] Also see Henrichs. J. Norsk. Mag. f. Laeg. 1923, vol. **84.**, for related instances of hereditary ichthyosis and mental disease.

[2] Bleuler: Mendelismus bei psychosen speciel bei der Schizophrenie, Arch. suisses de Neurol., No. 1, vol. **1.** Kahn: Erbbiologisch-klinische Betrachtung, Z. f. d. g. N. P., October, 1920, 264. Bremer: Heredodegenerationen, Arch f. Psych, 1922, **66**, 477. Harnhart: Schw. med. Wchnschr., 1923, **53**, 139. Bauer, J. Praktische Folgerungen aus der Vererbungslehre, Beiheft. med. Klin., 1925, No. 1.

should not be overlooked, but deductions therefrom should be founded on precise criteria, not haphazard guessing.

The foundations of precise hereditary constitutional factors in definite nerve disorders are still far from being secure. Certain forms, however, stand out with marked constitutional backgrounds. The more important of these are tics, myoclonias, Huntington's chorea, Thomsen's disease, myopathies, certain congenital brain defects, as cerebellar defects, speech defects, skin defects, optic defects (color-blindness). Careful history-taking will undoubtedly reveal other constitutional hereditary forms, for fundamental studies have shown that brain form, brain cytotectonic, cord form, cord myelotectonic, etc., are directly transmitted. Certain occupation neuroses, some migraines, many eye-strains, etc., are possibly influenced by the continuous transmission of structural variations.

Epilepsy.—The occurrence of epilepsy in the ascendants, as epilepsy, means little. When both parents are known to have the syndrome the chances of transmission of a defect are great. Epilepsy, like many another disease so-called, is resolvable into many different disorders. The convulsive seizure is only an end-result of a vast variety of antecedent events in some of which only can hereditary factors be said to be important.

Alcoholism.—Alcoholism, if excessive, should never be overlooked. If possible the cause should be recorded, for in the last analysis the craving for drugs, conscious as well as unconscious, is to be regarded as the individual's attempt to escape certain inner conflicts. Alcoholism may mean hysterical or compulsive reactions. It may be a schizophrenic symptom. Periodicity in drinking should be carefully inquired into, particularly in its relation to the cyclothymic constitution (manic-depressive psychosis).

Migraine.—The hereditary factor in migraine is much exaggerated. The frequency of the disease has served to bring about this confusion.[1]

Tuberculosis and Diabetes.—Tuberculosis, diabetes, tendency to arthritic disturbances may have hereditary factors, the exact significance of which it is hard to estimate; tuberculosis also especially, because of its extreme frequency. The presence of diabetes in a parent should not be overlooked. It seems to be indicative of faulty emotional control acting through a constitutionally or otherwise defective pancreas; as such it is found frequently in ascendants of neuropsychiatric cases.

Syphilis.—Syphilis as an congenital factor should never be disregarded. Not only does one meet with congenital tabes, paresis, hydrocephalus, optic atrophies, deafness, etc., but evidence is accumulating that reinforces the belief that a syphilitic heritage is one factor at least responsible for much nervous and mental disorder, of a less tragic though perhaps more annoying character than those just mentioned.

[1] See authors chapter on Migraine in Osler: Modern Medicine, 1928, third edition, vol. **6**, also chapter on Migraine later.

Objective evidences of a syphilitic inheritance should be looked for in the teeth, bony formation, pelvic, chest, and cranial contours, etc. Wassermann tests of the blood and of the cerebrospinal fluid of parents may be necessary to accurately diagnose a subtle neurosyphilis in an infant, child, or even young adult. In such efforts it must be remembered that a negative result should not be regarded as final.

II. **History of Patient.**—*Birth: when learned to walk, talk and read. Childhood diseases,* particularly with reference to constitutional disturbances following—mumps and testicles, ovaries, or other endocrine involvement—childish habits, enuresis, nail-biting, thumb-sucking, sleep-walking, stammering.[1]

Endocrinopathic inheritance is of much moment, which is discussed later in the endocrinopathy questionnaire (see later).

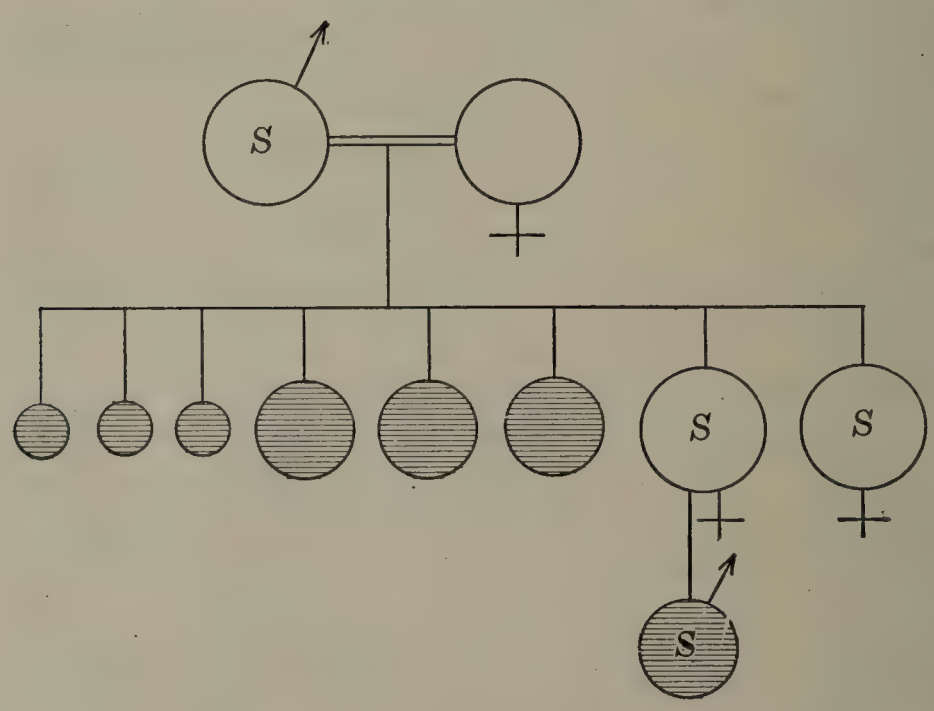


FIG. 3.—Congenital syphilis "unto the third generation." Syphilitic father of first generation. Second, three premature births, two dead children, and one dying at sixteen months. Congenital syphilitic next, with Hutchinson triad. This patient married, and had a child with snuffles, who died at age of six weeks. Remaining sister had infantile hemiplegia, Hutchinson teeth, and keratitis. (Mott.)

Diseases (especially convulsions, delirium, head-injury, gonorrhea, syphilis, rheumatism, neuritis).

Schooling history (backward, forward, interest, special likes and dislikes).

Habits (alcohol, drugs, masturbation, character of conscious erotic phantasies, ages of development).

Accidents (gunshot injuries, tropical diseases, poisonings).

Marriage: Menstruation; gynecological.

Previous attacks (special attention to so-called hysterical, to "nervous breakdown," and to excited or depressed episodes).

Crimes and misdemeanors.

[1] See works of Preyer, Sully, Shin, Stern, Hug-Hellmuth, Koffka and Watson, and the studies of Stanley Hall and his pupils in the Pedagogical Seminary for Infant and Child Behavior.

NOTE.—The history of the early life of the patient is especially important in order to obtain a comprehensive idea of the sort of person the patient was before becoming ill. A given disorder of the nervous system cannot be fully understood without understanding, not only the circumstances that gave rise to it, but the other and more important factor, the make-up or constitution of the individual in whom the disorder occurs.

The objective examination passes on to the *birth* of the patient. Was it as usual, or instrumental, or of excessive length? Hemorrhage, accidents, or pressure palsies may thus receive their interpretation.

Did the child learn to *walk* at an average period, *i. e.*, from nine to fifteen months (Preyer), and if not were there definite facts—excessive weight, intercurrent disease—to explain the tardiness? Was there precocity? Endocrine disorder is often correlated with marked anomalies of development. See Endocrine Questionnaire. Was *speech* acquired early or late? and did the child learn to *read* at the usual period (five to eight years, according to opportunities afforded)? These facts are of much importance, especially in estimating mental capacity, and for young children the probabilities concerning development and the need for special training. Children who learn to walk and talk as late as twenty-six to thirty-eight months respectively often remain very backward. The relations of speech to mental development are extremely close. Special tests, as the Binet-Simon or other special scales, are valuable for a general perspective of the intellectual development.[1] The clinical psychologist can be of great service here, if it be remembered that emotional factors play a large role in the responses to the usually employed intelligence tests and must be carefully evaluated.

Difficulties in teething, especially when attended with convulsions, should be noted. The rachitic chest form should not be overlooked.

Concerning *children's diseases*, much can be said. Severe measles, or scarlet fever, or other disorders may lay the foundation of a later developing epilepsy; diphtheria may cause various forms of neuritis with, at times, permanently weakened powers. Tuberculosis plays a very important role. The importance of influenza is often overlooked, while the relation of the acute streptococcic infections and their resulting joint involvements to chorea is well established. Excessive fatigue or exhaustion, either from an infectious disease, apart from its toxemia, or from overexercise or strain in rapidly growing children, bears a close relation also to choreic reactions. The infections also affect the endocrinous glands, thyroiditis, pancreatitis, hypophysial cysts, etc., and thus may exert a great influence in the metabolism of the growing child.

Enuresis should never be overlooked. If continuing past the third or fourth year it affords valuable evidence of an unconscious fixation which may later be substituted by a neurosis. *Thumb-sucking, nail-*

[1] See Whipple: Manual of Mental and Physical Tests, Warwick and York, Baltimore.

biting, teeth grinding, lip biting, tongue sucking, and other infantile traits should be inquired into. *Stammering* or other early speech disorder should be noted; *sleep-walking* also as well as food phobias, fear of the dark, etc.

The education of the patient, especially if a mental disorder or a psychoneurosis is under investigation, should be very thoroughly gone into. A knowledge of the earliest impressions gained, the picture books used, the principal childish associations formed, special tastes, animosities or dislikes are essential to the understanding of the obsessional, hysterical or allied reactions. The ideals inculcated, the religious and ethical training gained in the early years usually give a definite stamp to the personality and must be known if the adult personality is to be understood. The grosser factors of the classes passed, and the schooling received are desirable in estimating the grade of later mental capacity, and the application of intelligence tests in the study of the psychoses or psychoneuroses.[1]

Adult Diseases.—Syphilis stands in the first rank. Care should be exercised in obtaining a syphilitic history. Did you ever have a chancre? is the usual method of asking the question. The query, How old were you when you had a sore on the penis? although perhaps more abrupt, will give a higher percentage of positive answers, especially in those cases where its previous existence is largely inferred, as in general paresis or tabes. If the patient is apt to be squeamish, as in the case of many women, married or otherwise, questions concerning syphilitic infection are directed to the presence of symptoms such as continuous sore throat, hair-falling, etc. A physical examination for mucous plaques, leukoplakia of the mouth, and scars on the penis should never be omitted. In women the mucous membranes of the cheeks and sides of the tongue should always be examined.

The Wassermann or other blood reaction should be obtained in all cases where any doubt exists. Although the percentage of accidental syphilitic infections is low, nevertheless such exist. A single Wassermann test, positive or negative, is not conclusive, and great care should be taken in the choosing of a proper serologist. The subject of technic is a complicated and immensely important one.[2]

The subsequent evolution of the syphilitic infection should be inquired into and the character and length of time of treatment should be recorded.

Gonorrhea is not unimportant. It is of special relevancy in all arthritic disorders, in choreas (vaginal discharge) in young children, and in meningeal excitements. Gonorrheal neuritis is known.

Arthritis in its various forms calls for careful observation. Here one would best take roentgen-ray pictures, and not to be satisfied with

[1] Hug-Helmuth: The Sexual Life of the Child, Nervous and Mental Disease, Monograph series, No. 29, New York and Washington, 1919. Abst., Pediatrics.

[2] Plaut: Wassermann Reaction in Neurology and Psychiatry, Nervous and Mental Disease Monograph Series, No. 5, New York, 1910. Kaplan: Serology in Nervous Diseases, Philadelphia, 1914. See Solomon's work on Syphilis of the Innocent.

a description of the joint conditions. Tooth infection from the *Streptococcus viridans* is of importance as a chronic infection in producing several neurological syndromes.

The role of the infections in mental pathology is very marked. Typhoid fever and influenza[1] both constitute severe infections with marked influence on nervous tissues. Long standing focal infections of different organs of the body are not without very serious possibilities as factors in breaking down the resistance of the individual.

Under *shocks* is included sudden mental and moral influences tending to disturb the emotional life. Loss of money, of parents, husband, children, or loved ones, interference with one's hopes of a career, unfortunate entanglements, all call for investigation. The great importance of emotional disturbance in all nervous reactions should be borne constantly in mind. Faulty marital adjustments with resultant phantasy formations are responsible for a great amount of disease, whether evidenced as mental or physical. The psychoanalytic technic has revealed how much of this material is hidden from consciousness and goes on working year by year until a final "break down" occurs.

As to drugs, particular attention should be directed to alcoholism. The use of other narcotics, as opium, its allies, cocaine, the various hypnotics, bromides, etc., call for record. The use or abuse of these substances is an index of underlying difficulties which work havoc with the personality. The *craving* is the thing to understand. The cigarette habit, with inhaling, is often evidence of a strong narcissistic fixation.

Occupation toxemias should not be overlooked. They are daily assuming increased importance in America by reason of the increasing number of dangerous occupations. Workers in lead, arsenic, mercury, copper, pewter, pottery, dyes, sulphur compounds, and others suffer often from obscure symptoms, due to chronic poisonings. Occupation fatigues explain many neurasthenias.

The *erotic interests* should be inquired into. Infantile masturbation (often concealed) is almost universal. Persistent masturbation, is a symbol of unconscious conflict; it may be a cause of a neurasthenic group of symptoms. Unconscious masturbatory activity is the chief cause of the neurasthenic syndrome in the sense of Freud's definition of this syndrome. Frustration of the orgasm either in biological intercourse or its many variants is an important element in the development of neurotic symptoms (anxiety neurosis). The rarer anomalous erotic manifestations call for investigation in some cases. Masturbation may be unconsciously carried out in sleep; it may accompany phantasy, day dreaming without any genital contacts, and other areas of the body, may be used as unconscious, *displaced* genital areas. Thus many itchy patches on the thighs may be utilized for the inner satisfaction of the masturbatory impulse. The whole subject of the

[1] Jelliffe: Influenza and the Nervous System, New York Med. Jour., 1918, also see in monograph edited by Crookshank, F. G., Influenza, Heinemann, London, 1922.

unconscious substitutes for masturbation is highly complex. In many the cigarette habit serves such a purpose.[1]

Special attention should be paid to *convulsive seizures*, either occurring in the young or in adult life. Certain facts about such convulsions should never be omitted. These are the presence of dizziness, or of objects revolving (direction to be noted); of the state of consciousness; whether there is injury to the body during such attacks; if the tongue be injured; if urine or feces be voided during the attack; and if there be amnesia, complete or partial, following the attack.

The influence of *violence or injury* to the body is often of extreme importance. If there is accompanying mental shock, record of the fact should not be omitted.

The general *constitution* of the patient—his or her general capacity for work and fatigue—is to be noted. The question of general temperament, of outlook on life, may be tentatively entered in this place.

In the female, special attention should be directed to the *menstrual* history. The number and character of the births, the health of the children, the number of miscarriages with causes should be recorded.

Psychogenic factors play a large role in gynecological medicine, some gynecologists admitting that from 50 to 75 per cent of the usual difficulties are of such a nature.[2]

III. **Present Illness.**—*Onset; cause; physical pains; disabilities; mental and moral changes; emotional condition; hallucinations and delusions; judgment; memory; suicide and homicide; insight.*

NOTE.—Under this head an inquiry is made into all the circumstances surrounding and conditioning the onset of the disorder, the patient's attitude toward it and his insight.

Subjective History.—The patient's own account of his illness can either precede or follow the outlines of the family history and his general previous condition. The history is recorded in the questionnaire on the side of the blank, thus keeping it separate from the preceding and after-coming facts.

It is advisable for the patient to fix as nearly as possible the date of the onset of his illness. Certain facts which may or may not have had any connection with the malady under study may aid in fixing such a period of transition between health and sickness. The nature of its onset, whether acute and progressive, or acute and regressive, insidious and irregular, or very severe and immediate. What did the patient notice at that time? Then gradually trace, step by step, hour by hour, day by day, week by week, or year by year, the development of the disorder. What new symptoms have been added to the first—what have disappeared; has the picture remained the same, or has it gradually or suddenly altered?

A methodical going-over of the locomotor, sensory, emotional, intel-

[1] Stekel, W.: Die Onanie, Vienna, 1921. Freud: Three Contributions to the Theory of Sex, Nerv. and Ment. Dis. Month., Series No. 7.

[2] See Liepmann: Psychoanalysis and Gynecology. Stekel: Frigidity in Women, Boni and Liveright. New York, 1925.

lectual, skin, digestive, respiratory, and secretory systems should follow the patient's own account of the difficulty. Pains felt should be roughly charted and carefully located. If certain symptoms have disappeared, attention should be paid to the mode of their onset and of their departure. It is important to ascertain just what effect the illness has upon the social, familial, physical or psychical life in order to estimate the severity of certain symptoms. Also, has the patient consulted other physicians, or has he visited institutions or other than trained medical men of various kinds? What was the treatment? Its effect?

A thorough subjective anamnesis is one of the most difficult, and at the same time the most important features of a neurological or mental examination, particularly the latter. If the gradual evolution of the disorder is carefully elucidated, there is little danger of going wrong. If one hops from one thing to another, however, mistakes will be frequent.

IV. **General Observations.** — *Facial expression; appearance and demeanor; movements; disabilities; gaits; anomalies of innervation; speech; mental.*

NOTE.—The general observation of the patient is, of course, always important: whether he appears silly, resentful, indifferent; whether he has mannerisms, etc. It is particularly important, however, in stuporous and delirious patients who either will not speak or are not responsive and therefore not accessible. These patients should be observed particularly as to their general attitude of body and limbs, the expression of the face, the reflexes, and the reactions—volitional emotional, and organic (hunger, erotic bladder, bowels, etc.).

The Objective Examination. — During the subjective examination many facts concerning the general attitude of the patient have been gained. His expression and carriage, in bed or able to be about, the character of his intelligence and the responsiveness of his answers. Is his mind clear and is he oriented?

V. **Physical Examination.**—*Form; nutrition; weight; height; skin; bones and joints.* Roentgen-ray pictures as many as indicated.

Decubitus; scars (especially penis and mouth).

Respiratory system.

Circulatory system; heart position, size and sounds; blood-pressure.

Genito-urinary system.

Gastro-intestinal tract; stomach content (if indicated); *glands; abdomen.*

Sputum.

Blood composition.

Cerebrospinal fluid.

Urine.

NOTE.—It is hardly necessary to insist upon the necessity for a thorough examination of the visceral functions for every patient. It is especially important in the deliria in which the mental disorder may be the expression of an obscure somatic condition.

As has already been noted, a scheme is of great advantage, for

beginners particularly. With increasing experience, one may depart from a hard and fast method of case-taking, but in the beginning the student should habituate himself to a rigid and exhaustive method if he would avoid careless work.

The *cranium* should first be inspected. Is it regularly shaped, or is there asymmetry? Measurements may be taken if great anomalies exist. The teeth should be carefully examined. (See Endocrine Status). Roentgen-ray pictures are desirable.

The occurrence of isolated, or even several, so-called signs of degeneration may be recorded. There is no necessary connection between such anomalies and nervous or mental disease. They are found in superior as well as inferior deviates. Great departure from the average is worthy of record, but the hasty generalizations of the Lombroso school should be avoided. These deviations from the average structures will be discussed later.[1]

Careful and thorough percussion of the skull may reveal local points of tenderness (brain tumor), etc. The presence of cicatrices (epilepsies), depressions (fractures), or abnormal elevations is to be noted. Roentgen-ray examination of the skull is of great value, and should always be made for suspected fractures, for many brain tumors (acromegaly). The sella turcica is a very variable structure.[2]

VI. **Vegetative System Examination.**—A systematic presentation of methods for examining the vegetative reactions is only just being formulated.[3] The vegetative system consists of two more or less opposed systems, the autonomic, or extended vagus system, and the sympathetic proper. Every organ of the body is held in a balanced relation by these two systems. Changes in this balance underlie a true dynamic pathology of all of the organs of the body. A hyperactive autonomic type of reaction has been termed by Eppinger and Hess, vagotonic; of the sympathetic, sympathicotonic. These two contrasting types show a number of characteristic anomalies which are fairly constant and capable of objective examination. Not only are there a variety of anomalies of the autonomic and sympathetic reactions to be observed but a host of clinical signs are known which are dependent upon disorder of the metabolism of different parts of the body due to disturbances of the vegetative system acting upon the glands of internal secretion, endocrinopathies. Many of these are very marked as in myxedema, or acromegaly for instance; others, however, are less striking, but close observation will reveal a great many metabolic variations due to a uniglandular or polyglandular defect or imbalance.

It is important that the student should bear in mind that a polyglandular disturbance may have brought about marked anomalies of

[1] The student should have some acquaintance with such studies as Kretschmer on Body Formation and Character. Wertheimer, Draper and others.

[2] Ventrikulographie, etc. Jungling and Peiper, Theime-Leipzig, 1926. Gordon and Bell: Sella Turcica in Children. Endocrinology, 1925, **9**, 265.

[3] See Söderbergh and André Thomas: Revue Neurologique, June, 1926, vol. **33**, Thomas' presentation is of great value. Also see Laignel-Lavastine's large work on the Pathology of the Sympathetic, 1924, in French.

structure and yet at the time of observation may be no longer operative; that is compensation may have become effective and the structural anomaly or anomalies remain as a witness to a struggling series of interrelating compensations. On the contrary, and this is even more important, a polyglandular disturbance may be operating and yet not be sufficiently advanced to produce structural anomalies. In all events the minute testing out of each endocrine function is imperative. Another extremely important consideration should always be in mind. It is not always the most observable overstrained glandular activity that may be primarily at fault. Thus an obviously hyperthyroid disturbance may only be an over-compensatory activity for an under-functioning thymus, or other glandular defect. Many a thyroid has been removed unnecessarily because of failure to recognize a glandular interactionism which is constantly going on in the human body.

In this section attention will first be briefly centered upon some of the objective signs to be looked for resulting from disturbances of the vegetative reflexes, and secondly to those more closely related to endocrinous modifications. The two series of observations are very closely related at times. More thorough discussion of the syndromes or diseases are found in the first section of this book on diseases of the vegetative nervous system and the endocrinopathies.

From the very lowest to the highest forms of animal life a nerve network is present which has for its chief function the regulation of metabolic activity. It has steadily advanced in complexity from the Poriferæ to man. In this work it is called the vegetative or visceral nervous system and has come to be recognized as innervating a reciprocally active series of opposing mechanisms. One of these is called the sympathetic, the other the parasympathetic or autonomic. The vagus is the type of the latter. The splanchnics the former. The health, or adequate physiological activity of every structure of the body, including growth, blood formation, bony growth, inflammatiory reactions, etc., depend upon the balanced relation of the stimuli passing through the autonomic (vagus) and the sympathetic reflex arcs. The terms vagotonia and sympathicotonia indicate the tonus in the opposing branches of this system. Eppinger and Hess[1] devised these words to indicate the tendencies to excessive reaction in one or the other of these reciprocally acting opposites. The overreactive parasympathetic they termed vagotonic: the other, sympathicotonic. Such over-reacting trends may result from a variety

[1] Eppinger and Hess: Vagotonia, translated by Jelliffe and Kraus, Nervous and Mental Disease Monograph Series, No. 20, New York. Also consult Barker and Slade: Tr. Assn. Am. Phys., 1912, p. 471. Higier: Vegetative Nervous System, Nervous and Mental Disease Monograph Series, No. 29. Laignel Lavastine: Internal Secretions and Nervous System, ibid., No. 30. Rosenbloom: Methods of Endocrine Testing: Interstate Med. Jour., November, 1918, et seq. Müller, L. R.: Lebensnerven, 1924. Guillaume. La Sympathique et les Systémes Associés, Paris, 1921, Pottenger, Symptoms of Visceral Disease, II ed., St. Louis, 1922. Laignel Lavastine: Sympathetic Nervous System. The most complete monograph to date, 1924. Dresel: Vegetative Nervous System. and the complete monograph of André Thomas, Les Moyens d'Exploration du Systeme Sympathique et leur Valeur. Revue Neurologique, June, 1926, vol. **33**. Ferrao, A.: Arch. d. pat. e. Clin. Med., III, September, 1924.

ENDOCRINE STATUS.

Heredity, Endocrinopathic:

Giants: Dwarfs:

Goitres: Diabetes:

Structural Peculiarities:

Familial Diseases:

Skeletal Type:

6° +: 5°5′ −:

Arm Leg Ratio:

Asthenic: Athletic: Pyknic Types:[1]

Trunk Leg Ratio:

Hands: Fingers: Toes:

Lordosis: Kyphosis:

Growth: Curve:

Bony Consistency. *X*-ray.

Facial Types:

Acromeg: Acromic:

Cretin: Myxedem:

Gravian: Addison:

Gerodermic: Eunuchoid:

Thymic: Lymphat:

Mongolian: Ovarian:

Head Segments:

Hydroceph: Microceph:

Jaw Shape:

Prognath: U: L:

Teeth: Spaced: Absent:

Lat. Incisors: Canines:

Color: Gingivitis:

Dentition Dates:

Descriptive details:

[1] See Kretschmer: Character and Body Formation.

ENDOCRINE STATUS—*Continued*

Skin. Subcutaneous Tissue:

Color: Texture: Cold-heat Reactions:
Tonus: Pigm: Nevus:
Atrophies: Hypertrophies:
Infilt: Edema: Fat:
Eruptions: Acne: Psor: Ecz:
Distribution: (Chart)
Stroking React: Pilomotor Reflexes—Important:
Gooseflesh: Urticaria: Venous Pressure:
Vaso Dilator-constriction: Mottling: Temperature Both Sides:

Hair: Nails:

Type: Color: Lustre:
Texture: Rough: Fine: Curly: Dry:
Distrib: Face: Body:
Age: Develop:
Eyebrows: Eyelashes:
Ridging: Brittle: Spots:
Growths:

Mucous Membranes:

Nasal: Mouth:
Atrophy: Hypertrophy:
Pigment: Blanching:
Aplasia: Hyperpl:
Tonsil Persist:
Tongue Papillæ:
Hay Fever: Leucorr:
Colitis: Asthma:

Descriptive details:

[1] See André Thomas' important monograph upon Las Reflexes Pilomoteurs, see Plate II in present work, p. 122.

ENDOCRINE STATUS—*Continued*

Muscles: Ligaments: | Descriptive details:

Massive: Sparse:
Atrophies: Hypertrophies:
Aplasia: Hyperplasia:
Tonus: +, −: Carriage:
Fatigability:
Flat Feet: Extensible Joints:
Ptoses: Contractures:

Sex Character Development:

Male: Size: Shape Penis:
Implant: Prepuce:
Scrotal Tonus: Testicle:
Pubes: Pelvis:
Hair Male: Hair Fem.:
Beard: Body Hair:
Puberty Age:
Voice changes: Breast:
Erotism: Ardor: Frigid: Orgasm:
Homo vs. Hetero Trends: Friends:
Fecundity:
Familial:

Female: Genitals Size: Character:
Clitoris: Labia:
Uterus: Ovary:
Hair Male: Hair Fem.:
Beard: Body:
Menst. Time: Character:
Voice Variations:
Erotism: +, −: Frigid: Orgasm:
Homo vs. Hetero Trends: Friends:
Fecundity:
Familial:

ENDOCRINE STATUS—*Continued*

Cardiovascular:

Blood Pressure { over 150 / under 120

Pulse { over 85 / under 60

Rest: Exertion:

Pulse Pressure: Venous Pressure:

Blush: Blood Picture:

Mottling: Sergent Test:

Aschner: Solar reflex:[1]

Vessel Palpat:

Pharmacodynamic Reaction to

Atropine: Pilocarp:

Cocaine: Adrenalin:

Mercury: Idiosyncrasies:

Metabolism:

Hypothermia: Hyperth:

Frilosity: Hot Flashes:

Weather Reactions:

Cataract: Glaucoma:

Blood Sugar:

Sugar Tolerance:

CO_2 Quotient:

X-ray Examinations:

Sella Turcica:

Pineal Region:

Sinuses:

Teeth:

Thymus:

Thyroid: Palpation:

Epiphyses:

Bony Structures:

Descriptive details:

[1] Garrelon et Santenoise: Jour. Phys. et a. d. Path. Gen., 1924, **22**, No. 4.

of stimuli. They may come from without such as cosmic forces,[1] toxic agents, hormones, or emotional situations. They may come from within as a part of the push of the instincts. Chronic tonus variations appear as dispositional (*i. e.*, ontogenetically acquired) or constitutional (*i. e.*, phyletically bound) expressions of this parallelogram of forces seeking equilibrium. The *vagotonic* (vagus tonus or sympathetic paralysis) shows relative insusceptibility to sympathetic stimuli; hence cold skin, compressible slow pulse, deep-set eyes, contracted pupils. The skin is apt to sweat readily, at times in patches or blotches; the palms are apt to be thick. They are usually undernourished. Short-sightedness is frequent. Acneiform eruptions, particularly of the back, are usual. They frequently swallow as they talk, and seem to have much saliva in the mouth. Sore throats are frequent. The faucial reflexes are usually diminished. Increase of hair about nipples and masculine distribution of the pubic hair is frequent in the vagotonic woman.

Pilocarpine test shows marked hypersensitiveness and is a useful mode of examination. This is to be given hypodermically, 0.01 to 0.003 gm. Higier recommends 0.0001 per kilo of body weight. These tests are to be interpreted like all others solely as monosymptomatic and weighed with other signs. Careful records should be made of the blood-pressure, perspiration and pupillary reactions, every ten minutes for an hour.

The expression of the *sympathicotonic* (sympathetic hypertonus) is that of the warm-blooded type. Here one finds vivaciousness, dilated pupils, glistening eye, rapid pulse, warm and dry skin. There is relatively insusceptibility to pilocarpine and also to atropine (0.001 to 0.00065 gm.), while adrenalin, 0.001 gm. increases all of the signs present. Cocaine in hypodermic form should also be tested out in the same manner. The student is cautioned to be conservative regarding the results of pharmacological testing of the functions of the vegetative system. Eppinger and Hess overstated their case, yet the data obtainable may be of great service if not too rigidly interpreted. Insofar as a large chapter in neurology is still to be written concerning the nervous regulators of metabolism;[2] the pathways to be chartered, the midbrain synaptic relay stations identified and the cortical organizing areas differentiated, all data obtainable are welcomed. Special attention is directed to the masterly summary of Spiegel[3] where the advances to 1922 are brought together,[4] and the later (1926) summary of André Thomas.[5]

[1] As example see Pucher, G. W.: Chemical Changes Produced by Light Energy, Buff. Gen. Hosp. Bull., December, 1925.

[2] Nöel Paton: Nervous Regulators of Metabolism.

[3] Jour. Nerv. and Ment. Dis., June, 1922.

[4] Also consult Bergmann: Deut. Ztschr. f. Nerv., vol. **45**, 346. Pophal: Ergebn. d. inn, Med., vol. **19**, 782. Friedberg: Ergebn. d. inn. Med., vol. **20**, 173. Dresel: Deutsches, med. Wchnschr., 1919, vol. **35**, ibid, Ztschr. f. exper. Path. u. Therapie, vol. **22**, 34.

[5] André Thomas: Reactions anserines, Presse Méd., July 6, 1918. Le Reflex Pilomoteur, Paris, 1922. Les Moyens d'Exploration des Systéme Sympathique. Rev. Neur., June, 1926, vol. **33**.

It is to be borne in mind these are trends rather than fixed types, and that an individual may show vagotonic trends in one or more visceral segments and sympathicotonic reactions in others. These variabilities are pronounced in the so-called neurotic, *i. e.*, the individual with marked emotional (psychogenic) response.

Pathological reactivity of the visceral nerve reflex arcs shows itself in the eyes, nose, mouth, skin, respiration, circulatory, digestive, urogenital, cutaneous, and metabolic systems. Such may result from constitutional variations (endocrinous anomalies), chiefly in their response to body cravings which come partially to consciousness as emotional reactions, *i. e.*, psychic disturbances (symbolic systems, unconscious).

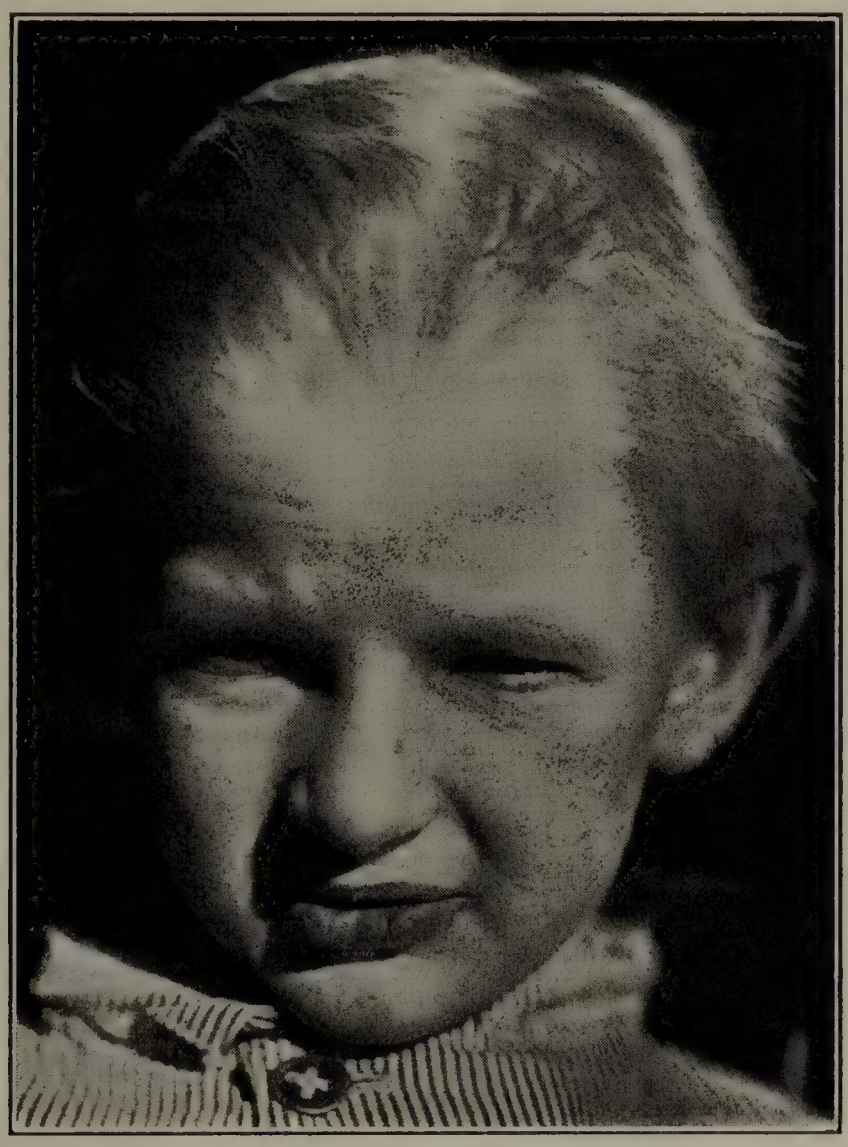


FIG. 4.—Lanugo hair. (Endocrinous disturbances.) (A. Josefson.)

Some of the signs that are partially understood are here very fragmentarily presented. Their relation to the problems of internal medicine are just beginning to be appreciated. Internal medicine as it becomes more dynamic needs neurological conceptions to interpret its findings.

1. *Metabolic Signs.*—Variations in fat and sugar tolerance, eosinophilia, lymphocytosis, pigmentations, bony and muscular anomalies.

2. *Cutaneous Signs.*—Horripilation (painful hair raising), goose flesh, contractions of testicle and of the nipple, seborrhea, hypertrichosis, baldness, hyperidrosis, bromidrosis, local syncopes, acrocyanosis, purpura, pruritus, pallor, dermographism, erythema, urticaria, Sergent white line.

3. *Respiratory Signs.*—Asthmatic attacks, laryngeal spasms, Aschner's sign (oculocardiac reflex), pressure upon the eyeball leading to slowing of the pulse with stopping of respiration in expiratory phase, coryzas, bronchitides.

4. *Circulatory Signs.*—Bradycardia, tachycardia, irregular extrasystolic pulse, dromotropia, vasomotor anginas, peripheral anemia and hyperemias, acrocyanosis, intermittent claudication, high tension.

5. *Abdominal Signs.*—Colicky diarrhea (sympathicotonic), spastic constipation (vagotonic), hyperchlorhydria, achylia, gastric atony, pylorospasm, gastrosuccorrhea, esophagism, hypersalivation, enterocolitis, solar reflex.

6. *Genito-urinary.*—Retention, incontinence, menstrual anomalies, ejaculatory disturbances, tenesmus, renal colic, priapism, frigidity, modifications of potency.

7. *Cranial Autonomic Signs.*—Eyes: Mydriasis or myosis, glaucoma, irregular lid slits, spasms of accommodation, von Graef's Moebius sign, exophthalmos, enophthalmos, dryness of eyeballs, Loewi's test (adrenalin mydriasis—adrenalin by conjunctiva, 1 to 1000 solution). Irregular pupils, Stelwag's sign (irregular and infrequent winking); nose signs are dry nose, or excessive secretion (hay fever), frequent spitting or very dry mouth.

The influence of various drugs upon different branches of the two systems is shown on page 175.

ENDOCRINE STATUS.—The chief observations which are to be recorded are given in the endocrine-status questionnaire (see pp. 38 to 41). The various syndromes are described later. The morphological examination includes the endocrine inheritance, observation of skeletal type, characteristic facial developments, variations in development of the head segments, special attention being paid to the teeth development, skin anomalies, hair, nails, mucous membranes, muscles, ligaments, sex-character development, cardiovascular reactions, pharmacodynamic reactions, general metabolic changes and *x*-ray examinations of the head and special glands. These are all systematically grouped on pages 38 to 41, a more detailed consideration following.

Facies.—Acromegalic, cretinistic, myxedematous, exophthalmic, Addisonian, eunuchoid, gerodermic, mongoloid, infantile, juvenile, lymphatic, chlorotic, adenoid, ovarian face of Spencer Wells.

General Development.—Weight and stature, excessive obesity or thinness. Microsomia, macrosomia, disproportionate development of upper and lower extremities, of the distal and proximal limb lengths. Epiphyseal calcifications, cervicodorsal kyphosis, genu valgum.[1]

Genital Development.—(*a*) In male: External genitals, descent of testicles, beard, distribution of pubic hair and hair of body, voice, fecundity or sterility, psychosexual characters, ardor, eroticism, signs of feminism, overarching scrotal folds simulating clitoris implantation of penis, precocious or retarded puberty. (*b*) In female: External

[1] See Kretschmer: Body Formation and Character. Draper: Constitutional Studies. Grühle: Körperban der Normalen, Arch. f. Psych., 1926, **77**, Heft. 1.

and internal genitals, breasts, pubic hair formation (male type rising in middle line, female type straight across), moustaches, menstruation, infantile uterus, eroticism or frigidity, masturbatory activities, orgasm in copulation, psychosexual satisfactions, fecundity or sterility, masculine types, precocious or tardy menstruation, menopause, character of pregnancy, excessive nausea and vomiting as unconscious protests or somatic difficulties.

Examination of trophic changes or disturbances.

(*a*) *In Skin.*—Myxedematous infiltrations, scleroderma, pigmentation (dyschromia), circumscribed or diffused, activity of vesicants, hypertrophies, atrophies, and precocious senile changes, anidrosis, hyperidrosis, eruptions, chronic ulcerations, circumscribed lipomatosis, adiposis, reactions to sunlight, roentgen-rays etc.

(*b*) *Hair and Nails.*—Character of hair: rough, fine, curly, dry, color; eyelashes and eyebrows. General hairiness—distribution. Crescents in nails, character of nail developments, splits, spots, and ridges.

(*c*) *Teeth.*—Primary and secondary dentition, dental forms, dental resistances, ridges, early caries; color, chalky-white or yellowish. Wide implantation, central incisors (pituitary?) lateral incisors related to gonadal development (?), canine, shape of palatal arch high (thyroid?) broad (hypophysial?).

(*d*) *Mucous Membranes.*—Pigmentation, hyperplastic, atrophic or catarrhal, lingual hypertrophy, ridges, etc.

(*e*) *Lens.*—Early cataract, opacities, floating bodies in vitreous.

(*f*) *Ligaments, Muscles, Bones.*—Atrophies, abnormal fragility, osseous growths, hypertrophy of bone, joints, muscular atrophy, hypertrophy, rheumatismal swellings, epiphyseal anomalies.

(*g*) *Blood.*—Blood count, eosinophilia, lymphatic hyperplasiæ.

(*h*) *Chemoregulatory.*—Albumin, phosphorus, iodin, sugar tolerance, calcium and magnesium metabolism, respiratory gases, alveolar carbon-dioxide tension. Hyperthermia, hypothermia.

Direct examination of endocrinous glands:

Hypophysis: Sella turcica by roentgen-rays, signs of hypophyseal tumor, results of hypophyseal extract. The form of the sella turcica is a partial index of the size of the hypophysis. Great variability in shape and size of the sella is usual. Erosion of the clinoid processes to adapt to the hypophysis is a frequent finding.

Thyroid: Size (20 to 30 gms. average), goiter and its character, retrosternal goiter, thyroid antibodies, thyroidine sensibility. Goetsch test.

Thymus: Radioscopy of region, tumor of superior mediastinum, thymic antibodies. The thymus persists longer than has been heretofore taught.

Suprarenal: Circulating adrenalin dosage, pain in suprarenal region, tumor, sensibility to adrenalin, adrenalin mydriasis, pigmentation. Sergent's white line.[1] This is best tested by marking out with

[1] Sergent: Études cliniques sur l'insuffience surrenale, Collected Papers, Maloine, 1914.

the finger tip, an oblong or square about the umbilicus; light pressure being used. There first results a slight goosefleshing in from ten to twenty seconds, this is followed by a blanching of the area which is wider than the pressure area. This white line persists in depressed suprarenal states and from a variety of exhausting causes—from two to five minutes or more. The usual reaction is a pinkish coloration, which in some individuals has a whitish line for a border.[1]

Genital Glands: Modification in size, neoplasms, effect of extracts.

Pineal: Signs of tumor. Pineal shadow on roentgen ray.

Pancreas: Sugar in urine. Blood sugar.



FIG. 5.—Infantile genitals from hypoövarism. (A. Josefson.)

VII. **Sensorimotor Examination.—Cranial Nerves.**—These should be systematically tested. (See questionnaire made out for a patient with tabes, pp. 48 to 51.)

I. *Smell.*—There are no satisfactory quantitative tests for smell apart from special physiological psychological tests that are of service chiefly in research work. The smell in each nostril should be tested separately, preferably by some well-known substance (oil of turpentine) and by a substance resembling well-known foods (asafœtida, onions) or bodily excretion (feces). The nostril of one side is stopped, and with the eyes closed the patient is asked to smell from a bottle containing the odorous substance; the other side is then tested with the same or different substance and comparisons are made. Variations in smell are very frequent, and much care must be exercised in drawing conclusions from smell anomalies. Influenza interferes with smell tests. Local conditions, empyema of the antrum, etc., must be excluded. Irritating substances, like ammonia, etc., should *not* be used. A search for subjective smell disturbances may be made at the same time. Anosmia unilateral or bilateral, is often present in fractures of the skull, in frontal lobe brain tumors and in certain epilepsies (uncinate gyrus).

[1] Massalongo: La linea bianca di Sergent nella insufficenza surrenale, La Riforma Medica, August 24, 1918, vol. **34**. Bernard, L.: Tribune Medicale, July 20, 1907.

II. *Eyes.*—Sight should be tested by the Snellen or other type cards. These should be well illuminated and the patient should stand with the window light behind him. Each eye should be tested separately. Patients unable to distinguish the largest letters should be tested as to their ability to see the fingers, determine light and dark. Some

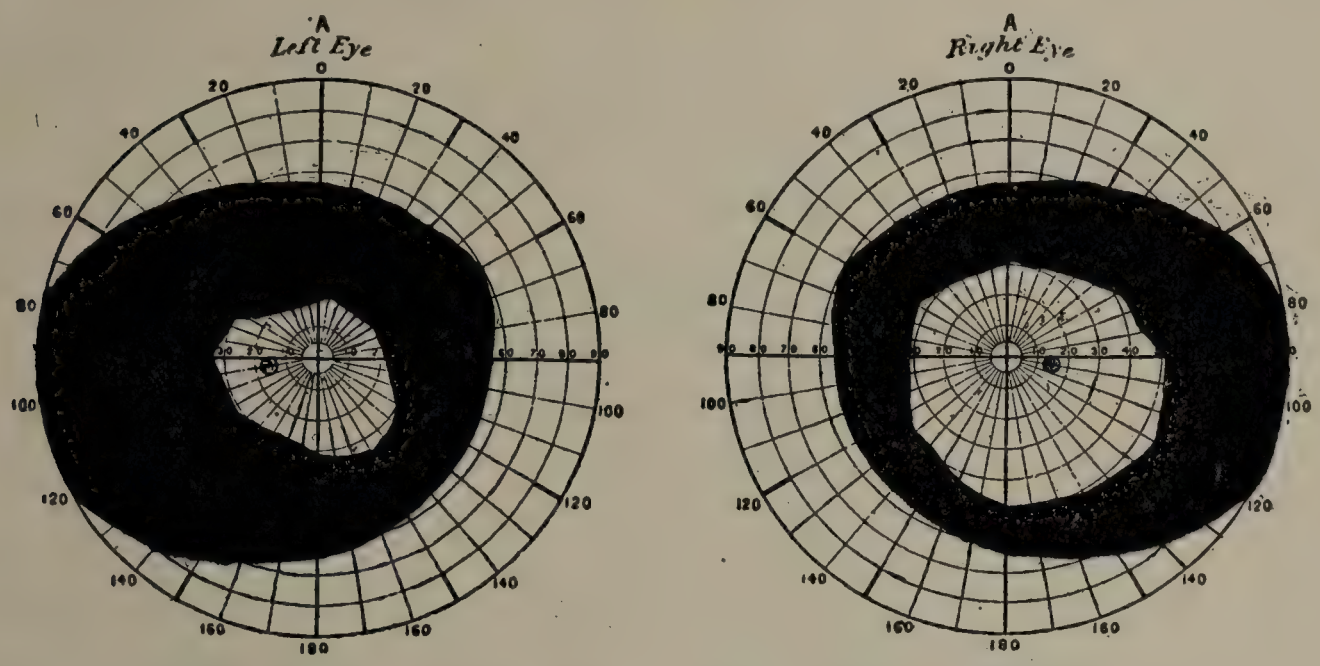


FIG. 6.—Crossed amblyopia in a case of hysteria. (Stewart.)

patients see better in a dim light than in bright light (hemeralopia) (see Plate VIII, p. 364, for family tree of patients who become blind in dim light—night-blindness, nyctalopia). Myopias and astigmatisms are important to bear in mind in testing the sight functions.

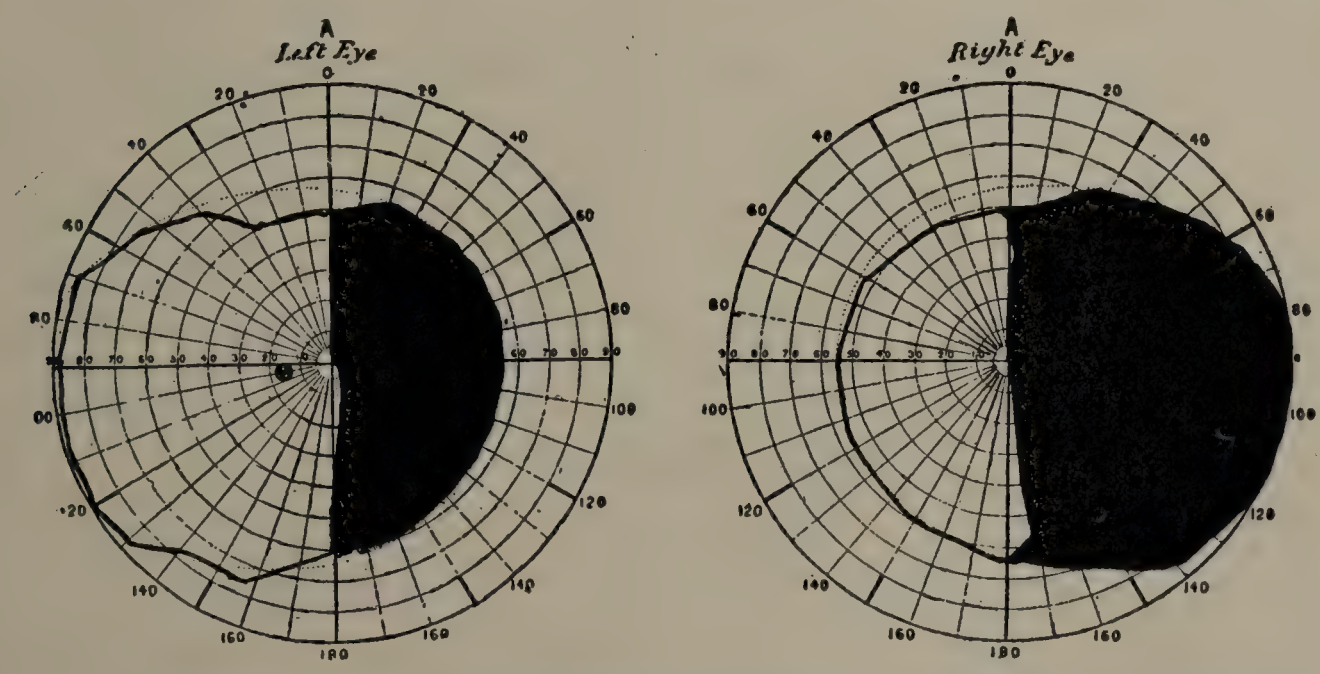


FIG. 7.—Right homonymous hemianopsia in a case of softening of the left occipital lobe. (Stewart.)

Color vision is important. Colored wools are matched as to shades in the full daylight. In color-blindness, if of the red-green variety, gray- or straw-colored wools are selected. In total color-blindness the individual confuses all colors; comparative degrees of brightness alone are distinguished. A number of ridiculous blunders may be made in testing for color-blindness with the wool tests.

No. *14250.* Diagnosis: *Tabes.*

NAME, *J. Jones.* Age, *42.* Occup.: *Clerk. M.S.W.*[*D.*]

Residence, *444 Spring St.* Race, *U. S. In U. S. 42 yrs.*

Date, *Jan. 5, 1910.* Examined by *Dr. J.*

Referred by *Dr. X.*

Heredity:
F., *d. 70; apoplexy.*
M., *d. 64; cancer.*
Children: *Only child.*
TBC: *0*
Alcohol: *0*

Parents related: *0*
Mental: *0*
Nervous: *0*
Epilepsy: *0*
Diabetes: *0*
Syph.: *0*
Eruptions: *?*

Birth: *Normal.*
Walk: *N.* Speak: *N.* Read: *6 years.*
Children's Diseases: *Measles.*
Enuresis: *0* Thumb: *0* Nail-biting, etc.: *0*
Sleep-walking: *0* Stammering: *0*
Other Childish Traits: *Cheek-biting.*
Education: *Pub. Sch., High Sch., to 17 years.*
Adult Diseases:
Syphilis: *26 years.* Treatment: *1 month, Internal: Hg.*
Shocks: *0*
Habits: Alc.: + Tob.: ++
Eroticism: *Average:*
Trauma: *0*
Occupation Toxemias: *0*
Convulsions (injury, tongue, urine): *0*
Constitution: *Healthy.* Weight: *150.*
Marriage: *At 32.*
Menses:
Children: *1; d. in infancy (convulsions).*
Mis.: *2; 4 mos.* Dead: *0*

History:

Chief complaint: Slight unsteadiness in gait and severe "rheumatic" neuralgic pains in lower extremities for past four years. Five years ago had a transitory attack of dizziness, with double vision and an eye palsy, cross-eye, which lasted two months.

Occasional weakness of the bladder, dribbling. Paresthesiæ occasional.

First page of Sensorimotor Questionnaire: Made out from examination of a patient with Tabes.

0 = negative. + = present, positive. *N* = normal or average.

SENSORIMOTOR STATUS—*Continued.*

Cranium: Hor., *N.* Bin., *N.* Vert., *N.*
Sensibility: *O. K.* Deformity: *0*
Asymmetry: *0*

Cranial Nerves:

I. Smell: { Ol. ter.: *O. K.* Subjective: *0*; Asafet.: *O. K.*

II. Sight: { R. 20/40; L. 20/100 } Position of Eyes: *O. K.*
Hemianopsia: *0* Scotomata: *0*
Fundus: *0* Fields: *Limited; con.*

III., IV., VI. Eye movements: *O. K.*
Nystagmus: *0* Palp. fiss.: *R = L.*
Diplopia: *0; 5 years ago.* Ptosis: +
Pupils: *R > L, 9 and 4 mm.*
Reflexes: { Light: *Lost L; dim. R.*; Accom.: *O. K.*; Symph.: *Dim. R > L.*; Consensual: *Lost L+R.*
Oculocardiac reflex: *O. K.*

V. Motor: *O. K.*
Sensory: *O. K.* Jaw-jerk: *O. K.*
Tender spots: *0* Cornea: *O. K.*
Conjunctiva: *O. K.*

VII. At rest: *O. K.* *R = L.*
Forehead: *O.K.* Eyes closed: *O. K.*
Teeth: *O. K.* Whistle: *O. K.*
Involuntary: *O. K.* Rosenbach: *O. K.*

VIII. Hearing: *Dim.* Equilibrium:
Rinné: + Weber: +
Tinnitus: *0* Vertigo: *?*

History—Contin'd.

O. K. = average, or usual reaction.

SENSORIMOTOR STATUS—*Continued.*

Cranial Nerves (continued):

IX., X., XI., XII. Fauces: *O. K.*

Speech: *O. K.* Stammering: *0*

Tongue: *Straight.* Swallow: *O. K.*

Scars: *0*

Tremor: *0* Respiratory: *O. K.*

Taste: *O. K.* Cardiac: *O. K.*

Shoulders: *O. K.* Neck: *O. K.*

Upper Extremity:

Atrophy: *0* Malformations: *0*

Hypertrophy: *0* Symmetries: *O. K.*

Spasm: *0*

Hypotonus: + + Twitching: *0*

Muscular power: *Dim.* *R=L.*

Dynamom.:

Nerve trunks: *Not tender.*

Triceps Rx.: *Dim.* Radial Rx.: *Dim.*

Tremor: *0* Rest: *0* Static: *0*

Ataxia: + F. N. T.: *Atax.* + F. F. T.:

Stereognosis: *O. K.*

Light touch: *O. K.* Adiadokok.: *0*

Position: *O. K.* Diapason: *Dim.*

Pain: *O. K.* Thermal: *O. K.*

Vasomotor: *0* Trophic: *0*

Hair, pigmentation, etc.

Trunk:

Power: *O. K.* Deformity: *0*

Spine: *O. K.* Malposition:

Epigastric Rx.: + Abdom. Rx.: +

Cremaster Rx.: + Anal. Rx.: +

Bladder: *Sluggish.* Rectum: *Sluggish.*

Light touch: *O. K.* Localization: *O. K.*

Deep Sens.: *Dim.* Diapason: *Dim.*

Pain: *O. K.* Thermal: *O. K.*

History—Contin'd.

F. N. T. = finger-nose test. *F. F. T.* = finger-finger test.

SENSORIMOTOR STATUS—*Continued.*

Trunk (continued):

Equilib.: *Unsteady.* Asynergia: +. *Slight.*

Vasomotor: *0* Trophic:

Dermographia: *0* Pigmentation: *O. K.*

Lower Extremity: Deformity: *0*

Atrophy: +; *legs flabby.*

Hypertrophy: *0* Asymmetries: *0*

Spasm: *0* Kernig: *0*

Hypotonus: + + Tremor: *0*

Muscular power: *Diminished.*

Synergistic tests: *R=L.*

Patellar: *0* Achilles: *0* Clonus: *0*

Babinski: *0* Chad: *0* Opp: *0*

Nerve: *Tender.* Lasègue: *Tender.*

L. touch: *Dim.* Pain: *O. K.*

Deep: *Dim.* Therm.: *O. K.*

Localization: *O. K.* Position: *Impaired.*

Diapason: *Impaired.*

K. H. T.: *Atax.* Romberg: + + +

Gait: *Markedly ataxic.*

Closed eyes: *Made worse.* Sidewise:

On heels: ++ On toes: ++

Asynergia: *O. K.*

Vasomotor: *0* Trophic: *0*

Status Corporis: *Medium nutrition.*

Heart: *O. K.* Murmurs: *0*

Blood: *O. K.* Wassermann: + +

Lungs: *O. K.* Arteries: *O. K.*

Abdomen: *O. K.* Blood-pressure: *160.*

Liver: *O. K.* Skin: *O. K.*

Urine: *O. K.* Joints: *O. K.*

Cerebrospinal fluid:

Wass.: ++ Cells: *50.* Globulin: ++

History—Contin'd.

Treatment:

Intraspinous injections of salvarsanized serum. (Swift-Ellis.)

Course of mercurial inunctions.

K. H. T. = knee-heel test.

EXPLANATION OF PLATE I.

The Pyramidal Tract[1] in its Corticospinal and Corticonuclear Portions.

The corticomedullary (corticospinal) *Co.M*, is colored light red; the corticonuclear portion, *Co.N*, darker red; the reticular substance (*S.R.*) of the tegmentum in yellow and the motor nuclei of the cranial nerves orange. The corticopontine portions and the cerebellar paths of the tegmentum are omitted.

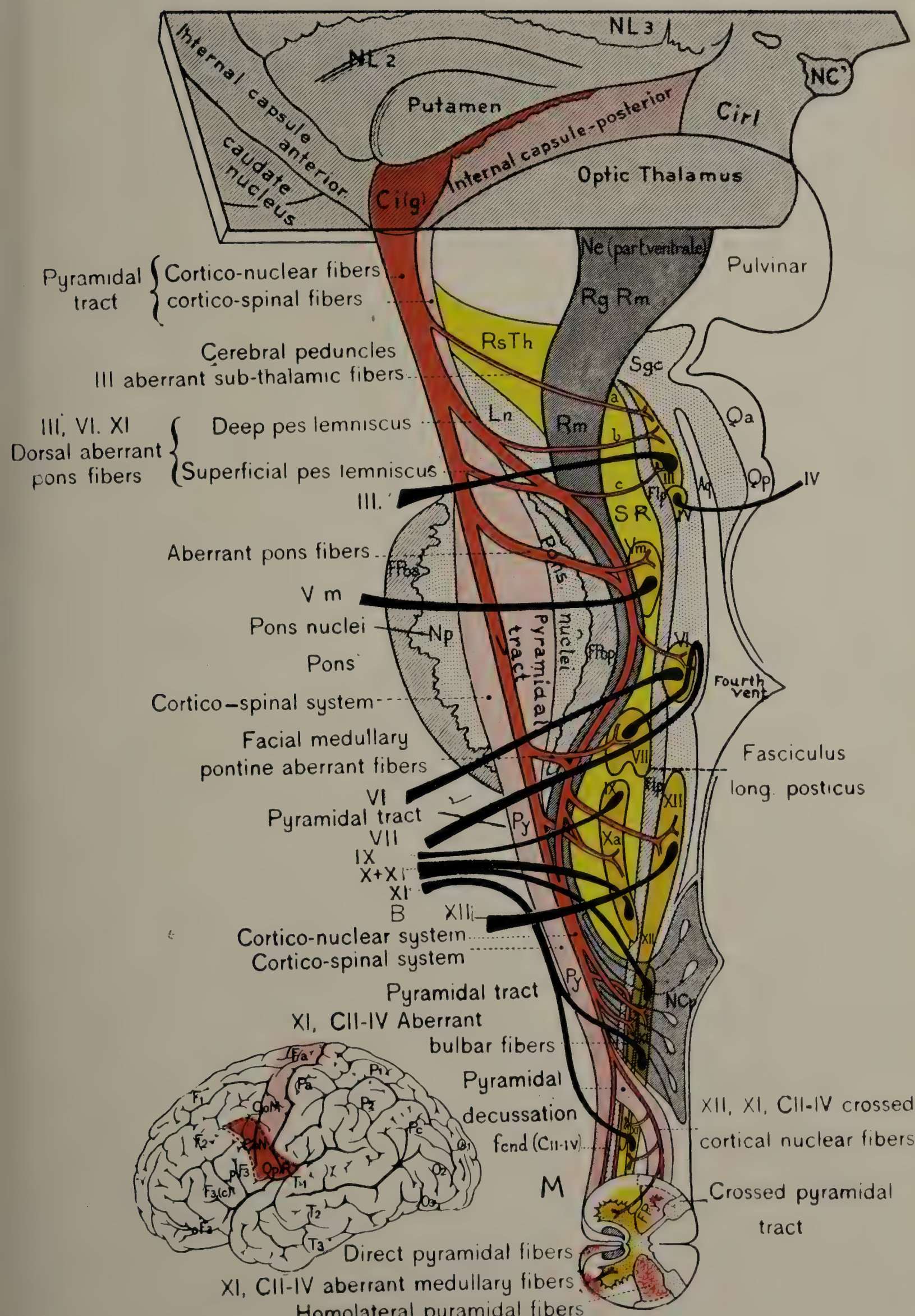
Abbreviations: *Aq*, aqueduct of Sylvius; *Cia*, anterior segment; *Ci(g)*, knee; *Cip*, posterior segment; *CirP*, retrolenticular segment of the internal capsule; *CoM*, cortico spinal system (pink); *CON*, corticonuclear system (red) of the pyramidal tract, *VP Flp*, posterior longitudinal fasciculus; *FPba*, *FPop*, anterior and posterior pontine fibers; *FPyc*, crossed pyramidal tract; *FPyd*, direct pyramidal tract; *fPyh*, homolateral pyramidal fibers; *fab*, medullary aberrant fibers; *fap*, pontine aberrant fibers; *fabp*, medullary, pontine aberrant fibers; *fasth*, subthalamic or superior pontine aberrant fibers; *fcnc*, *fcnd*, crossed and direct cervical corticonuclear fibers; *Ln*, locus niger; *NC*, caudate nucleus; *NC'*, tail of caudate nucleus; *NCp*, nuclei of posterior columns (Goll and Burdach); NL_1, NL_2, NL_3, the three segments of the lenticular nucleus; *Np*, pontine nuclei; *PLp*, deep pes lemniscus; *PLs*, superficial pes lemniscus or aberrant pontine fibers; *a*, *b*, *c*, the three modes by which the pontine aberrant fibers enter the third nerve nuclei; *Pul*, pulvinar; *Qa*, *Qp*, anterior and posterior corpora quadrigemina; *RgRm*, region of the median lemniscus; *Rm*, the median lemniscus; *Sgc*, subependymal gray substance; *SR*, reticular formation, colored yellow; *Th*, thalamus; V^4, fourth ventricle; *VP*, pyramidal tract; *III*, *IV*, nuclei and root fibers of the oculomotorius and trochlearis nerves; *Vm*, nucleus and root fibers of the trigeminus (masticators); *VII*, *IX*, *XI*, *XII*, nuclei and root fibers of the facial (*VII*), glossopharyngeal (*IX*) spinal accessory (*XI*), and hypoglossal (*XII*), nerves; *Xa*, anterior root of the spinal vagus; *X–XI*, root fibers of the spinal vagus.

The encephalic trunk and its three great divisions, cerebral peduncles (*P*), the pons (*PO*), the medulla (*B*), is shown in sagittal section with its connections with the internal capsule (*Cia*, *Cig*, *Cip*, *Cirl*) and the thalamus (*Th*) in part, and with the spinal cord (*M*) for the other part. *The anterior segment* contains the pyramidal tract (*V.P*) (and is made up from above below) of the foot composed of the fibers passing through the knee (*Cig*) and the posterior segment (*Cip*) of the internal capsule, the anterior segment of the pons with the pontine nuclei (*Np*) and the anterior (*FPoa*) and posterior (*FPop*) pontine fibers and the anterior pyramid of the medulla (*Py*); and the *posterior segment* or *tegmentum*, is separated above by the locus niger (*LN*), below it abuts the posterior pontine fibers (*FPop.*), and the pyramid of the medulla (*Py*).

The *tegmentum* is made up of (1) a gray substance the *reticular formation* (*S.R*)—colored yellow—which extends from the subthalamic region (*RsTh*) to the lateral columns of the cord and contains the motor nuclei of the cranial nerves—colored orange—disposed in two longitudinal columns; the anterior column includes the motor nuclei of the trigeminus (*Vm*), the facial (*VII*), the nucleus ambiguus or the anterior spinal vagus nucleus, (*Xa*); the posterior column includes the nuclei of the (*III*) and (*IV*) pair, the nuclei of the (*VI*), the long nucleus of the hypoglossal (*XII*) and the long inferior nuclei or spinal accessory (*XI*); (2) of the longitudinal fibers of which a part group themselves in fascicles more or less compact to form the *posterior longitudinal fasciculus* (*Flp*) and the *median lemniscus* (*Rm*). The *median lemniscus*, represents an important sensory pathway which terminates in the thalamus (*Th*) and takes part of its origin from the posterior column nuclei (Goll and Burdach). The *posterior longitudinal fasciculus* consists of an important association fiber system between the nuclei of the oculomotorius, the spinal nuclei, the facial, to serve the important functions of lateral movements of the head, eyes, and trunk for maintaining the static equilibrium of the body. (See Plate IX.)

[1] From Déjerine. Sémiologie des Affections du Systéme Nerveux.

PLATE I


NL 3
NL 2
Internal capsule anterior
caudate nucleus
Putamen
Internal capsule-posterior
Cirl
NC'
Cilg
Optic Thalamus
Ne (part ventrale)
Pulvinar
Rg Rm
RsTh
Sgc
Ln
Rm
Qa
Qp
IV
SR
Vm
Pyramidal tract
Np
Pons
Fourth Vent
VII
IX
XII
Xa
Py
NCp
Pyramidal decussation
fcnd (CII-IV)
M
Pyramidal tract
Cortico-nuclear fibers
cortico-spinal fibers
Cerebral peduncles
III aberrant sub-thalamic fibers
III, VI. XI Dorsal aberrant pons fibers
Deep pes lemniscus
Superficial pes lemniscus
III.
Aberrant pons fibers
V m
Pons nuclei
Cortico-spinal system
Facial medullary pontine aberrant fibers
VI
Pyramidal tract
VII
IX
X+XI
XI
B
XII
Cortico-nuclear system
Cortico-spinal system
Pyramidal tract
XI, CII-IV Aberrant bulbar fibers
Fasciculus long. posticus
XII, XI, CII-IV crossed cortical nuclear fibers
Crossed pyramidal tract
Direct pyramidal fibers
XI, CII-IV aberrant medullary fibers
Homolateral pyramidal fibers

Pyramidal Tract.—The corticospinal portion (*CoM*) of the pyramidal tract (pink) takes its origin in the ascending frontal convolutions (*Fa*), descends in the posterior segment of the internal capsule (*Cip*) and in the anterior portion of the cerebral peduncle. The corticonuclear (*CON*) portion (red) arises from the Rolandic operculum (*OpR*) at the foot of the ascending frontal convolution and the second frontal, descends through the knee (*Cig*) of the internal capsule and along the lenticular border of the posterior limb of the internal capsule (*Cip*), then to the cerebral peduncle, where it divides. One portion of the corticonuclear contingent follows the course of the corticospinal fibers to the anterior segments of the pons and the anterior pyramidal region of the medulla. Another portion, the system of the *aberrant fibers of the pyramidal tract*, breaks up at different levels, penetrating the tegmentum, descending in the median lemniscus (*Rm*) and the interolivary portion of the medulla to make up the fibers entering the motor nuclei of the cranial nerves.

In the medulla portions of the corticonuclear fibers fuse more or less with the aberrant fibers, pass by the inferior hypoglossal and spinal accessory, and behave like the corticospinal fibers. One part descends in the direct pyramidal (*FPyd*), another traverses the middle line with the crossed pyramidal (*FPyc*), a third descends as the homolateral pyramidal (*FPyh*) and the homolateral cord. All of these fibers end finally in the motor nuclei of the anterior horns of the cord, functioning the movements of rotation, flexion, and bending of the head on the neck (C_{II} to C_{IV}).

The aberrant fibers of the pyramidal tract which detach themselves from the corticonuclear contingent are: (1) Some inconstant fibers in the subthalamic region, the *subthalamic aberrant* fibers (*fasth*) destined for the oculomotor nuclei (*IIIa*) and to the anterior corpora quadrigemina. (2) In the region of the cerebral peduncles the *aberrant pyramidal fibers proper*, deep pes lemnisci (*PLp*), and superficial (*PLs*), which leave the nuclei of the oculomotor nerves (*b* and *e*), then descend in the median lemniscus (*Rm*) and contain fibers for the (*VI*) and (*XI*) nucleocortical *oculorotary* and cortical *cephalorotary* fibers. (3) In the pons regions the *pontine aberrant fibers* (*fap*) which reinforce the preceding in the median lemniscus to enter the motor nuclei of the trigeminus (*Vm*), the hypoglossal (*XII*) and the spinal vagus (*Xa*). (4) In the neighborhood of the pontomedullary sulcus, the *medullopontine aberrant* fibers, which are constant and well developed, reinforcing the preceding and destined to the nuclei of the facial (*VII*), the mixed nuclei of the glossopharyngeal (*IX*) and the spinal vagus (*Xa*), and the hypoglossus nucleus (*XII*). (5) In the medulla, *medullary aberrant fibers*, homologues of the homolateral pyramidal fibers, inconstant fibers which descend the length of the periphery of the homolateral anterolateral cord and terminate in the superior cervical cord nuclei of the rotators and flexors of the head and of the neck.

These aberrant fibers present great individual variation in their development—as seen in the different degeneration pictures studied by the Marchi method. Their chief characteristic is their descending pathways in the tegmentum, particularly in the median lemniscus and the anterolivary space in the medulla. This disposition explains why these fibers can be affected or spared by peduncular lesions, according to the site above or below, or whether it involves the anterior or posterior (tegmental) regions. The mode of termination of these corticonuclear fibers, possibly direct, possibly by intercalated neurones, in the motor nuclei of the cranial nerves has not yet been elucidated (Dejerine).

The field of vision should be tested with avoidance of suggestive factors. This may be done with a perimeter (which frequently introduces the error of suggestion) or the rapid finger test employed. The patient sits about three feet from the examiner. Each eye is tested separately. The patient puts his hand over his left eye, and looks fixedly at the examiner's left eye, the right eye being closed. With the left hand held midway between patient and examiner some distance to one side, it is then gradually brought toward the middle line, the fingers being waved slightly. The patient is directed to say "now" as soon as he catches sight of the slightly waving fingers. Four axes should be tested. Variations from the examiner's own fields can be noted. A square of white paper (1 cm.) on a gray card may be used instead of the fingers. For a general test of the color fields squares of different colors may be used. The most striking features to be sought for are hemianopsia, temporal or nasal; concentric limitations, irregular limitations, quadrant hemianopsias, psychic blindness and scotomata. Hemianopsia should *always* be searched for. It may be done rapidly by use of the usual finger-sight test and if there are any anomalies careful chartings by a perimeter should be made. Scotomata are sought for in the same manner. Single perimetric tests are usually not sufficient. Frequent examinations, especially for the different color fields are necessary, especially to trace advancing changes. For careful perimetric work the methods of Bjerrum should be followed out.[1] A useful method of making perimetric charts is by means of a modified Bjerrum screen. A large sheet of white paper 12 to 15 inches is covered with a thin black cloth or black paper and both fastened to a board. A small thumb-tack in the center can serve as a fixation-point for the patient's eye. The test object is carried on the blunt end of a steel pen, 0.5 cm. in length which is fixed transversely across the dark end of a metal bar so that its point projects about 1 mm. on the opposite side. The test object thus fixed on the end of the rod is moved slowly from the blind region across the screen until the patient sees it, then the carrier is simply pressed against the board so that the pin penetrates the cloth and marks the paper beneath it. A large number of observations can thus be quickly made and they can be easily transferred to a perimeter chart.

Perimetric plotting of the macula fields may be desired.

In all cases the fundus should be examined. A knowledge of the eye-ground changes is essential for good neurological investigation. Works on ophthalmology must be consulted for the many anomalies, but the most important to be observed are signs of pressure, of atrophy, of retinal hemorrhage or congestion, and irregular pallors (temporal pallor in multiple sclerosis), edema, choroiditis, retinitis, venous congestion, arterial occlusions, etc. Optic atrophy may exist without

[1] See Walker: Arch. of Ophthalmology, 1915, p. 369. Cushing and Walker: Brain, 1915, p. 341. Cushing, H.: Brain, 1921, **44**, 341 (for Temporal Lobes).

blindness. (See Fig. 2, Plate IX, page 368.) Cocaine 2 per cent will dilate the pupil to advantage.

Ocular Malingering.[1]—The accurate examination of sight requires good psychological acumen as well as skilled ophthalmological technic for there is room for intentional malingering or for unintentional hysterical falsification on the part of the subject. The neurologist must have a ready appreciation of what an eye ought to do and should be able to rapidly vary his tests so that the subject is taken as much as possible off his guard. The previous history of employment or other use of the eyes is important.

Refractive defects because of their universality should be given no more than a limited valuation. There is a reserve of accommodation which may not be apparent at once if the subject has worn correcting glasses, but which may be tested by approximating the lids, the vision being thus appreciably sharpened. There should normally be a reserve of accommodation sufficient to make a hyperopia of four or five diopters compatible with 20/100 vision. A myopia in which the error is not greater than three diopters, in which the reading of No. 1 type proves the macula sound, has room for considerable improvement under good hygienic conditions even though distant vision is below 20/200.

The routine examination may begin with the momentary use of a flashlight which will reveal important external disease, opacities of the cornea and the pupillary reaction. It can be easily and inadvertently determined whether the patient customarily fixes with one eye or both. A preliminary retinoscopy, which can be performed merely by having the patient look off into the distance, gives an idea of what the patient's unaided eye is capable of and the kind of lens that may be needed. It is important to watch the patient rather than the test cards. The manner of conducting the examination is important in order to provide the unexpected, allow the test to proceed rapidly and to keep the details of the test from the patient's knowledge, at the same time that one maintains an attitude of friendliness and apparent carelessness. Ignorance of the visual angle on the part of most subjects should be made use of. A mirror may be used to reflect a reversed test-card so that the distance is increased without the subject's knowledge. Insistence upon some one point may throw the subject off the track and lead him to a much freer and more correct report upon other far more important details.

A flashlight presented to both eyes will prove whether one eye is totally blind or not because of the presence or absence of squint. Then a 5-degree prism interposed before the supposedly good eye will move with the good eye if the other is blind but will not move at all if binocular fixation is usual. Binocular vision may also be proved by the Maddox double prism. This may be put before the good eye

[1] Alger: Ocular Malingering, New York State Jour. Med., December, 1918. Jones and Llewellyn: Malingering, Blakiston, 1917.

so that two lights are seen one above the other and if the other eye is good three lights will be seen when this is uncovered. Then a card may be passed gradually in front of the double prism, while the patient states the disappearance of either flame. The seeing of two flames when the prism is half covered proves the use of both eyes. An attempt to state that one light disappears before the other as the card is carried over the whole prism proves the subject's statements incorrect. The overlapping of two cards caused by a 4-degree prism will be confusing if the eye is good but otherwise cause no inconvenience.

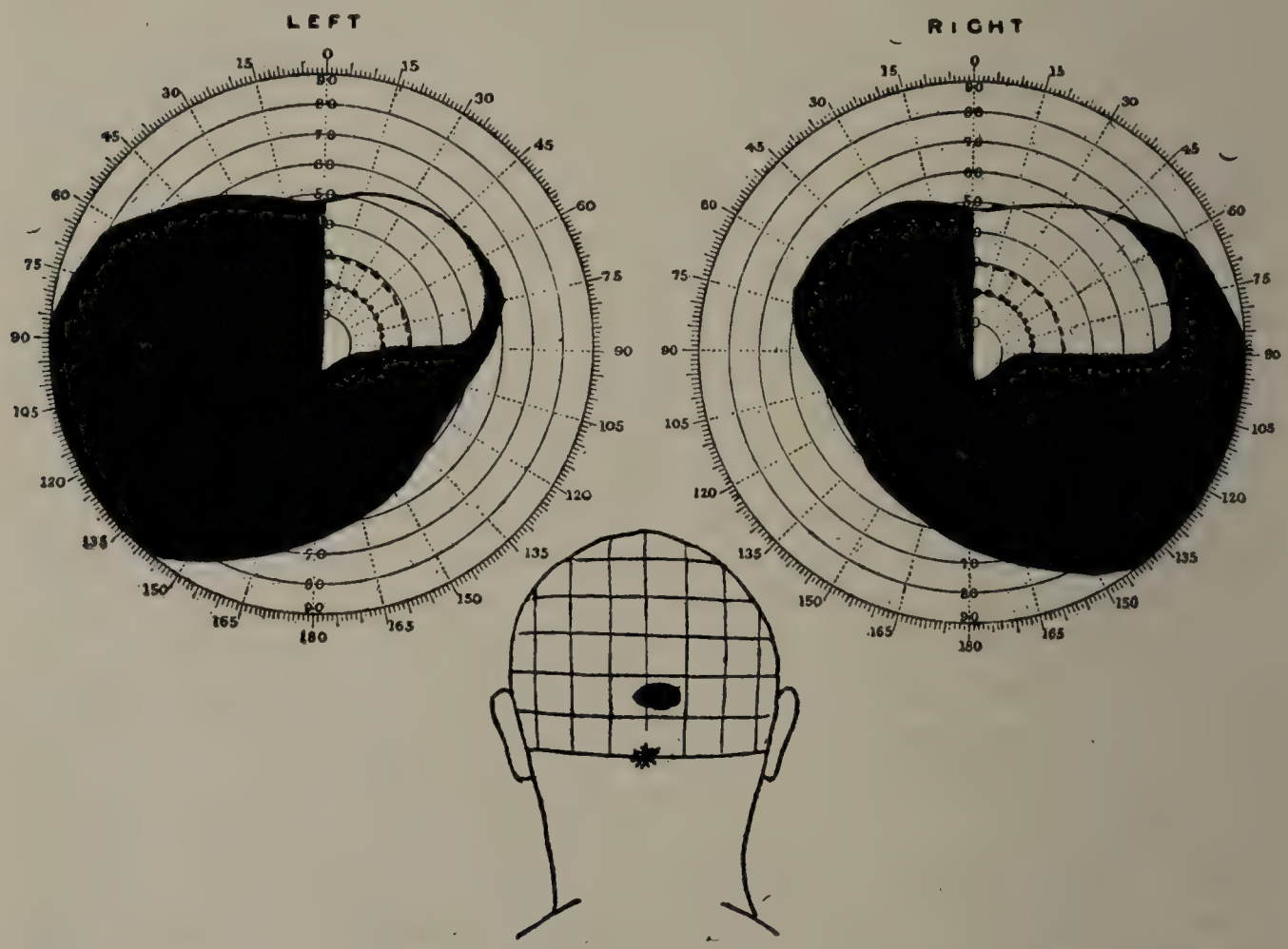


FIG. 8.—Disturbance of vision from bullet wound of posterior end of the calcarine fissure. (Lister and Holmes.)

A more positive form of testing is to block out the good eye with a plus glass before the patient is aware of it. The patient may read the test card through the Stevens phorometer with both eyes open when he will read one card up and one down. Then his attention may be distracted and the prisms reversed when he will try to read as before without realizing that he is using the other eye. The red and green glass test is useful only if conditions are just right. More simply a red glass may be put before the good eye and the impression given that the subject should see red with this eye and not with the other. Then his ability to read red letters on a white card may be tested, which would, however, not be possible through the red glass and could be done only with the other eye. The ordinary stereoscope can also

be used over the patient's distant glasses, care being taken to detect the possible closing of the alleged bad eye.

To test the relative quality of both eyes fine type bar reading is useful with a pencil held vertically four or five inches in front of the patient. Unless both eyes are reading there will be hesitation and twisting of the head. This test can also be applied to distant vision by means of an upright to conceal one or more letters in each line from each eye while all are visible to both. The diploscope of Remy permits a row of letters to be seen apparently with both eyes but in fact the letters of either side are seen only with the eye of the opposite side. The unsuspecting patient will insist upon the contrary. Several devices have been used to make this test applicable to distant vision as well. Some observers use one consisting of two short adjustable cylinders which the patient can hold to his eyes. The aperture before the good eye may be closed while the patient is led to suspect that the poor eye has been blocked and the sensation will be that he is using the good eye. If the two trial cards used are exactly alike they will usually be fused and appear as only one card. Then extra letters may be interpolated at different places on each card and these will often be read as if on one card thereby proving not only binocular vision but its degree.

Third, Fourth, and Sixth Cranial Nerves.—The functions of the third, fourth, and sixth cranial nerves are conveniently tested, first together and later separately. Free movements of both eyes outward (external rectus) indicates integrity of the sixth cranial nerve. If the eyes can be moved freely, amply and equally upward, downward, and inward, the third and fourth cranial nerves are usually intact.

CHIEF TESTS FOR EYE MUSCLES.

Test movement.	Muscle involved.	Innervation.	Synapse.	Chief lesions. (See under Cranial Nerves.)
Raise upper lid	Levator palp. sup.	Third nerve	Midbrain	Faint drooping; Müller's muscle sympathetic.
Look up, and in	Rectus superior	Third nerve	Midbrain	
Look up, and out	Obliquus inferior	Third nerve	Midbrain	
Look in (converge)	Rectus medialis (int.)	Third nerve	Midbrain	
Look to side	Rectus externus (lateralis)	Sixth nerve	Midbrain	
Look down, and in	Rectus inferior	Third nerve	Midbrain	
Look down, and out	Obliquus superior	Fourth nerve	Midbrain	
Convergence—vision	Ciliaris	Autonomic	Cervical plexus; Cervical ganglion	

Having seen if the eyes move freely in all directions, nystagmus is tested for under the same conditions. It consists of a slow movement of the bulb in one direction, with a rapid jerk back in the opposite direction. Notes on the direction of the slow and rapid movements should be made. They are of great value in determining labyrinthine and cerebellar disturbances. Nystagmus may be present on central

fixation, or only become apparent as the eyes are directed to one side. Slight tremors of the globes on extreme lateral position may be of small diagnostic importance. Nystagmus should be tested for in the vertical and horizontal and also in oblique axes. Rotatory nystagmus may be looked for. Optic nystagmus,[1] revolving disk, also. (See later.)

In the presence of a nystagmus, certain supplementary tests are advisable. The most important are the turning stool, and hot- and cold-water and pointing tests.



FIG. 9.—Paralysis of the fourth nerve. Inability to look down. It will be noted that as the eyeball does not turn downward the eyelid does not descend, at the same time the patient can close his eyes when told to do so. (Russel.)



FIG. 10.—Cerebral syphilis. External rectus palsy, right eye.

First the patient is seated on a revolving office chair, and turned to the right or left ten revolutions, with moderate rapidity. On arresting the revolutions careful note should be made of the direction of the nystagmus in each eye, and also the length of time it persists. The head may be held erect, and bent forward or backward to test each semicircular canal. Specially constructed chairs are needed for careful work. (See Testing of Labyrinth, p. 407.)

Hot or cold water is syringed gently into the external auditory meatus of each ear. A large bulb syringe is used. Cold causes a definite rotatory nystagmus toward the ear not irrigated. The patient also has vertigo and marked disturbance of equilibrium. If hot water be used the quick nystagmic movement will be toward the ear irrigated, and the ataxia is different. Labyrinthine trouble or cerebellar disorder involving the vestibular apparatus cause modifications in the nystagmus. (See Pointing Tests and Vestibular Tests.)

Diplopia is next tested for. The patient should be asked if he has ever seen double and a single light, or one finger held to the right or left, up or down, used to test if double vision exists. Should it be

[1] Fox and Holmes: Optic Nystagmus and Cerebral Lesions, Brain, 1926, **49**, 333.

present the position of the images in relation to one another should be noted; whether they separate or approach as a candle is farther or nearer, and a red glass should be placed before one or the other eye to determine the location of the images, and their relative position.

Conjugate deviation tests are discussed under Diseases of the eye movements (p. 378.)

Monocular diplopia, seeing double with one eye, is occasionally met with. It is due to gross corneal or eye defects, occasionally in central scotomata, but usually it is a product of a psychical wish projection.

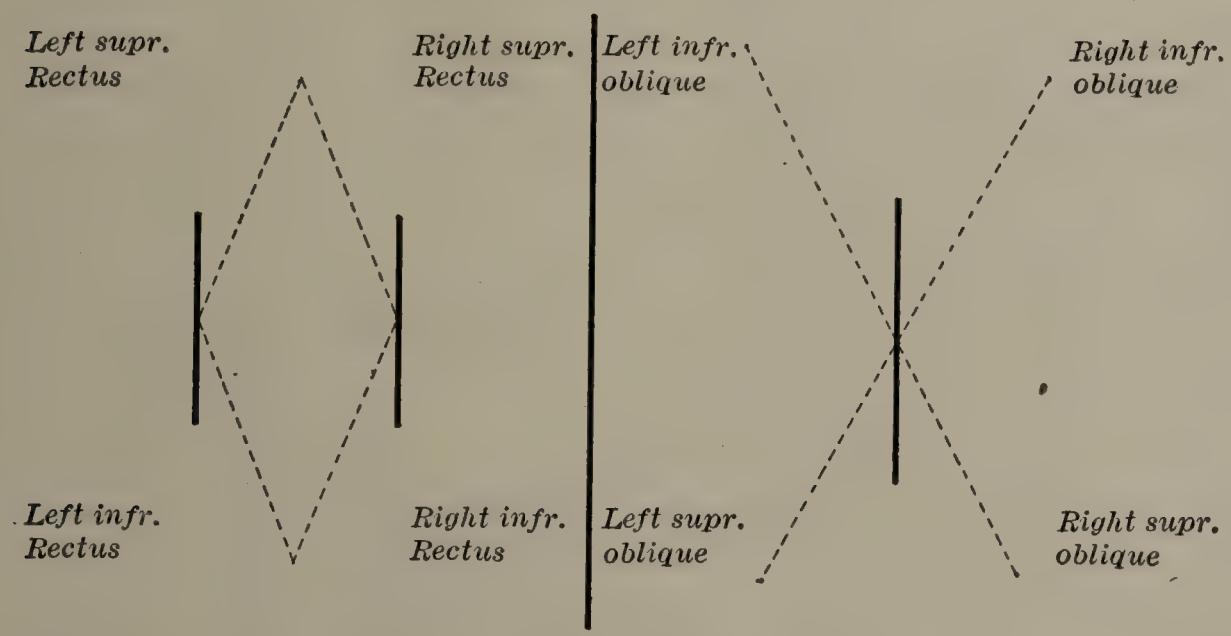


FIG. 11.—Werner's "artificial memory" for the double images in ocular paralyses (Ophthalmic Review, 1886). *A* shows the position of the images in paralysis of the recti muscles; *B*, in paralysis of the oblique muscles. The dotted lines indicate "false" images, the thick black lines "true" images. (Stewart.)

Pupils.—The size should be compared and noted. Right equals left right larger than left or *vice versa* and recorded thus: (r = 1 : r > 1, 1 > r) and a rough measure given, 2, 3, 4 mm., as the case may be; mydriasis myosis. The form and the presence of irregularities, changes in outline, oval, polygonal, ragged, and the implantation (ectopia) should be carefully noted. Particular attention should be directed to the estimation of changes due to drugs, to accidents or injuries, violent emotions, to inflammatory products, and to changes due to chest disorders or neck disorders or to other involvements of the cervical sympathetics.

The reactions to light should then be tested, at first with clear daylight, the patient looking at some distant object. In bed cases, electric hand lamps are very serviceable; with these the light is best directed somewhat obliquely. The reaction may be prompt and wide, prompt and restricted, slow, sluggish, diminished or absent. Each eye is to be tested separately, and then the consensual test applied by illuminating one eye only and noting the reaction in the other. Ready fatigability of the pupillary reactions to light should be tested.

Wernicke's hemiopic phenomenon should be sought for in hemi-

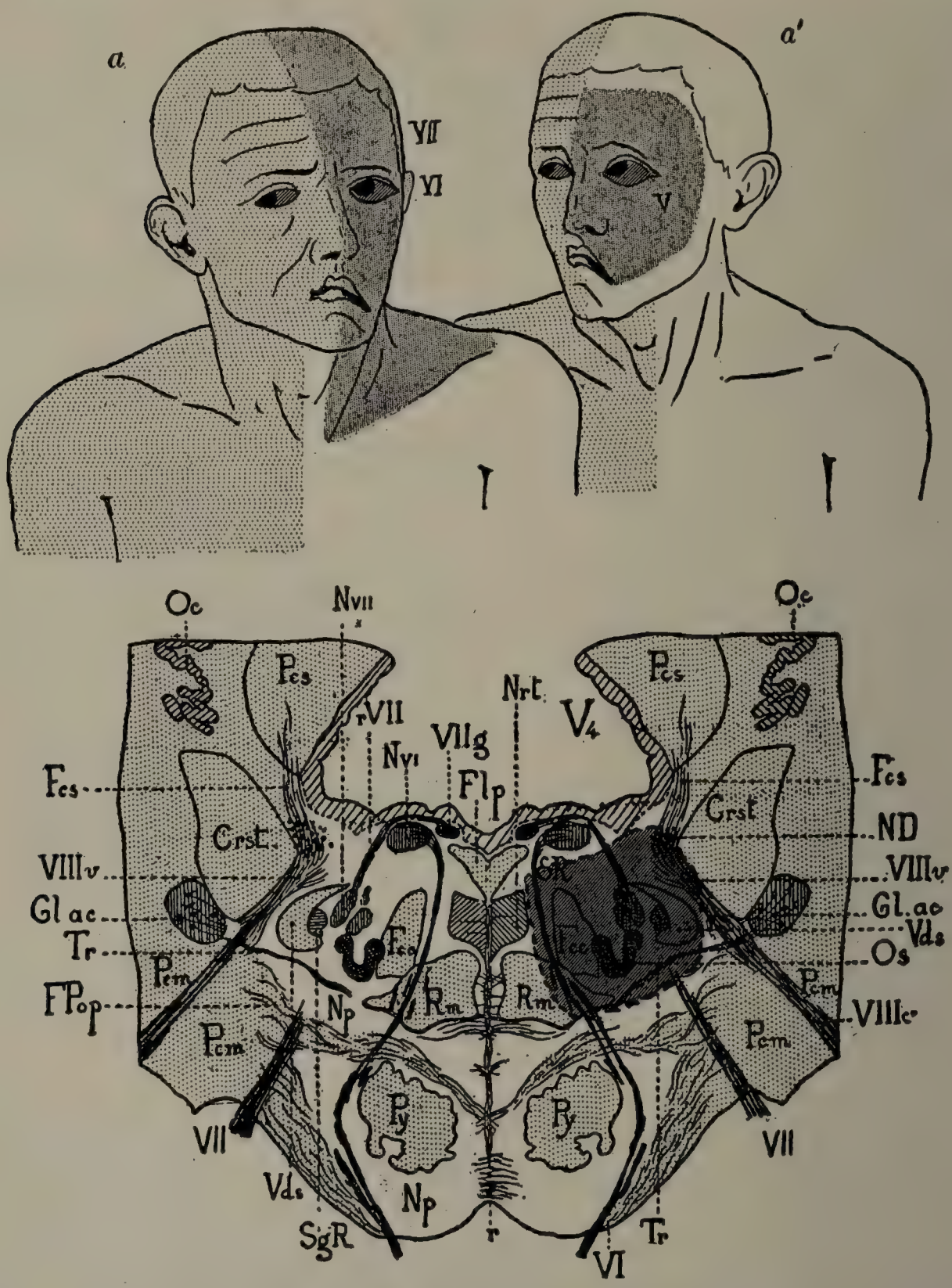


FIG. 12.—Pontine syndrome, with eye palsies of central origin and syringomyelic dissociation. There is here a crossed hemianesthesia with alternating paralysis of the VI and VII cranial nerves, anesthesia of the V nerve due to hemorrhage in the lateral and lower portion of the pontine tegmentum of the left side. The *right-hand figure* shows the hemianesthesia, dissociated as in *syringomyelia* (hemianalgesia and hemithermanesthesia due to lesion of the crossed sensory pathways of the lateral portion of the reticular formation. There is preservation of the tactile and postural sensibilities and of the stereognostic sense, because of the incomplete extension of the lesion to the median lemniscus (*Rm*). The *left-hand figure* shows (1) atrophic paralysis of the VII nerve with reaction of degeneration, lagophthalmia, drooping of the lips, loss of facial mimicry, paralysis of the entire left facial (VII) indicated (Fig. *a*); (2) anesthesia of the face, following involvement of the descending root of the trigeminus (see V on *a'*); (3) paralysis of the external rectus with convergent strabismus by reason of the overaction of the antagonists. Furthermore, there is a paralysis of the lateral movements of the eyeballs toward the left notwithstanding the integrity of the posterior longitudinal fasciculus (*Flp.*), of the nucleus of the VI and of the adjacent reticular formation. The lesion of Deiters' nucleus, and of the labyrinthine oculorotary fibers which unite Deiters' nucleus (*ND*) to the nuclei of the III and VI causes this. By reason of the overaction of the antagonists the patient looks to the right. (After Dejerine.) For abbreviations on the anatomical sketch see section on Midbrain and Plate X.

anopsias. In this the pupil does not react if the light falls upon the blind segment of the retina. It is indicative of a lesion in the optic neurones between the chiasm and the corpora quadrigemina.

Tests for the accommodation reflexes are then made. The patient looks at the finger as it is moved near to or away from the eye, and there is corresponding contraction or dilatation of the pupils. Here the reaction may be prompt or slow, with slight or marked amplitude. In blind people the request to look at their own nose and then across the room may bring out the reaction. A lost or partially lost light reflex, with unimpaired accommodation reflex, is known as the *Argyll-Robertson pupil.* It may be present in one or both eyes, and may be partial or complete. It is due to a dissociation of the sensorimotor reflexes, occasionally sympathetic, and may be present occasionally in a large number of disorders, although most frequently it is found in syphilitic disorders (Fig. 14).

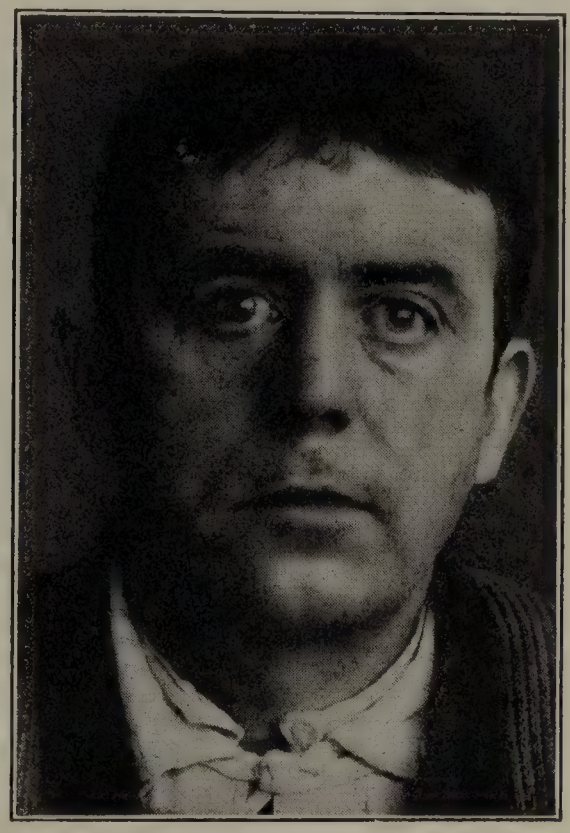


FIG. 13.—Inequality of pupils. Left pupil larger than right. Immobile. Cerebral tumor.

The mechanism is variously although not entirely satisfactorily explained because of the complexity of the afferent and efferent fiber tracts and their connections (Lewandowsky scheme, see Fig. 14). Marina's hypothesis of its peripheral origin (disease of ciliary ganglion) explains many of the tabetic and paretic cases, but does not explain some of the traumatic or mesencephalic cases. Cajal's scheme of the pathways is as follows:

1. Retinal neuron with its optic fibers ramifying in the anterior corpora quadrigemina.
2. The mesencephalic neuron with its axis-cylinder formation of the posterior commissure.
3. The neuron of the interstitial nucleus of the tegmentum (calotte) with its collaterals destined to the motor nuclei.
4. The neuron of the bulbar nuclei of the oculomotor communis et externus and of the patheticus with axis-cylinders going to the muscles of the eye. None of the schemes are free from some criticism.[1]

Spiegel assumes that the break takes place in the synapse between the splincter nucleus and its afferent neurons.[1]

A sympathetic reflex is tested by pinching the skin of the cheek or some other part of the body. The pupils dilate under the influence of painful stimuli. Psychogenic factors (chiefly fear) may occasion dilatation also. Adrenalin, homatropine, cocaine, pilocarpine may be used to obtain data.

[1] See discussion by Spiegel: Wien klin. Wchnschr., 1925, **189**, 216.

Ophthalmoplegia externa is the name given to a paralysis of the external eye muscles; *ophthalmoplegia interna* to those of the iris and ciliary muscle, the pupil is dilated and immobile to light and convergence. *Complete ophthalmoplegia* is found when all of the pupillary phenomena are absent and there is loss of all eye movements with ptosis.

Ptosis, or drooping of the upper lid (paralysis of levator palpebræ), (third nerve) mild drooping (Müller's muscle) (cervical sympathetic).

Henneberg's reflex, consisting in a slow spastic contraction of the orbicularis oris when the hard palate is stroked, may be mentioned as one of the reflexes of the cranial nerves.

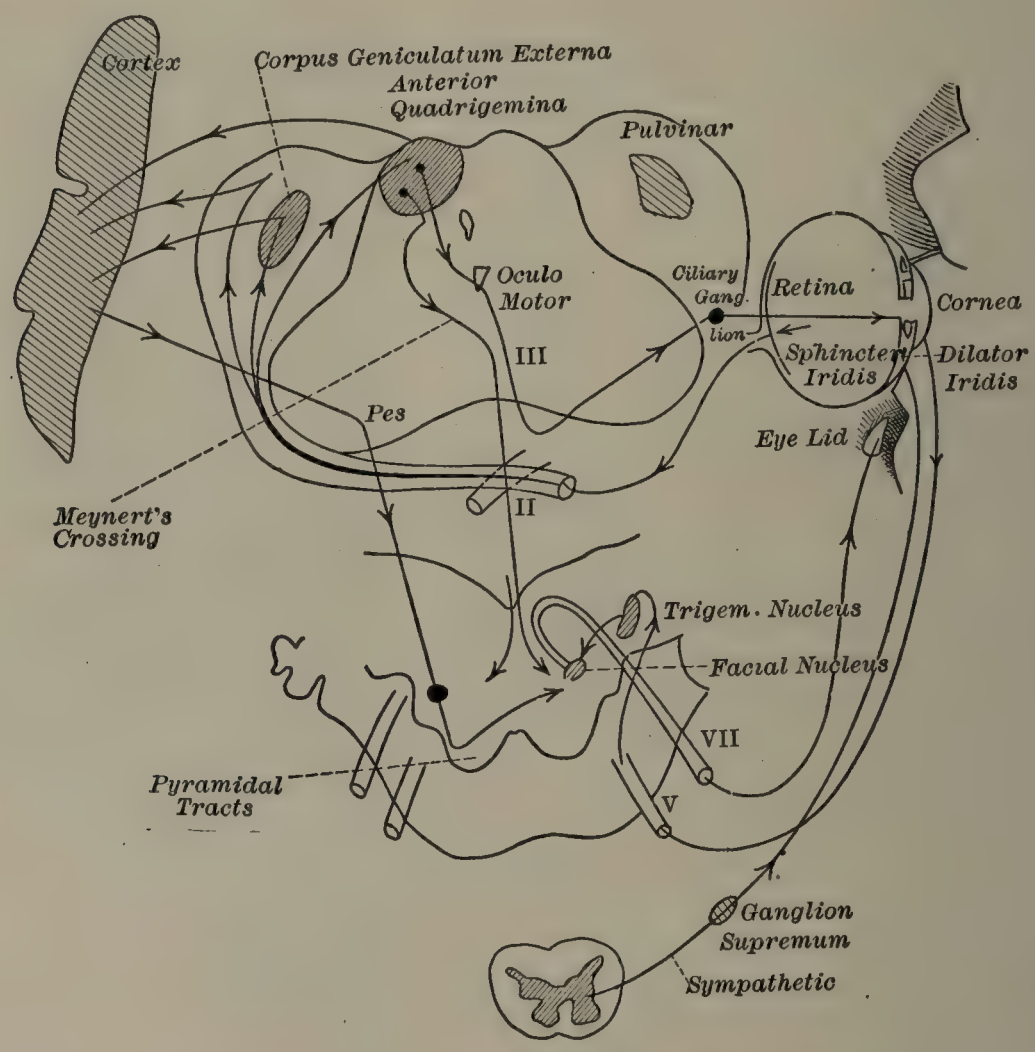


FIG. 14.—The paths of the chief eye reflexes: (1) Pupillary reflex: Retina; optic; ×; corpora quadrigemina; ×; oculomotor nucleus, oculomotor nerve; ciliary ganglion; sphincter iridis. (2) Closing eyes to light: Retina; optic; ×; corpora quadrigemina, Meynert's fibers; ×; facial nucleus; facial nerve; lid muscles. (3) Winking on approach of object: Retina; optic; ×; corpora quadrigemina or pulvinar, external geniculate; cortex; pyramidal tract; ×; facial nucleus; facial nerve; lid. (Same paths also in 2.) (4) Corneal reflex: Cornea; trigeminus; trigem. nucleus; facial nucleus; facial nerve; lid. (Lewandowsky: Funktionen d. Zentralnervensystem, p. 122, Fig. 29.)

The following conditions of pupillary activity are of chief clinical importance and will be discussed seriatim: (1) Complete immobility. (2) Reflex immobility. (3) Dilator or sympathetic paralysis. (4) Inequality and deformity of the pupil.[1]

1. Absolute or complete pupillary immobility. All stimuli via the vegetative nerves to the muscles of the iris are absent except a minimal

[1] Higier: Vegetative Neurology, Nervous and Disease Monograph Series, No. 27, 1919. Menninger: State of Pupils in Coma, J. Nerv. and Ment. Dis., 1927.

effect of the sympathetic. When the sphincter is paralyzed this influence upon iris activity is very slight. The pupils are dilated more than normally and are somewhat distorted. Since a certain degree of tone in the sphincter muscle is automatically maintained through the ciliary ganglion, destruction of this ganglion or its efferent neurones causes an almost maximal dilatation greater than is observed in disease of the nucleus or root of the oculomotor. Absolute immobility is observed in syncope, central lues, epileptic attacks, occasionally in hysterical dissociation, in war shock and in very marked fear from other causes.[1] Immobility in the mydriatic state is due in these cases to inhibition of cortical origin, immobility in the myotic state to an increase of the sphincter tone (Bumke).



FIG. 15.—Tabes with bilateral ptosis. Operated upon to hold eyelid open.

2. Reflex or isolated immobility is more difficult to explain. This is the Argyll-Robertson phenomenon. Its characteristics are one or both-sided failure to react to light thrown either on the same or other side, intact vision, narrowness, inequality and irregularity of the pupillary outline, retained reaction to accommodation and to convergence. The question as to the genesis of this condition and particularly the myosis which so often accompanies it is of great theoretical as well as of practical importance. It has been one of the most frequently discussed questions in neurology for decades.[2] It is dealt with fully in the chapters upon Diseases of the Cranial Nerves and Neurosyphilis.

3. The picture of sympathetic or dilator paralysis is known in physiology as Budge's symptom complex, in the clinic as Bernard-Horner's syndrome.[3] In addition to vasomotor and sweat anomalies, there is sinking in of the eyeball (enophthalmos) narrowing of the lid slit (m. orbitalis), lowering of the upper lid and raising of the lower lid (m. tarsi), narrowing of the pupil (m. dilator pupillæ) and retention of the psychic and optic nerve reflexes of the pupil. Cocaine (2 per cent) does not cause dilatation here.

In experimental section of the sympathetic the paralysis lasts only until the preganglionic part of the sympathetic chain again joins the preganglionic and the postganglionic the postganglionic.

[1] Guillain and Barré: Pupils and War Shock, Annals de Méd., 1917, No. 5, vol. v.

[2] Compare Lutz: The Light Pupillary Reflex, Arch. of Ophthalmology, 1918, vol. **47**, for a very complete discussion of this problem which cannot be entered into here more fully. Gozzano: Zeit. f. d. g. N. P., 1925, **95**, 644, for discussion of sympathetic reflexes.

3 Raeder. Brain, 1924, **47**, 1149.

The contrast between the very active sphincter contraction and the equally sluggish relaxation is typical of sympathetic paralysis (Bumke).

The localizing value for a diagnosis of sympathetic paralysis or irritation is self-evident. Both conditions indicate pressure upon the sympathetic chain due to a tumor, a traumatic lesion of the spinal cord (hematomyelia), or a tumor, degenerative process (gliosis) or an infective process in the upper dorsal segments of the spinal cord involving Budge's center. More rarely a lesion of the oblongata (thrombosis of the posterior inferior cerebellar artery, syndrome of Babinski-Nageotte) or a lesion of the spinal roots in the cervico-dorsal region (neuritis syndrome of Dejerine-Klumpke). The sympathetic lesion may be localized in its cervico-dorsal, bulbar or basal parts according to the accompanying symptoms (disturbance of the hand muscles, tongue, deglutition apparatus, trigeminus).

Pharmacological experiments with sympathicotropic substances during the last few years have added something to the differential diagnosis of sympathetic disease. For this cocaine and adrenalin mydriasis have been used. These tests can be easily performed provided there is a healthy uninjured, not inflamed conjunctival sac which is absorbing normally.

Cocaine in a weak solution (under 3 per cent.) stimulates the dilator mechanisms. Absence of cocaine mydriasis indicates weakness of the sympathetic. If a paralysis has been shown by this method in the absence of other signs, it becomes necessary to discover the location of the lesion for prognostic and therapeutic reasons. Whether it be preganglionic or postganglionic, above or below the superior cervical ganglion and whether it be in a place accessible to the surgeon. The very active hormone, adrenalin, will help to solve this question. A 0.1 per cent. solution dropped into the conjunctival sac normally produces no reaction. (Two drops are dropped in every five minutes for three times [Cords].) If the irritability of the dilator be increased as is the case in postganglionic disease dilatation of the pupil will result after fifteen minutes.

As Higier has shown, it is probable that after the nerve to the dilator is gone the contractile muscle tissue becomes more irritable. (Munk's isolation phenomenon, Langendorf's paradoxical mydriasis.) This adrenalin mydriasis is analogous to the rapid and maximal dilatation of the pupil after electrical, sensory or pyschic stimuli in animals in whom postganglionic section of the sympathetic has been experimentally obtained.

In many instances of disease in some way involving the region of the anterior or middle cranial fossæ (orbital disease, fracture of the base) adrenalin mydriasis has been observed. The explanation may be that the sympathetic fibers going to the eye come from the carotid plexus and join the first branch of the trigeminal nerve distal to the Gasserian ganglion. A combination of disease of the first branch of the trigeminal with the adrenalin mydriasis of postganglionic sympathetic paralysis can therefore be of great localizing value and make

an exact localization of the fracture, fissure or tumor of the base quite possible. Adrenalin mydriasis tests in psychotic patients are often vitiated by the very strong affective factors present.

Bilateral as well as unilateral mydriasis occurs in diseases of internal secretion (pancreas, diabetes, hyperthyroidism). This is a partial index of an increase in the irritability of the sympathetic system.

Deformity of the pupils (asymmetry, irregularity) and inequality (anisocoria) are sometimes congenital, usually, however, acquired and of somatic origin. Neurosyphilis is the most frequent cause. Sometimes local disease, as pressure upon the cervical sympathetic will cause the deformity. Transient inequality of the pupils is often found in severe unilateral migraine or in myalgias of the head and neck muscles with painful points in the neck (inequality due to pressure of the contracted muscle upon the cervical ganglion).

Hippus of the pupil and rapid mydriasis are of very slight clinical value.

From the point of view of vagotonia, the eye shows many signs worth clinical investigation. The vagotonic shows lacrimation and accommodation spasm which is relieved by atropine. The Löwi reaction (positive adrenalin mydriasis) in diabetes and Graves' disease is indicative of lowered sympathetic tone. If there be spastic conditions in other vegetative reflex arcs as gastric crises, psychogenic asthma, acute myosis may be found as an accompanying evidence of spasm. The opposite to vagotonia, *i. e.*, stimulating the sympathetic is shown in some cases of Graves' disease by paralysis of convergence (Moebius's sign) and exophthalmos (spasm of Müller's muscle).

Atropine acts slowly but persistently upon the pupil of elderly people, on the other hand rapidly but briefly in young people and vagotonics. v. Graefe's sign is an indication of an increased tone in the autonomic levator palpebræ. It usually follows upon pilocarpine instillation in young vagotonics.

Paradoxical Pupillary Reactions.[1]—Since Budge first reported the phenomenon in 1855 many observers including Schiff, Kowalewsky, Braunstein, Lewandowsky, Langendorf, Anderson and Meltzer and Auer have reported paradoxical dilatation in the homolateral pupil following lesions in the course of the cervical sympathetic from the origin of its preganglionic fibers in the upper thoracic cord to the postganglionic fibers in the long ciliary nerves. Because the pupil, on the side upon which the efferent dilator paths had been severed, exhibited greater power of dilatation than its fellow, Langendorf called the phenomenon paradoxical pupil dilatation. None spoke of the possibility that paradoxical effects might be induced by lesions of the afferent paths.

Byrne has found that lesions such as section, ligation, infection, alcoholic injection, etc. of one sciatic nerve were followed, after a period of

[1] Byrne, Joseph: Paradoxical Pupil Dilatation Following Lesions of the Afferent Paths, Am. Jour. Physiol., 1921, **66**, 113. Further Studies of Paradoxical Pupil Dilatation, etc., Am. Jour. Physiol., 1922, **66**, 369.

incubation averaging about ten or twelve days by hypersensitization to adrenalin, etc., of the pupil-dilator mechanism of the contralateral eye. After similar lesions of the brachial plexus the period of the incubation was shorter (about eight days) and the paradoxical effects occurred in the homolateral eye. It was found finally that lesions of any of the cranial or spinal sensory nerves induced paradoxical sensitization in the related pupil.

After section of one sciatic nerve paradoxical effects appeared in the contralateral eye about the twelfth day regardless of whether the divided nerve ends had been sutured together or not. It was found, however, that after the paradoxical period had passed off which, in the absence of infection, usually occurred from the fifteenth to the thirtieth day it could readily be reinduced by injury of the proximal segment of the divided nerve.

An intimate functional relationship exists between the efferent portion of the pupil dilator mechanism and every portion of the periphery through the medium of the sensory nerves. This relationship has important clinical significance since unilateral lesions of the sensory nerves are attended by well marked pupil inequality which in general follows the rule found for paradoxical dilatation. Because of this relationship great care must be taken to give due consideration to lesions of the sensory nerves in interpreting the significance of pupil inequality in surgical and medical conditions. Compare the Klumpke-Dejerine sign.

Psychical dilatation or contraction of the pupils may follow upon the idea of looking at an object at a distance or close to.

Pilcz reaction: tight compression of lids causes mydriasis. Non-operative with Argyll-Robertson pupil.

The Fifth (Trigeminus) Nerve.—The *motor* functions of the fifth nerve are most readily tested by having the patient move his jaw to the right and left. The examiner's hand, exerting contrary pressure, can determine differences in innervation (external pterygoid, temporals). The finger, wrapped in a towel, can be used to bite upon to determine variations in bite (masseter, temporal). Wooden tongue depressors are also serviceable, one on each side. By pulling weakness may be demonstrated. The state of hardness of the temporals and masseter muscles can be directly palpated. The jaw should be protruded (internal pterygoid).

The use of a tuning-fork of low pitch is useful in determining variations of function of the tensor tympani muscle.

In one-sided somatic paralysis of the fifth nerve, the opened jaw deviates to the paralyzed side by the action of the sound external pterygoid. The patient chews on the sound side. It may or may not be accompanied by sensory changes. Elevation of the eyelid on strong biting is a frequent associated movement.

The jaw reflex may be tested at the same time. With the mouth partly open a pencil or flat object is placed upon the teeth and lightly tapped with the hammer. There is a quick contraction of the mas-

seters and temporals, and usually an associated movement of closing the eyelids.

Sensory variations in the functions of the fifth nerve demand very careful testing by reason of its wide distribution.

The supraorbital, infraorbital and mental points should first be pressed upon to determine their degree of sensitiveness. Then the palpebral, conjunctival, and corneal reflexes should be tested. This is best done with a long pin with a globular glass head. With the patient looking away from the examiner, the palpebral margin is touched with the head of the pin, then the conjunctiva, and finally the choroid, and running along the globe over the cornea the effect is noted. Both eyes should be compared and the tear secretion noted.

TESTS FOR FACIAL AND JAW MUSCLE.

Movement tested.	Muscles involved.	Innervation.	Primary synapse.	Chief lesions producing loss of function.
Wrinkle forehead	Epicranius	Facial	Medulla	Peripheral injury;medullary inflammation; cerebral blocking.
Elevate eyebrows	Epicranius; corrugator	Facial	Medulla	Rarely psychogenic dissociation.
Frown	Corrugator	Facial	Medulla	Rarely psychogenic dissociation.
Smile (joke)	Sympathetic components; quadratus lab. sup.	Facial; sym.	Medulla; cervical chain	Rarely psychogenic dissociation; neck lesions.
Close eyes	Orbicularis palpebrarum	Facial	Medulla; cervical chain	Rarely psychogenic dissociation.
Sniff	Nasalis depressor septi	Facial	Medulla; cervical chain	Rarely psychogenic dissociation.
Pull mouth to side	Quadratus lab. sup.	Facial	Medulla	Rarely psychogenic dissociation.
Show teeth	Quadratus lab. sup; zygomaticus	Facial	Medulla	Rarely psychogenic dissociation.
Pull mouth down	Quadratus lab. infer.	Facial	Medulla	Rarely psychogenic dissociation.
Push up lower lip	Mentalis	Facial	Medulla	Rarely psychogenic dissociation.
Point lips; whistle	Orbicularis oris	Facial	Medulla	Rarely psychogenic dissociation.
Compress lips	Orbicularis oris	Facial	Medulla	Rarely psychogenic dissociation.
Move jaw laterally	Pterygoid, mandibular	Trigeminus	Medulla; cervical chain	Peripheral injury; Gasserian disease; local sure.
Bite on stick	Masseter, temporalis	Trigeminus	Medulla; cervical chain	Medullary.
Push jaw forward	Pterygoid, med. and lat.	Trigeminus	Medulla; cervical chain	Medullary.
Move ears (?)	Auricularis, etc.	Facial	Medulla	Rarely present.

The amount of tear secretion may be measured by hanging two small strips of litmus paper on each lower lid, by bending in the paper at the top so as to make a small ledge to hang. The rate of moistening of the two sides will show quantitative variations in the amounts secreted. The dryness of the two sides of the nose or mouth should be recorded. Salivation, thin or thick saliva.

The ordinary sensibility of the face should be tested first with a camel-hair brush—the two sides compared. Then with the point and head of a very sharp pin; then the skin should be pinched on each side and differences noted. Deep pressure over the malar, frontal, and jaw bones made to determine deep pressure sense, and the use of hot and cold test-tubes to learn if variations in thermal sense exist. Finally

a slowly vibrating tuning-fork should be applied to the bones of the head and face to determine their bony sensibility reactions.

The interior of the mouth and the surface of the tongue should not be neglected in these tests, and special care should be taken in outlining changes of sensibility in the ear areas, and within the auditory canal.

Careful inspection of the teeth should not be omitted, and anomalies of dentition carefully noted. (See Endocrine Status). The two sides of the bony structures of the face should be compared for bony atrophies (hemiatrophy) or hypertrophies (acromegaly).

The Seventh (Facial) Nerve.—This is chiefly a motor nerve. The state of the musculature at rest should first be recorded. The folds and the angles of the mouth and muscle-twitching carefully noted. The patient is then asked to wrinkle his forehead (frown), raise the eyebrows, close his eyes tight, show the teeth, whistle, and puff out the cheeks. In this way the musculature of the two sides can be compared. A joke will serve to bring out emotional reaction variations of the two sides. Some individuals can move their ears, or their scalp wittingly and reflex movements connected with these structures are of interest.[1] Mask-like postures and fine tremors or twitches involuntary mimetics (laughing and crying) should be noted.

Not only should the presence of paralysis or paresis be carefully noted, but spasms observed under voluntary and emotional stimuli. Their degree, character, and intensity should be carefully recorded.

Sensory, secretory, and motor changes are to be tested, and many differences are to be noted in the distribution of the palsies according to the location of the lesion of this nerve: (1) after its exit from the stylomastoid foramen, (2) within the Fallopian aqueduct, (3) between its emergence from the pons and the geniculate ganglion, or (4) within the pons. Also see taste testing. (See Fig. 20.)

The Eighth (Cochlear and Vestibular) Nerves.—Here two different nerves with separate yet correlated functions need to be tested. They are the cochlear nerves (hearing) and the vestibular nerves (equilibrium).

Hearing is roughly tested most satisfactorily by speech. Having first ascertained that the auditory canals are free from wax, the examiner, some six to ten feet away, pronounces six numbers, asking the patient, who has one ear closed, to repeat them after him. The voice is then raised or lowered, and the distance varied to determine the hearing capacity. The opposite ear is tested in the same manner and comparisons made. A watch-tick or tuning-fork may also be used. Careful tests with tuning-forks and whistles are needed in complicated cases. In mentally defective children hand clapping is useful as a test for hearing.

Bony conduction should also be tested. This is done by putting a vibrating tuning-fork over the mastoid, and the patient indicates

[1] Sherrington, C. S.: Reflexes Elicitable in the Cat from Pinna Vibrissæ and Jaws. Jour. Physiol., 1917, **50**, 404.

when he no longer hears it, the fork is then brought to the auditory meatus to test the air conduction. Rinné's test is positive, *i. e.*, air

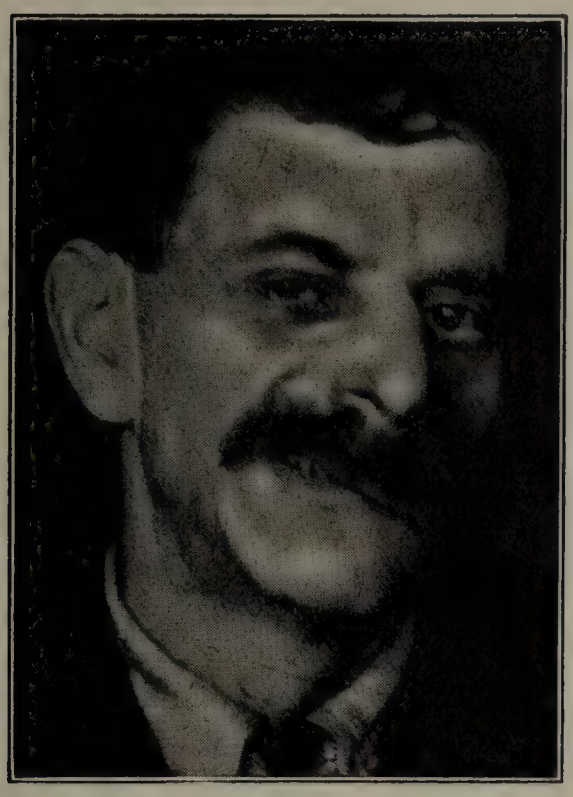


FIG. 16.—Facial palsy. Smiling.



FIG. 17.—Facial palsy. Whistling.

conduction present after loss of bone conduction is the normal formula: the negative Rinné indicates middle-ear disorder. The tuning-fork on the center of the forehead is heard in both ears equally under

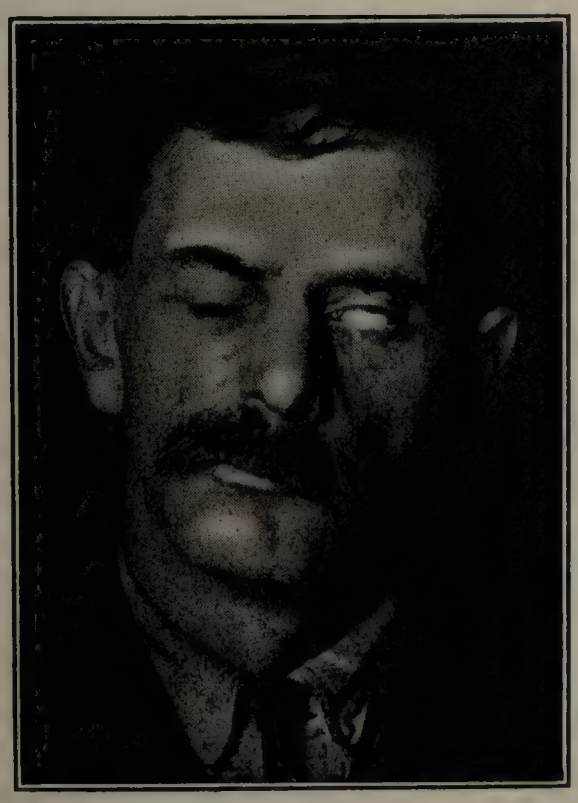


FIG. 18.—Facial palsy. Closing the eyes.

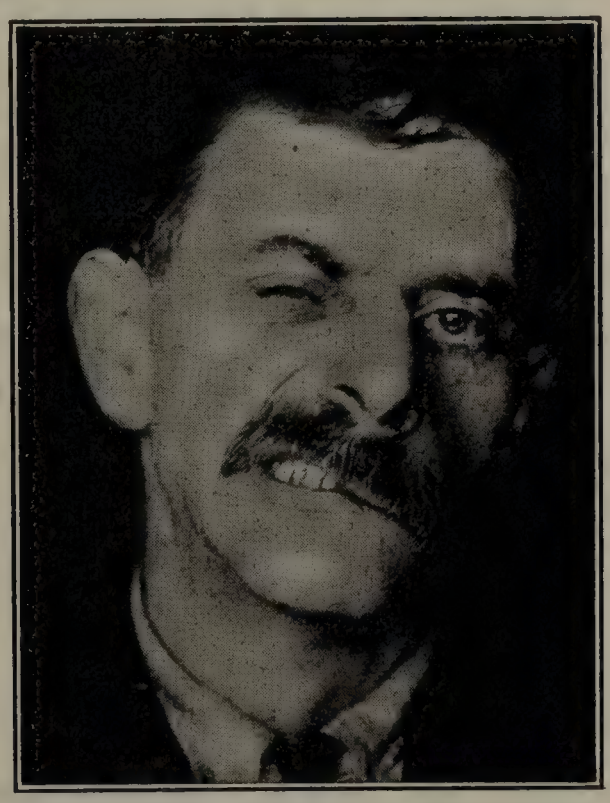


FIG. 19.—Facial palsy. Showing the teeth.

normal conditions (Weber). In middle-ear affections it may be heard unequally on the two sides. Positive Weber (*i. e.*, louder on affected side) with negative Rinné is largely indicative of middle-ear disorder. Deafness due to central disorder is usually associated with other local-

izing signs, yet it may be an isolated phenomenon of beginning tumor, encephalitic process, tabes, multiple sclerosis, etc.

Bony conduction tests, Rinné, Weber, Schwabach and Gellé are also of service in differentiating labyrinthine deafness,[1] and they are of value in testing the general bony conductivity of the cranial walls as well, they thus afford useful information concerning functional

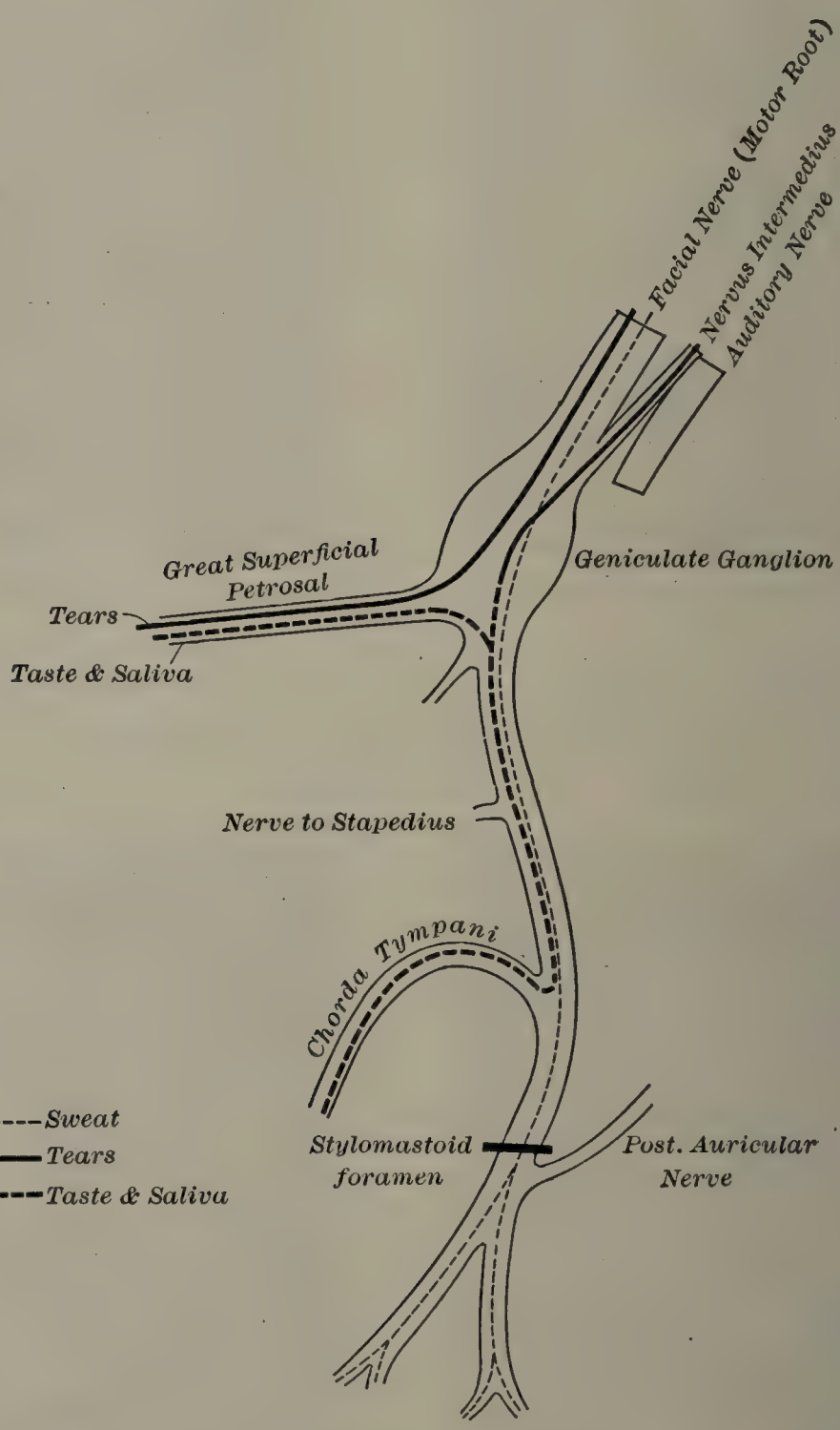


FIG. 20.—Diagram of facial nerve, showing course of secretory and of taste fibers of the accompanying glossopalatine and vegetative fibers. (Stewart.)

states of the trigeminus, which might falsely be referred to acoustic anomalies. The two possibilities, acoustic and deep bony sensibility should be differentiated in the tests. In this differentiation the usual mastoid site is inadvisable as it registers the acoustic reaction too massively. Other parts of the cranial walls should be chosen. A watch can be utilized for these tests as well.

[1] Bard: Du role de la sensibilité osseuse, Ann. de Méd., January and February, 1918.

Forks of very slow vibration are of value in determining the functional capacity of the stapedius muscles. Tests with continuous tone series following Bezold's methods are indicated in all complicated cases, since defects in lower or in higher tone perception may indicate a difference in the site of the lesion.

Certain patients show hyperacusis, tinnitus. Ringing in the ears is an extremely elusive sign. It is an evidence usually of middle-ear or of cochlear irritation. The sounds vary greatly. They may be beating, buzzing, or whistling, and may at times be the point of departure of illusions, or hallucinations. The pulsating types of tinnitus are usually associated with the heart beat. They are found in certain tumors, in aneurisms, or in anxiety states with cardiac irregularities. Continuous tinnitus, low-pitched or high-pitched, is the more common. The effects upon the tinnitus by lying down and also the effects of certain drugs, amyl nitrite, etc., are of service in differentiating the causes and probable site of a tinnitus. Tinnitus may be a psychogenic (autonomic) symptom; it may be otosclerotic.

Malingering for deafness has become frequent since the World War and numerous communications have been made upon the subject. The following notes may be of service in testing malingering.[1]

Most malingerers of deafness who are subject to the military draft know that deafness of a certain grade in either ear will exempt them. Complete bilateral deafness is therefore for various and obvious reasons is rarely, if ever, claimed. Two types of unilateral deafness are therefore assumed, *i. e.*, (1) deafness advanced, but not complete; and (2) absolute deafness.

The tests upon the combined results of which one may be usually able to base a definite conclusion are: (1) Weber's test; (2) the loud conversational voice test; (3) the binaural stethoscope test; (4) tests eliciting incongruous variations in response; (5) the noise apparatus, reading test (Lombard's test).

Kerrison's method of examination is about as follows: Only one registrant at a time is admitted to the room. Learn from him which ear is the deaf or deafer one, and whether he regards this deafness as partial or complete. If only one ear is complained of make a rapid test of the hearing of the better ear to determine if it is functionally sound or at least only slightly subnormal. This preliminary testing of the sound ear is essential to a proper interpretation of tests to be applied later.

Weber's Test. Apply a vibrating tuning fork of 256 double vibrations to the vertex of his skull, and if he refers the sound to his supposedly deaf ear, one may be favorably impressed as to his honesty. If he refers it to his sound ear, one is skeptical, and proceeds to the next test.

Loud Voice Test.—The registrant's eyes are now blindfolded. Requesting him to close with a finger his better ear which has already been determined to be approximately sound functionally, words and

[1] Kerrison: Transactions of Am. Otol. Soc., 1918. Jones: Value of Ear Examinations to Neurologists, Ibid.

numbers are repeated to him at first in low voice and then in progressively lower and louder tones. If, when a pitch and intensity at which he should be able to hear and interpret the words with the sound ear even though tightly occluded is reached he still states that he cannot hear it, he is an intentional malingerer. This is a useful test which will expose many malingerers. An alert individual, however, may throw this procedure out of court by stating that he hears the sound through his occluded normal ear.

Stethoscope Test.—For this the ordinary clinical stethoscope with funnel-shaped chest-piece is used. One ear-piece is completely occluded with wax. Occlusion with cotton will not exclude the sound. Standing behind the registrant, the stethoscope is adjusted with the occluded ear-piece in his "deaf" ear. Words in a low whisper are spoken into the funnel-shaped chest piece, which naturally he should hear perfectly. The stethoscope is removed for the ostensible purpose of trying some other test, and then replaced, the occluded ear-piece being this time placed in his sound ear. If he is able to hear now approximately as well as before, fairly sound evidence is present that his deafness is either assumed or grossly exaggerated. This is a fairly reliable test.

Tests for Eliciting Contradictory Responses.—The registrant's eyes are now uncovered, the sound or better ear is closed with a finger, and the "deaf" ear is subjected rapidly to the commoner classical tests, *e. g.*, hearing distances for watch, acoumeter, whisper or conversational voice; tuning-fork tests to determine lower tone range, etc. His responses, so far as he admits hearing in any of these tests, are carefully noted. Following this, he is again blindfolded and the same tests are repeated many times, fairly rapidly and in varying order. If he is a malingerer and has not in the first instance claimed almost total deafness, his responses will almost surely demonstrate incongruous and contradictory variations.

Lombard's Test.—This test, which requires a Bárány noise apparatus, is one of the most dependable means of determining absolute or very advanced deafness confined to one ear. It depends upon the fact that to the normal man the sound of his own voice is necessary to the proper regulation of its tone and intensity. It is carried out as follows: the noise apparatus is adjusted in his sound ear and its machinery started in order to accustom him to its grating noise. He is given a book and told to read aloud in his natural voice and not to stop reading when the noise instrument is set in action. As soon as the noise begins, a man whose opposite, or open, ear is profoundly deaf will at once raise his voice; and if his deafness is absolute, he may literally shout. The malingerer, on the other hand, claiming a one-sided deafness which is not real, will continue to read in an even tone or in a tone only slightly elevated. This is a test which a malingerer who has been coached may easily turn to his advantage. Otherwise it is one of the most useful at one's disposal. With a quiet room and sufficient time at his disposal, there are few malingerers who will be able to deceive a careful and experienced examiner. If the registrant claims absolute unilateral

deafness and his reactions to the commoner tests seems to justify this claim, he should raise his voice almost to a shout when a noise apparatus is operated in his sound ear. If he claims only partial but advanced deafness, his reactions to the various tests should be approximately constant. The examiner is able then, usually to detect the malingerer; but if the malingerer really suffer from some degree of impairment, it may be and usually is, quite impossible to determine even approximately his grade of deafness. And so it may happen that a man partially deaf may be misjudged.

Bilateral Deafness.—Naturally, as there are many cases representing all grades of bilateral deafness malingering may take the form merely of an exaggeration of an existing defect. If such a person were persistently to assume absolute or very profound bilateral deafness, it is difficult to say what tests would expose the deception. The time-honored tricks of attempting to ensnare him by a sudden and startling question, the use of insulting or disparaging remarks, etc., are mentioned in many test-books. Such crude methods are usually uncalled for, however, for the reason that the risks to the malingerer of such flagrant lying are too great. The pretense of partial though disabling deafness, on the other hand, though less likely to be exposed through the testimony of acquintances, is not so difficult to detect for the reason that the malingerer is burdened with the necessity of maintaining uniformity in his responses to the various tests. The surest means of exposure in such cases is the application of the classical tests first to one ear and then to the other, and then, after blindfolding him, repeating the same tests and comparing the two sets of responses.

Auditory-orbicularis Reflex.—When a loud sound is heard close by there is a tightening of the orbicularis palpebrarum fibers, chiefly those innervated by the cervical sympathetic. This is a useful test for psychogenic deafness. One ear being tightly closed with cotton, the patient faces the physician who with a magnifying glass of about two inches focus minutely observes the orbicularis muscle response to the loud honk of a bicycle or automobile horn which is blown just behind the patient or in a closely contiguous space. Practically no one can consciously inhibit this sympathetic reflex response.

Vertigo.—This symptom belongs largely to the symptomatology of the vestibular apparatus. Equilibrium is a complex adjustment of eye muscles, kinetic static sense and the muscle sense in general, involving the comparative integrity of a large number of neurones from the periphery to the coördinating centers, which latter are fairly satisfactorily proved to be in the cerebellum. The vestibular apparatus is the chief ganglion of the cranial end of this whole apparatus or system termed by Sherrington the proprioceptive system. The cerebellum is its chief center. A comprehensive anatomical survey of the pathways involved is to be found in the extremely valuable plate worked out by Dejerine. (See Plate X, page 386.)

Tests for Equilibration.—The more standard equilibration tests are the Romberg and the Babinski asynergic tests. The *Romberg* test

is obtained by having the patient stand erect with closed eyes, with heels and toes together. Under normal conditions there should be only a very slight swaying, but the person with well-marked Romberg sways sidewise, or backward, or forward, or may even fall if the feet are not spread apart. Slight degrees of Romberg, or unilateral localization of Romberg, may be brought out by having the patient stand on one or the other foot. Other observations of the Romberg can be made by having the patient bend forward or backward or sidewise, and in this way quantitative suggestions may be received as to the localization of the fiber tracts involved.



FIG. 21.—Asynergia of Babinski. (Schaller.)

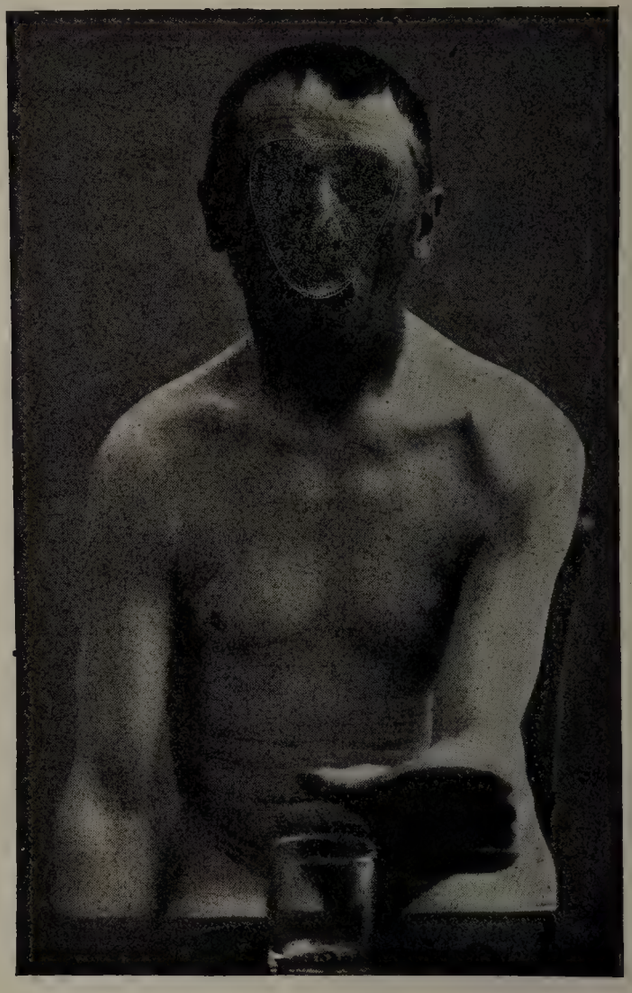


FIG. 22.—Dysmetria of Babinski developed on attempting to take hold of a glass. The fingers are held very far open. (Thomas.)

In certain cerebellar disturbances there is a special loss of equilibrium which Babinski has termed *asynergia*. This may be tested in a number of different ways. The more valuable are by means of walking. The patient usually has a great deal of ataxia and walks with difficulty. If supported on either side, it is noticed that in walking he shows a marked tendency to walk from underneath himself, so that his legs finally are put out far in advance of his center of gravity. The same type of loss of cerebellar equilibrium may be demonstrated by having

the patient stand erect and then slowly bend backward, making an *arc de cercle.* In the healthy condition the muscular adaptation bends the knees forward and the indvidual assumes a well-balanced position; but in the asynergic, the legs are held straight, the trunk is bent backward and the patient has a tendency to fall backward. A patient, on grasping a glass opens the hand far wider than needed. This is *dysmetria,* a sign of cerebellar reflex arc disturbance.

For the upper extremities a similar type of muscular incoördination shows itself in the typical *diadokokinesis* tests. These tests are made by having the patient perform certain alternating or opposing movements very rapidly, such as quickly pronating and supinating the hand in a semiflexed position, or making rapid piano-playing movements with the fingers, or sudden flexions and extensions of the arm on the forearm. In involvements of certain of the cerebellar tracts usually associated with equilibrium there is a marked inability to perform certain of these movements; they are done clumsily, irregularly and slowly, depending on the grade of sensory tract involvement.

For testing the equilibrium of the trunk the patient should be on the back, with the arms folded, and with the legs drawn up in the obstetrical position. Here considerable variation will be found. The healthy individual, if on a fairly hard foundation, can equilibrate fairly well; there is but very little swaying, while the ataxic patient sways considerably. Cerebellar patients also, and certain type of cases with fronto-cerebellar pathway disturbances show what is known as *cataleptic rigidity* in this position. After swaying for a certain length of time the legs and body steadily become fixed with sufficient rigidity to enable them to be photographed. This sign of cataleptic rigidity is considered by Babinski as characteristic of cerebellar defect.

In further testing equilibrium the *gait* occupies an important place. The patient should be directed to walk along a crack, or a straight line, first with the eyes open; he should be asked to suddenly reverse, inquiry being made if there are symptoms of vertigo in the reversion either to the right or to the left, for both sides should be tested, and then the patient should be directed to return with his eyes closed when, if disturbance of equilibrium be present, it will become intensified, and staggering to the right or to the left, forward or backward, will be noted (lateropulsion, propulsion, or retropulsion).

The patient should also be tested by walking on the toes, on the heels; he should also be asked to walk sidewise.

Inspection of the shoes (heel and sole) is highly desirable if there are minor modifications of gait. There are characteristic worn spots for various forms of motor weakness or stiffness.

Various modifications in gait are known as *ataxic* (reeling or drunken) as *spastic* (scissors gait); high stepping (*steppage); shuffling;* pendulum gait of hemiplegics; gait with little, short steps, *titubating, choreic, propulsion.* Stilted gait is a type seen in schizophrenia. Irregular gaits are noted in hystericals; here walking sidewise is useful in distinguishing a hysterical hemiplegic from a hemorrhagic one. The

hysteric tends to swing the leg out in the arc of a circle, the hemiplegic raises the pelvis and the toes tend to stick to the ground (spastic).

The subjective sense of giddiness is often extremely complicated. Certain patients complain of objects turning about them, in which case the direction of the moving object, in terms of the hands of a clock, should always be noted. Association of giddiness or vertigo with eye disturbances is very widespread.

The internal ear has such an intimate relation with the central nervous system that its study is of especial interest to the neurologist.[1] The Bárány ear tests are of particular value in making a differential diagnosis between labyrinth and intracranial lesions and in furnishing additional data for intracranial localization. Nystagmus and vertigo, with loss of equilibrium, associated perhaps with nausea and vomiting, may be produced either by a disturbance of the internal ear or by an intracranial lesion. In many instances the symptoms of internal ear disturbances and of a cerebellar lesion are identical. It is in such a differentiation that the ear tests are often invaluable. It not rarely happens that a careful neurologic study indicates a lesion of the cerebellum, whereas the ear examination, by giving additional data to the neurologist, demonstrates conclusively that one is dealing with a lesion of the labyrinth.

A differential diagnosis between peripheral and central lesions by means of the ear tests depends on certain general principles.

A peripheral lesion of the labyrinth or eighth nerve is suggested by the following, as given by Jones:

1. An impairment of the function of both the cochlear and kineticostatic labyrinth. If, for example, the hearing tests show cochlear deafness and the tests of the semicircular canals show that their function is also impaired, it immediately becomes probable that an end-organ lesion is present.

2. The history or presence of tinnitus; the absence of tinnitus does not necessarily indicate that the end-organ is not involved, but its presence is very suggestive of labyrinth involvement.

3. Proportionate impairment of the responses from the horizontal canal and of the responses from the vertical canals. If, for example, the tests show that the horizontal canal retains only one-half of its normal function and that the vertical canals similarly retain only one-half of their normal function, a lesion of the end-organ itself is suspected.

4. Proportionate impairment of both nystagmus and vertigo. If the horizontal canal produces one-third of the normal nystagmus and one-third of the normal vertigo, it is suggested that the lesion is in the horizontal canal itself or in the fibers from the canal within the eighth nerve; if, in addition, the vertical canals produce one-third of the normal nystagmus and one-third of the normal vertigo, an end-organ

[1] Jones, I. H.: The Value of Ear Examination to the Neurologist, Tr. Am. Otolog. Soc., vol. **20**, p. 55. Equilibrium and Vertigo, J. B. Lippincott Co., 1918. Alexander V. Marburg: Handbuch der Neurologie des Ohres, Berlin-Vienna, 1924. This is an important work for the expert. Bárány: Arch. f. Ohrenh., 1906, **68**, 1.

lesion is strongly suggested. In a word, it is the "proportionate impairment" of responses that speaks for a peripheral lesion.

A central lesion is rendered probable by the following:

1. A normal cochlea but impaired or non-responsive semicircular canals.
2. Normal responses from the horizontal canal but absent responses from the vertical canals.
3. Normal responses from the vertical canals but impaired responses from the horizontal canal.
4. Normal vertigo but impaired nystagmus from the horizontal canal.
5. Normal nystagmus but impaired vertigo from the horizontal canal.
6. Normal vertigo but impaired nystagmus from the vertical canals.
7. Normal nystagmus but impaired vertigo from the vertical canals.
8. Normal vertigo and normal nystagmus from any semicircular canal, but impaired past pointing in any direction of any one extremity.
9. Normal vertigo and normal nystagmus from any semicircular canal but an impairment or absence of the normal falling.
10. Spontaneous vertical nystagmus is pathognomonic of a central lesion and is indicative of involvement of the brain stem caused either by their infiltration or pressure. A lesion of the labyrinth may produce many forms of spontaneous nystagmus—horizontal, rotary, oblique, or a mixed nystagmus of the various types; but an ear lesion does not produce a spontaneous vertical nystagmus, either upward or downward.
11. If there exists a spontaneous nystagmus to the right and non-responsive semicircular canals of the right ear, an intracranial lesion is suggested. The non-responsive labyrinth, if the labyrinth itself alone were responsible, would produce a nystagmus to the left.
12. A spontaneous nystagmus of increasing intensity or of long duration is indicative of a central lesion. A spontaneous nystagmus due to a lesion of the labyrinth shows its greatest intensity at the onset of the disease, becomes less and less marked and disappears after a few days.
13. If a stimulation of any semicircular canal produces a "perverted" or "inverse" nystagmus, it is pathognomonic of a central lesion and is indicative of brain-stem involvement. Such phenomena as the following are frequently seen: Douching the right ear with cold water with the head back sixty degrees, stimulating the right horizontal canal, should produce a pure horizontal nystagmus to the left. If on stimulation there occurs a vertical nystagmus upward or downward, a rotary, oblique, or mixed nystagmus, it may be spoken of as "perverted." If, instead of a horizontal nystagmus to the left, there is produced a pure horizontal nystagmus to the right, it may be termed an "inverse" nystagmus. Neither a perverted or an inverse nystagmus can possibly be produced by a lesion of the labyrinth or eighth nerve; a peripheral lesion produces a poor nystagmus or no nystagmus at all, but an absolutely false response of necessity demonstrates a central lesion.
14. If ear stimulation produces a conjugate deviation of the eyes instead of a nystagmus, it is pathognomonic of a central lesion.

This outline indicates how additional data may be furnished to the

neurologist by the ear tests in determining whether a lesion of the internal ear or of the brain stem or cerebellum is present. In the broader field of localization, examination of the ear and of the vestibular apparatus is also of distinct value. The particular feature of the ear examination is that the aurist sends in a stimulus to the brain centers, and then notes the responses of the different parts of the body to this stimulus. For example, by stimulation of the ear there results a nystagmus in a given direction, a pointing of the extremities to the right or to the left, as the case may be, and a falling to the right, to the left, forward or backward as the case may be. Now, if the ear and these central paths from the ear are intact, all the normal responses will appear. If there is a failure of all or any of the responses it is positive evidence of an interruption along that particular path or paths that fail to bring about these responses.

The chief members of this complicated series of reflexes have been studied very exhaustively in the past ten years, and the results of animal experimentation of Magnus and de Kleijn on lower animals may be summarized as follows: Magnus[1] large work may be consulted for details:

I. Reflexes responding to movements	
1. Rotatory reactions	
(*a*) On head	Apparatus of semicircular canals. Otolithic apparatus may also be stimulated by movement.
(*b*) On eyes	
(*c*) On limbs	
(*d*) On trunk	
II. Reflexes resulting from position	Otolithic apparatus.
1. Tonic labyrinthine reflexes on the bodily musculature	
(*a*) On limbs	Utricle.
(*b*) On neck (and trunk)	Utricle.
2. Labyrinthine righting reflexes	
(*a*) Asymmetrical	Saccule.
(*b*) Symmetrical	Utricle.
3. Compensatory eye positions	
(*a*) Vertical	Saccule.
(*b*) Rotatory	

In order to utilize the knowledge obtained from these tests it is essential to have in mind the various pathways constituting the vestibular apparatus. It has been partly shown that the pathways from the horizontal semicircular canal are different after entering the brain stem from those of the vertical canals; furthermore, that each set of tracts divides into two separate pathways; one pathway, the vestibulo-ocular tract, is responsible for the eye movement, and the other pathway, the vestibulocerebellocerebral tract, conveys the impulses from the ear to the cerebral cortex, producing vertigo. If the horizontal canal fails to produce both nystagmus and vertigo the lesion indicated

[1] Magnus: Die Körperstellung, Springer, Berlin, 1924.

is at a point before the division of the horizontal canal fibers into their two separate pathways. Further, if the horizontal canal produces normal vertigo but no nystagmus, the lesion indicated is in the vestibulo-ocular tract at a point beyond the point of division into the two paths. If the horizontal canal produces normal nystagmus but no vertigo, the lesion indicated is at a point along the vestibulo-cerebello-cerebral path beyond the point of division into the two pathways. Similarly, if the vertical canals produce normal vertigo but no nystagmus the lesion indicated is in the vestibulo-ocular tract at a point beyond the division into the two paths. If the vertical canals produce normal nystagmus but no vertigo, the lesion indicated is at a point along the vestibulo-cerebello-cerebral path beyond the point of division of the two pathways. The ear tests have proved themselves surprisingly helpful in localizing lesions in the cerebello-pontine angle, medulla oblongata, pons, cerebellar peduncles, cerebellum and the parietal, temporal and occipital lobes.[1] (Other tests, optic, caloric nystagmus etc., see cranial nerve section.)

Examination of the Pharynx.—Careful note should be made of the position of the fauces while at rest and during phonation, and of the movements of the soft palate during phonation. The pharyngeal reflexes are tested by touching successively the right and left pillar, and the vomiting reflex is best tested by irritating the back of the pharynx with a wooden spatula.

While under normal conditions the uvula should hang in the middle line, a certain amount of variation in position is very frequently observed.

While the pharynx is being examined, note should be made of the pharyngeal vault, and also of the contour of the posterior pharyngeal walls. Further, the integrity of the superior pharyngeal muscles should be tested by having the patient swallow, and noting whether the act is coördinated, or whether there is regurgitation through the nose.

The state of tonus in the pharynx is of great significance. Many individuals show chronic tightness or laxness in the pharyngeal tonus. These are often direct results of psychogenic tensions. They are the forerunners of chronic pharyngeal catarrhs, tonsillitis, tinnitus, and often so-called middle ear catarrhal deafness. The vagotonic pharynx is of much clinical significance and the relation of this, in the unconscious to fellatio phantasies is of far reaching importance in speech production, and chronic nasopharyngeal disease, as well as in manifest delusional symbolic acts such as spitting, salivation, and many unconscious sexual symbolizations. Nauseas, vomitings, with psychogenic components often show these pharyngeal tonic states associated with unconscious phantasies of impregnation, particularly in psychoneuroses (hysteria) and in psychotics.

[1] Compare Magnus. Die Körperstellung, 1924. Studies of Magnus, de Kleijn: de Kleijns, Versteegh, Proc. Roy. Soc. Med., 1924, **17**, 11. Acta Otolaryn., 1923, **6**, 170, for methods of testing rotatory eye movements in labyrinthine disease.

TESTS FOR MUSCLES OF PHARYNX, PALATE AND TONGUE.

Test of function.	Muscle.	Disordered function.	Chief innervation.	Synapse.	Usual lesion.
Push tongue contralaterally	Genioglossus	Pushed to paralyzed side	Hypoglossal	Medulla	Acute or chronic inflammation of medulla, encephalitis, poliomyelitis, hemorrhage, thrombosis, tuberculosis and syphilis; some dystrophies.
Bring tongue up and back	Styloglossus	Backward; hollowing action interfered with	Hypoglossal	Medulla	
Free tongue movement	Lingual	At rest in midline; on protrusion goes to paralyzed side; unilateral atrophy	Hypoglossal	Medulla	
Pull uvula	Azygos uvulæ	Nasal regurgitation or unilateral position to healthy side; voice tone nasal in double paralysis	Facial nerve, pharyngeal sympath.	Medulla	
Raise velum	Levator palati	Pharynx relaxed; the bilateral palsy causes flapping; nasal regurgitation	Facial nerve, pharyngeal sympath.	Medulla	As above and in some seventh nerve palsies; diphtheria.
Drive water into esophagus by swallowing	Palatopharyngeal	Nasal regurgitation; nasal voice	Trigeminus	Pons and medulla	Syphilis of base; hemorrhage; fractures; tumors.
Swallow water, draw larynx up aids epiglottis closure with tongue	Stylopharyngeus	Impaired swallowing; food in the trachea	Glossopharyngeal	Medulla	Balbar and basilar lesions.
Swallow water; drive into the esophagus	Constrictors of pharynx	Imperfect swallowing	Sympathetic pharyngeal plexus	Medulla; cervical	Bulbar and basilar lesions.
Breathe, speak; and with mirror observe the larynx	Laryngeal muscle	Rough and difficult breathing and impaired phonation	Mostly recurrent laryngeal	Medulla	Bulbar and local lesions.

Examination of Taste.—Taste is a complex function and utilizes at least three different nerves. It is roughly tested by solutions which are sweet (sugar), bitter (quinine), acid (vinegar), salty (salt solutions). The solutions should be kept in wide-mouthed bottles, and are applied in small quantities by means of a glass rod applied to different parts of the tongue to determine the functional capacity of these parts. Small amounts should be used, and it is best to reserve the bitter test until the last, commencing with the sweet, and following with the sour, the salt, and the bitter. The mouth should be washed out between the tests if careful records are to be made.

While testing for taste the condition of the secretion of the buccal mucous membrane can be tested. This is best done by rubbing the floor of the mouth with a glass rod and noting the rapidity of excretion and the amount, comparisons being made between the right and left sides.

Examination of the Larynx.—This naturally has to be carried out by means of a laryngological mirror, when malposition or changed position of the vocal cords during inspiration, expiration and phonation are observed. Vocalization is an extraordinarily complicated function and the great intricacies of laryngeal innervation cannot be detailed here. Fig. 23 shows the position of the vocal cords in four characteristic palsies.

Examination of Hypoglossus.—The function of language and its minute analysis will not be entered into in this place. (See Aphasia, p. 430). The general aspects of neurological interest to be observed are whether the *tongue* is protruded in the middle line, whether it is freely movable, up, down, right and left, and can be made to push out both cheeks. Careful search should be made for scars on the tongue, and the presence of a leukoplakia on the sides of the tongue or of the mucous membrane of the cheeks should not be overlooked.

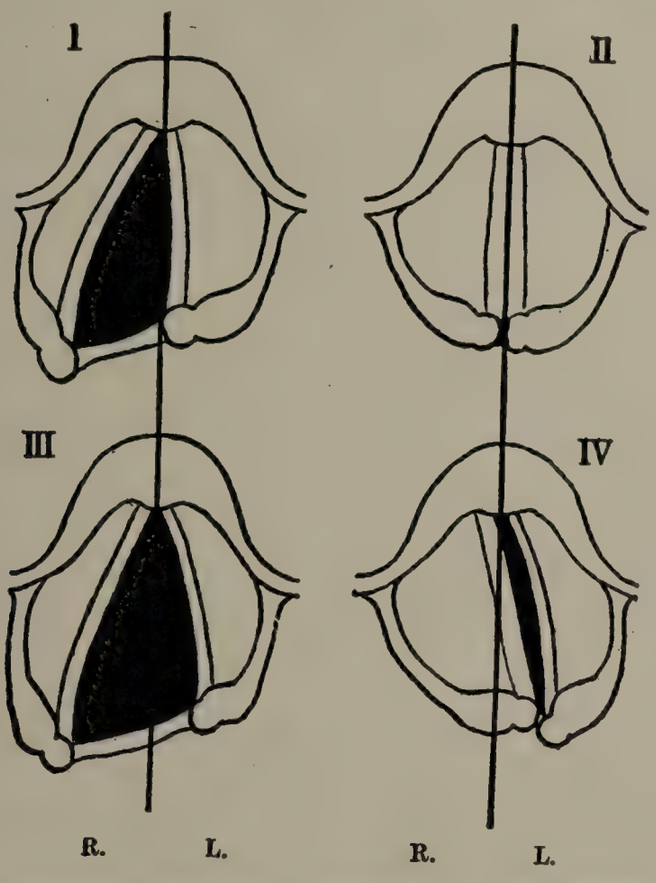


FIG. 23.—Organic laryngeal paralyses. (Barwell.) *I*, left abductor paralysis during inspiration; *II*, left abductor paralysis during phonation; *III*, left recurrent laryngeal paralysis, during inspiration; *IV*, left recurrent laryngeal paralysis, during phonation.

Tremors of the tongue may be very fine, involving the whole organ (fibrillary), or coarse and irregular. Careful attention should be devoted to the search for tongue tremors.

In testing ordinary speech, certain test phases are advisable. The patient should be directed to repeat the alphabet, and the numbers up to twenty-five, and he should repeat something well known, such as the Lord's Prayer, or some bit of poetry, and during the recital careful attention should be directed to the enunciation of the individual letters, to the presence of stumbling over words, of running words together, to the omissions of words, or the omission of syllables, and particularly to the repetition and the displacement of syllables. In order to bring out some of these defects, certain test phrases are utilized. Among those are the following: Truly Rural; Third Riding Artillery Brigade; Methodist Episcopal; National Intelligencer. Naturally the type of case will suggest certain defects, which can then be examined for in greater detail.

In stating the speech defects due to laryngeal loss, special attention should be directed to the presence of cough, of stridor, and to the pitch of the breathing.

The speech is further tested by having the patient repeat foreign words, read spontaneously, read after writing, and define spoken, written, and printed words.

Aphasic Status.—A very superficial aphasic status should include the following tests:[1]

First ascertain the usual habits of the patient and of the parents with reference to the use of the right and left hand in every-day acts.

1. Is spontaneous speech possible and is it intelligible or non-intelligible? Record should be made of the choice of words, complete stenograms of the words uttered should be made. Attention should be given to variations in pronunciation, cadence, and rhythm. Observe "word salad," "jargon aphasia," "jumbled, precipitate speech," "stumbling speech," "torrent speech," "hot-potato speech."

2. Does the patient understand words? This is tested by asking him questions, which should not be too simple, such as what is his name, etc. He should be asked to touch his nose, his left ear, the top of his head, his right knee. These should all be asked without any trace of movement on the part of the inquirer and preferably so that the patient cannot see the lip movements.

3. The knowledge of written or printed words should be tested. The same commands as previously mentioned (2) should be written out and shown to him.

4. Can the patient repeat words spoken such as Sasaktai, Constantinople, hoi-polloi, or mouse, rose, sunfish, etc.

5. Can he write spontaneously, left hand, or on typewriter. Observe misplacing and similar defects, as in speaking (1). Is he able to write from dictation? Is he able to copy what he sees?

6. Can he name objects pointed out; is he able to recognize objects after their name is spoken, or their names written? Can he pick out objects named, written, or shown?

7. Can he obey commands calling for simple gestures, such as bowing, throwing a kiss, clenching the hand in defiance, either in response to spoken wish or to imitative gesture?

8. Can he understand the use of objects, such as striking a match, using a paper cutter? (Apraxic tests).

For fuller consideration see Henry Head's (1926) work upon Aphasia.

Stuttering is a spasmodic form of speech disturbance which calls for special mention. Special attention should be paid to the particular kind of stuttering. Stammering or lisping, is a minor defect. Stuttering is usually a severe type of compulsion neurosis, and particular attention should be devoted to what consonants the individual hangs upon. Labial, dental or lingual hitches have their special significance which the psychoanalytic technic may aid to decipher.

[1] Henry Head: Aphasia, 1926, for detached analysis of aphasic patients. Kinnier Wilson has written a delightful small work. Psychic Minature Series, Henschen. Flechsig Festschrift, Mon. f. N. & P., 1927, **65**.

Certain patients show a very marked slowing of speech (bradylalia), while others show an intermittent enunciation, and still others a peculiar, monotonous, semi-singsong type of enunciation known as "scanning speech." In complicated speech disturbances due to involvement of the hypoglossal nerve one obtains the so-called bulbar thick speech: the patient speaks as though he had a hot potato in his mouth. Further, in extensive speech disturbances due to coexisting lesions in different parts of the speech mechanism one has other disturbances known as anarthria, or more particularly as dysarthria, or "jumbled speech."

Cerebellar dysarthria[1] present special points of interest which are manifested in peculiar types of speech disturbance. Such occur in hemorrhagic softenings, tumors of the cerebellum, in scleroses, hereditary ataxias, in olivo-cerebellar pontine syndromes (Thomas) and other syndromes, here considered, more in detail, in midbrain and cerebellar syndromes. These are related to the asynergias. Are the speech movements slowed? Are they precipitate (propulsion)? Are they non-discriminative as to the labials, dentals, linguals, gutterals? Can the individual speak in a low voice? Does intensity rise and fall in an irregular manner; is there no loss of critique, as in paresis, and is there a sense of irritation at not being able to control the speech mechanism.

The Tenth Nerve.—The study of the heart action and tests is referred to the section on the Vegetative Nervous System.[2]

The Eleventh or Spinal Accessory Nerve.—This nerve supplies the sternomastoid and the trapezius. Its functional capacity is tested by the ability to raise the shoulders and to turn the neck, pressure being made on the chin in resistance. There are a number of striking displacements resulting from paralysis of this nerve which will be discussed later under the head of Paralysis.

Ninth and Twelfth Nerves.—The glossopharyngeal and hypoglossal nerves may be considered more or less together in their testing. The glossopharyngeal is involved in the function of taste of the posterior third of the tongue and of the soft palate. It is also a nerve of common sensation for the back of the tongue, part of the soft palate, and upper part of the pharynx. It supplies the middle constrictor of the pharynx and the stylopharyngeus with motor fibers.

For further details of the testing of the other nerves see the chapter on Cranial Nerves.

Head and Neck.—The muscles that move the head and neck, their function, their nerve supply, and the spinal segment in which the motor synapses are located are as follows (Figs. 27, 28, 29 and 30):

[1] Milianet Schulmann: La Dysarthries des Cerebelleux. Progres Médicale, 1920.

[2] For a useful and conservative summary consult Mosler and Werlich: Die physikalische Vagusprüfungen, München. med. Wchnschr., October 8, 1920.

TESTING MOVEMENTS OF HEAD, NECK AND TRUNK.

Test movement.	Muscles involved.	Innervation.	Synapse.
Pull down angle of mouth and skin	Platysma myoides	Facial, cervical	Medulla,
Bend head forward (flex)	Rectus capitis anterior	First to third cervical	C1
Bend head back (ext)	Splenius capitis, cervicis Semispiralis capitis, cervicis Rectus capitis posticis, maj. minor	First to fourth cervical Eleventh nerve	C1–C4
Rotate head	Sternocleidomastoid Platysma, obliquis Cap. inf., longus colli	First to fourth cervical Eleventh nerve	C1–C3
Bend head laterally	Rectus cap. later	First to fourth cervical	C1
Holding spine erect	Spinalis cervicis	First to fourth cervical	C1–C4
Bend spine backward	Longissimus dorsi, iliocostalis dorsi et lumborum	Thoracic nerve	T2, L1
Bend trunk forward	Rectus abdominis, etc.	Eighth to twelfth thoracic	T3–T12
Rotation of trunk, rt. and left	Semispinatus dorsi	Thoracic nerve	T4–T12
Bend trunk sideways	Quadratus lumborum	Lumbar plexus	T10, L2
Cough, open larynx (diaphragm fluoroscope)	Diaphragm	Phrenic	C3–C5
Breathe deep (costal muscle)	Scaleni, intercostales	Cervical and thoracic	C4, T12
Bear down and cough closed larynx	Diaphragm, abdominal and pelvic	Phrenic, lumbar plexus	C3, S4

Upper Extremities.—A systematic examination of the upper extremities is next in order; the muscular apparatus first claiming attention. The two sides of the body should be examined systematically. (See Figs. 27 to 30.)

Anomalies of structure should first be noted, such as habit or occupational positions, alterations of posture, etc. Gross differences in the size of the bones, the wrists, hands, etc., should be measured.

Atrophy.—This may be determined by simple palpation and by measurement. After natural differences in the muscular volume are taken into consideration, striking variations should be carefully measured. The circumferences over the biceps and just below the elbows on the two sides should be compared. Special individual muscular atrophies may be picked out later by means of electrical reaction tests.

Hypertrophy.—This is best brought out by palpation and by measurement. Due weight should be given to natural variations—right and left sides—and to the influence of definite occupations—blacksmiths, iron-workers, etc.

Hypotonus.—This is indicated by unusual flaccidity of the musculature and movements. Sudden pronation or supination of the arm, extension or flexion at the shoulder-, wrist- or elbow-joints, may show sudden sharp resistances, followed by marked flaccidity. Marked overextension, etc., is a sign of hypotonus.

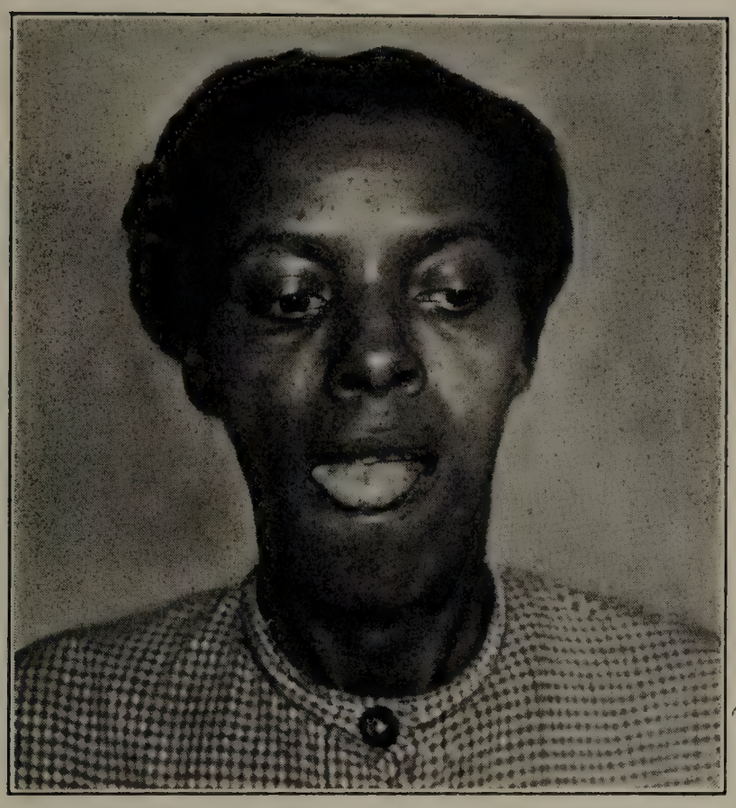


Fig. 24.—Partial atrophy of tongue in occlusion of inferior cerebellar artery.

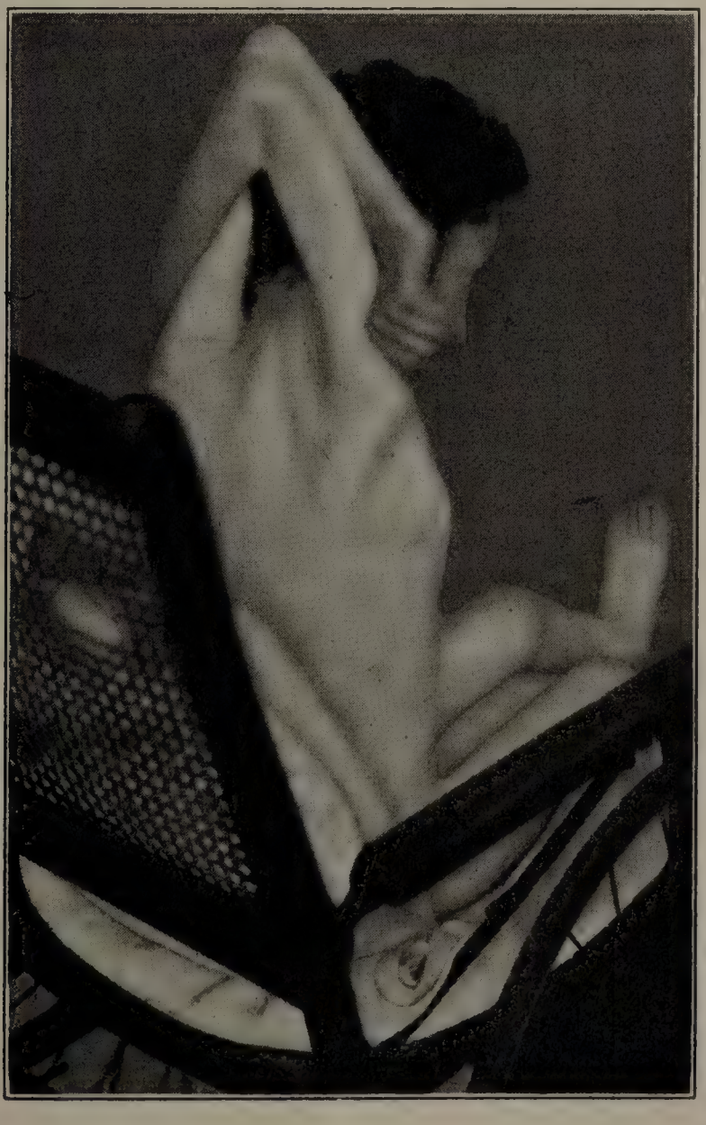


Fig. 25.—Pronounced atrophy and hypotonus in a patient with amyotonia congenita. (Abrahamson.)

The observer should bear in mind that muscular tonus is controlled by vegetative, somatic sensorimotor and psychical factors.[1]



FIG. 26.—Marked hypotonus in a patient with amyotonia congenita. (Neustädter.)

Spasm.—This indicates hypertonus. When permanently present contractures result. The particular muscles which show hypertonus, or spasticity or contractures, should be recorded. In certain spastic conditions the hypertonus may be relieved by passive movements.

Muscular Power.—This is first tested by having the patient execute all the chief movements of the shoulders and arms. The chief tests are as follows:

TESTS FOR SHOULDER-GIRDLE FUNCTIONS.

Test movement.	Muscles involved.	Innervation.	Synapse.
Raise shoulder blade	Trapezius and levator scapulæ	Eleventh nerve, third to fourth cervical Dorsalis scapulæ	C2–C15
Draw scapula to midline	Rhomboidei	Dorsalis scapulæ	C4–C5
Fix and rotate scapula	Serratus anterior	Long thoracic	C5–C7

TESTS FOR SHOULDER-JOINT AND UPPER ARM.

Test movement.	Muscles involved.	Innervation.	Synapse.
Raise arm to horizontal	Deltoid	Axillaris	C5–C6
Press arm to side	Pectoralis maj., minor	Anterior thoracic	C5–C8
Press arm in and back	Latissimus dorsi and pectoralis	Subscapularis	C6–C8
Rotate arm out	Supraspinatus, infraspinatus Teres minor	Suprascularis	C5 (C5–C6)
Rotate arm in	Subscapularis, teres major	Subscapularis	C5–C6

[1] See Lewy, F. H.: Tonus and Bewegung, Berlin, 1923.

TEST FUNCTION OF FOREARM: ELBOW-JOINT.

Test movement.	Muscles involved.	Innervation.	Synapse.
Flex arm palm up	Biceps, brachialis	Musculocutaneous	C4–C6
Flex arm, thumb or dorsum up	Brachioradialis	Radialis	C5–C6
Extend arm	Triceps	Radialis	C5–C6
Turn arm out (pronate)	Pronator quadratus, pronator teres	Median	C6, T1
Turn arm in (supinate)	Biceps	Musculocutaneous	C4–C6
	Supinator brevis	Radial	C5–C7

TEST FUNCTIONS AT WRIST-JOINT: HAND MOVEMENTS AS WHOLE.

Test mouement.	Muscles involved.	Innervation.	Synapse.
Bend hand on forearm	Flexor carpi radialis	Median	C6–C7
	Flexor carpi ulnaris	Ulnar	C8
Extend hand on forearm	Extens. carp. rad. long. et brev.	Radial	C4–C7
	Extens. carp. ulnaris	Radial	
Bend hand out	Extens. carp. rad. long.	Radial	C6–C7
	Flexor carp. radialis	Median	C6–C7
Bend hand in	Flexor carp. ulnaris	Ulnar	C8
	Extens. carp. ulnaris	Radial	C7–C8

TEST FUNCTIONS OF FINGER MOVEMENT.

Test movement.	Muscles involved.	Innervation.	Synapse.	Chief producing lesions.
Close hand on palm	Lumbricales	Ulnar and median	C7–C8	Poliomyelitis; plexus wound; peripheral nerve wound.
	Interossei	Ulnar	C7–C8	
Bend middle and terminal joint	Flex. dig. sublim.	Ulnar and median	C7–C8	
Extend fingers	Flex dig. prof.	Ulnar and median	C7, T1	
	Ext. dig. com.	Radial	C6–C8	
	Extens. indicis	Radial	C6–C8	
Extend middle and terminal phalanges	Interossei	Ulnar		
Spread fingers	Interossei dorsalis	Ulnar		
Bring fingers together	Interossei volares	Ulnar		

TEST FUNCTION OF THUMB AND LITTLE FINGER.

Test movement.	Muscles involved.	Innervation.	Synapse.
Flex metacarpus and proximal phalanx	Flexor pollicis brevis	Median and ulnar	C6–C7
Flex terminal phalanx	Abductor pollicis brevis, flexor long. pollicis	Median	C6–C7
		Median	C6–C7
Extend metacarpus and phalanges	Ext. pollicis longis	Radial	C6–C7
	Ext. pollicis brevis	Radial	C6–C7
Abduct metacarpus	Abductor long. pollicis	Radial	C6–C7
Oppose thumb and fingers	Opponens pollicis	Median	C6–C7
Flex little finger	Flex. dig. prof.	Median and ulnar	C6, T1
	Flex. dig. subl.	Median and ulnar	
	Flex min. dig. brev.	Ulnar	
Extend little finger	Ext. min. dig. proprius	Radial	C8
Abduct little finger	Abd. dig. min.	Ulnar	C8, T1
Oppose the little finger and thumb (sheet of paper)	Opponens min. dig.	Ulnar	C8

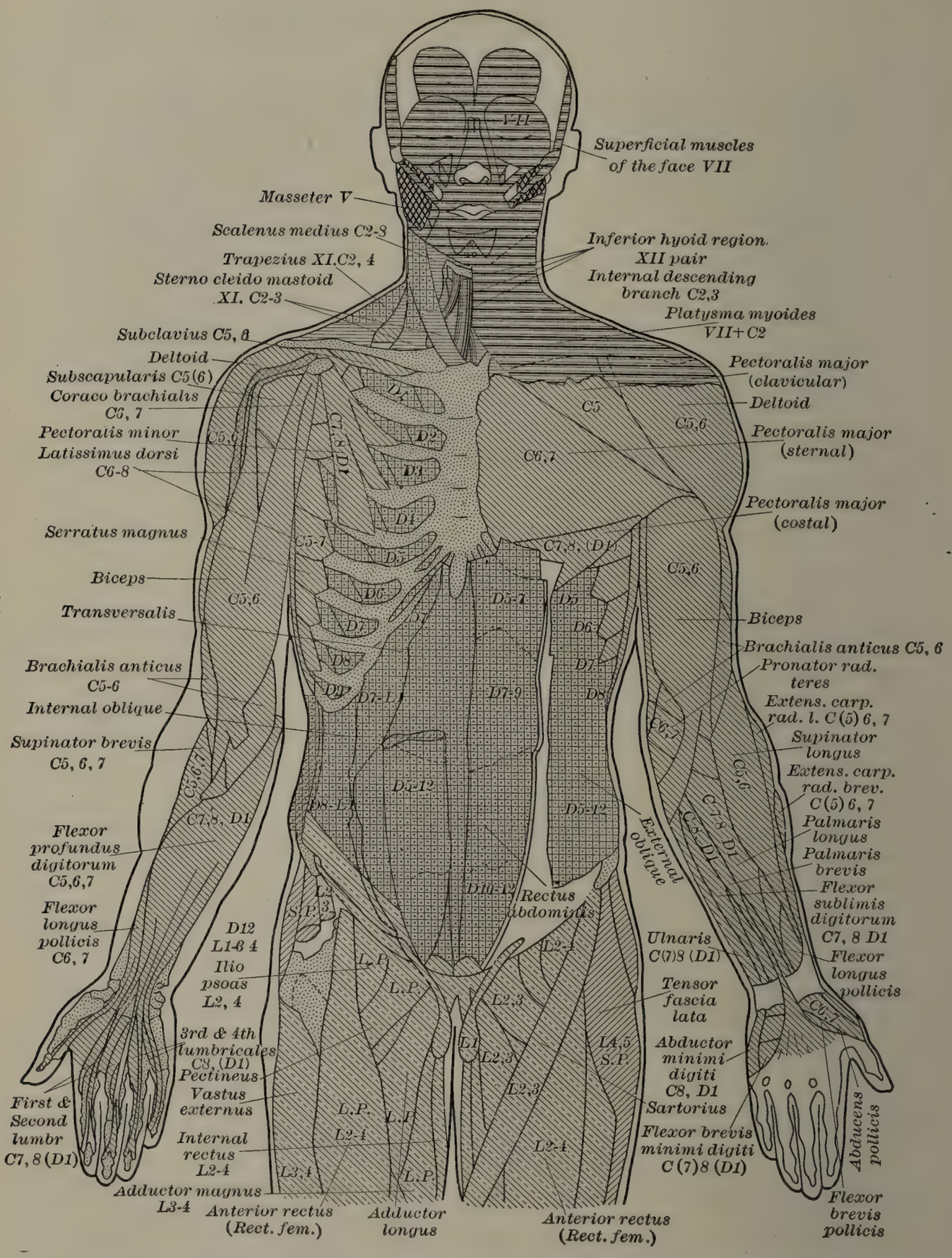


FIG. 27.—*Radicular* innervation of the muscles of the anterior regions of the head, neck, trunk, and upper extremities. On the left, the superficial muscular layer; on the right, the deep muscular layer. (After Dejerine.) *C*, cervical; *D*, dorsal; *L*, lumbar.

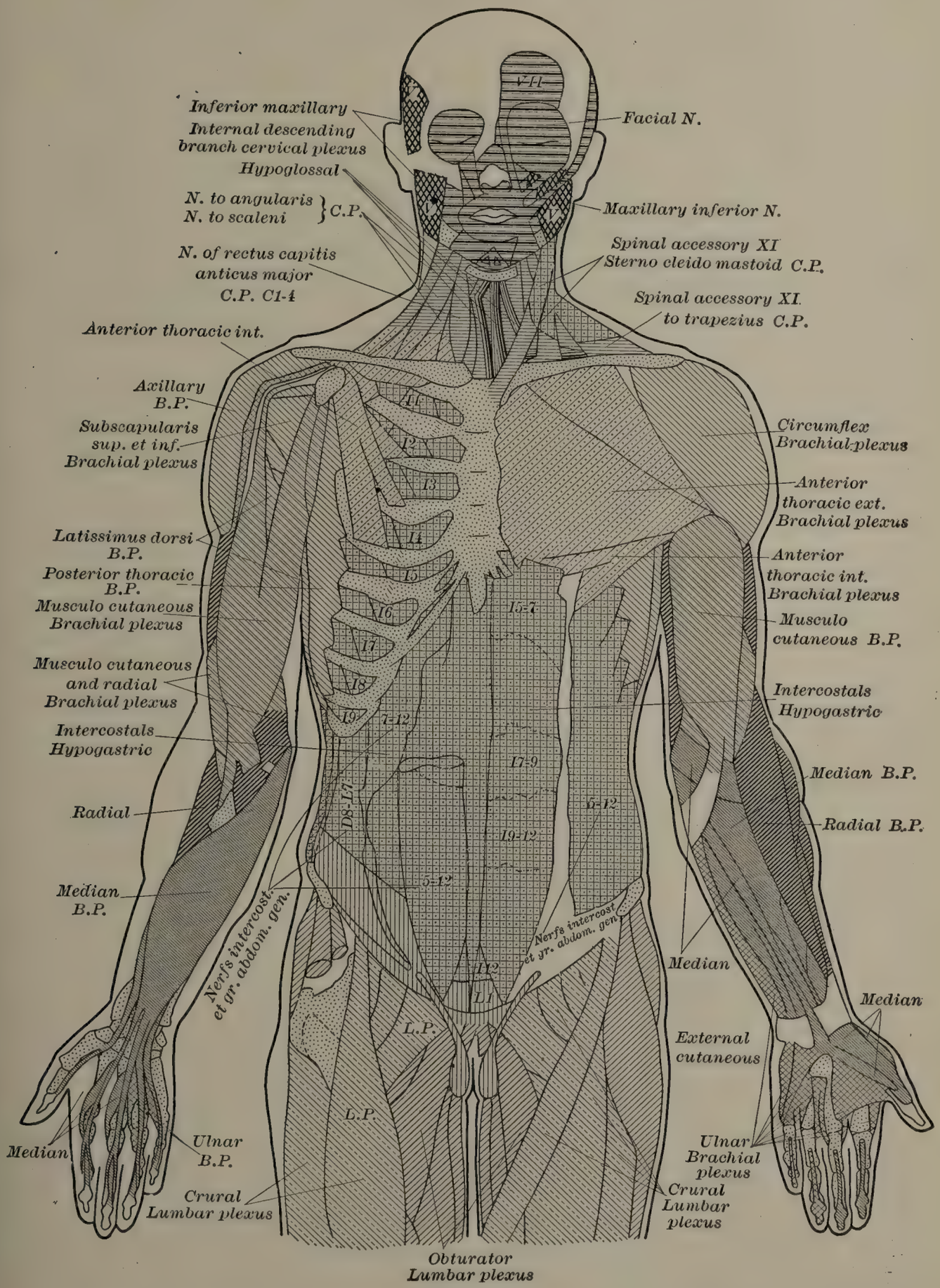


FIG. 28.—*Peripheral* innervation of the anterior muscles of the head, neck, trunk, and upper extremities. To the left the superficial muscular layer; the deep muscular layer to the right. The same markings on this and the preceding figure are followed for the facial, trigeminal, spinal accessory, hypoglossal, cervical plexus, and intercostal nerves respectively. Different shadings differentiate different innervations in the brachial plexus. (After Dejerine.) The chief plexuses are indicated. *C.P.*, cervical plexus; *B.P.*, brachial plexus; *L.P.*, lumbar plexus; *S.P.*, sacral plexus; *D.* in Fig. 27 indicates dorsal roots; *I* 5–7, *I* 9–12, etc., in Fig. 28 intercostal nerves.

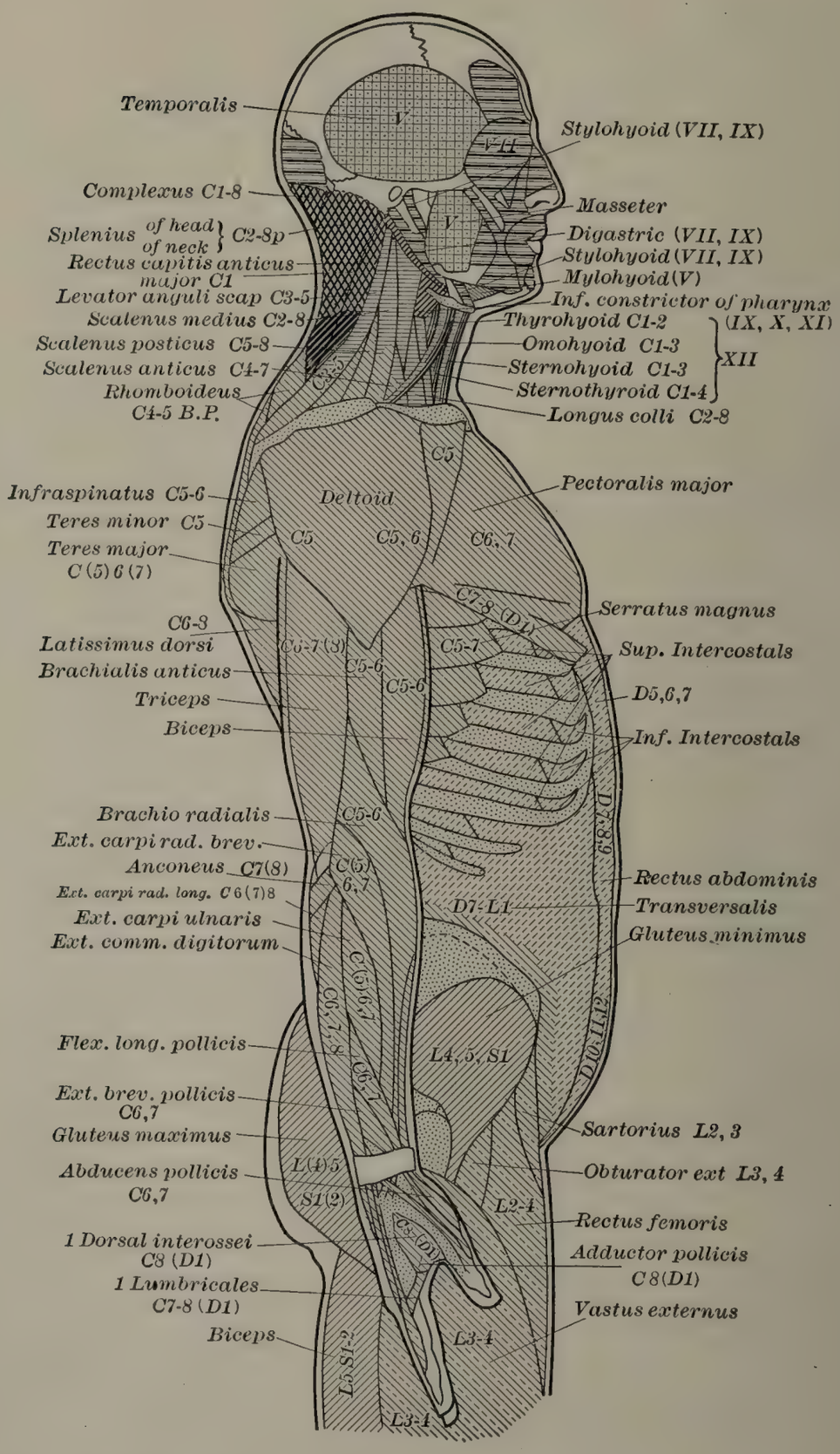


FIG. 29.—*Radicular* innervation of the lateral aspect of head, neck, trunk, and upper extremity. The platysma myoides, sternocleidomastoid, trapezius, and oblique muscles of the abdomen, gluteus medius, and tensor of the fascia lata have been removed. The shading and cross-hatching follow the muscles and innervation as in Figs. 27 and 28. (After Dejerine.)

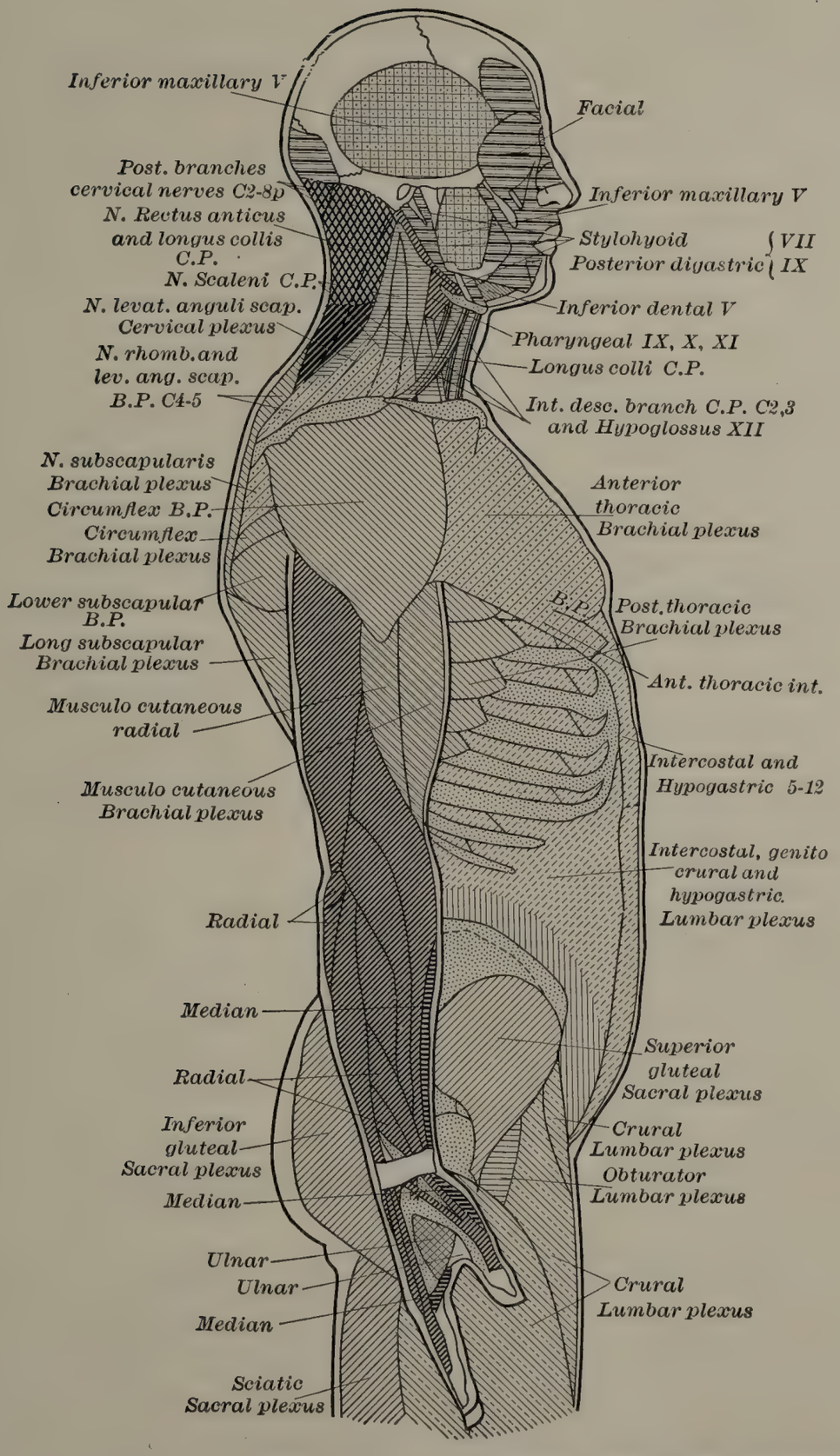


FIG. 30.—*Peripheral* innervation of the lateral aspect of the head, neck, trunk, and upper extremity. Markings and cross-hatchings, as in previous figure, to indicate the various innervations. (After Dejerine.) Letters *C.*, *D.*, *L.*, *C.P.*, *B.P.*, *L.P.*, and *S.P.* as in previous diagrams.

Shoulder, Arm, Hand, and Fingers.— Deltoid.—Request the patient to raise the arms laterally to a horizontal position. Inability to do so indicates deltoid weakness or paralysis.

Trapezius.—Ask the patient to raise the shoulders as close to his ears as possible against the pressure of the examiner's hands. This will demonstrate the strength of the upper part of the trapezius. The middle and lower portions are tested by desiring him to bring the scapulæ as close together as possible.

Latissimus Dorsi.—Raise the arms laterally to a level, then, while keeping them fully extended, bring the arms downward and backward as if to make the hands meet beyond the sacrum. The examiner standing behind the patient resists the movement.

Pectoral Muscles.—Stretch out the arms straight in front and then approximate the hands against resistance by the examiner, meanwhile watching both heads of the pectoral muscle.

Serratus Magnus.—Ask the patient to push with his hands against those of the examiner or against a solid object. If the serratus has lost its power the scapula will project and the digitations of the muscle, which ordinarily should be visible, will not be seen. Inspiration is weakened in paresis or paralysis.

It is rarely possible to detect paralysis of the levator anguli scapulæ and rhomboids unless the trapezius is also involved.

Biceps.—Let the patient flex his extended arm, his elbow resting in the observer's left hand, while the latter's right hand, grasping the wrist of the patient, offers the necessary resistance. Also supinate the hand against resistance.

Triceps.—The triceps may be tested as are the biceps, excepting that the previously flexed arm is to be extended against resistance.

Supinator Longus.—Test as for the biceps, except that the hand should be midway between supination and pronation. If the muscle is paralyzed it will fail to become conspicuous on the radial side of the upper part of the forearm.

Flexors of the Wrist.—Grasping the patient's hand, the palm being upward, ask him to bend the hand up toward his forearm against resistance.

Extensors of the Wrist.—The patient's hand being held palm downward, he is requested to bend it backward against resistance. Moderate weakness of the extensors of the wrist may be manifested by asking him to squeeze the examiner's hand, in which case the wrist will become involuntarily flexed, the weakened extensors being unable to counteract the flexors. Marked or complete paralysis of the extensors causes wrist-drop.

Flexors of the Fingers.—Because of the usual difference in the strength of the two hands the examiner should cross his forearms and place his right hand in the right hand of the patient, and *vice versa*. Then let the patient squeeze the hands. If the observer keeps his own fingers extended and bunched loosely together he will be able to withstand a very hearty grasp without discomfort.

Adductor Pollicis.—Ask the patient to pinch with his thumb and finger one of the examiner's fingers.

Opponens Pollicis.—Direct the patient to approximate the ends of the little finger and the thumb—while thus approximated the examiner pulls his finger through or use a piece of paper and pull. Ability to do so easily shows weakness of the opponens.

The *interossei* and *lumbricales* muscles of the hand flex the proximal phalanges, and extend the middle and terminal phalanges. The dorsal interossei abduct, the palmar adduct, the fingers from and toward a longitudinal line drawn through the center of the middle finger. Test by making the patient separate and approximate the fingers, and flex the proximal phalanges, keeping the middle and terminal phalanges extended. Paralysis of these muscles causes "claw-hand."

The strength of the hand grasp is well tested by the dynamometer. Readings of three tests for each hand should be recorded. Dynamometer records are available for definite comparisons. The dynamometer is also useful by many repeated readings (20) for obtaining an idea of the fatigability of muscles (neurasthenia, myasthenia, alterations in attention, etc.). Weiler has constructed a useful dynamometer with a graphic register. Tilney has also devised a useful instrument.[1]

Expressed in tabular form the muscles, their action, nerve supply, and spinal synapses are as follows:

MUSCLES OF SHOULDER GIRDLE, ACTIONS, NERVE SUPPLY AND SPINAL SYNAPSE SEGMENTS.

MUSCLE.	ACTION.	NERVE SUPPLY AND SPINAL SEGMENT.
Trapezius.	*These muscles move the shoulder girdle:*	Spinal accessory, cervical plexus, medulla N, ambiguus C1–5, C3–4.
	(*a*) Vertical plane. *Elevation.*	
Latissimus dorsi.	Trapezius—upper fibers.	Long subscapular, C6–8.
Levator scapulæ.	Levator scapulæ.	Cervical plexus, posterior scapular, C3–5.
	Rhomboidei.	
	Sternohyoid.	
Rhomboidei.	Omohyoid.	Posterior scapular, C5.
	Depression.	
Pectoralis major and minor.	Trapezius (lower fibers).	External and internal anterior thoracic, C5–7, C8, D1.
	Subclavius.	
	Pectoralis minor.	
Subclavius.	Latissimus dorsi.	
	Pectoralis major (lower fibers).	Brachial plexus, C5–6.
Serratus magnus.	(*b*) Horizontal plane.	
	Forward:	Posterior thoracic, C5–7.
	Serratus magnus.	
	Pectoralis major	
	Pectoralis minor.	
	Backward:	
	Trapezius.	
	Rhomboidei.	
	Latissimus dorsi.	

[1] Froment, J.: Examem méthodique des paralysies périphériques et testes objectifs Paris Méd., October 6, 1923.

MUSCLES OF SHOULDER-JOINT, ACTIONS, NERVE SUPPLY AND SPINAL SYNAPSE SEGMENTS.

MUSCLE.	ACTION.	NERVE SUPPLY AND SPINAL SEGMENT.
	Abduction:	
Deltoid.	Deltoid.	Circumflex, C5–6.
Teres minor.	Supraspinatus.	
	Adduction:	
Supraspinatus. }	Teres major and minor.	Suprascapular, C5–6.
Infraspinatus. }	Pectoralis major.	
	Latissimus dorsi.	
	Coracobrachialis.	
	Biceps.	
	Triceps.	
Teres major.	*Flexion (forward):*	Lower subscapular, C5–6.
	Deltoid (anterior).	
	Subscapularis.	
Teres major.	Pectoralis major.	Lower subscapular, C5-6.
	Coracobrachialis.	
	Biceps.	
Subscapularis.	*Extension (backward):*	Lower and upper subscapular, C5–6.
	Deltoid (posterior).	
	Teres major.	
	Infraspinatus.	
	Latissimus dorsi.	
	Triceps.	
	Rotation (out):	
	Deltoid (posterior).	
	Infraspinatus.	
	Teres minor.	
	Rotation (in).	
	Deltoid (anterior).	
	Teres major.	
	Pectoralis major.	
	Latissimus dorsi.	
Coracobrachialis.	*Flexors:*	Musculocutaneous, C7.
Biceps.	Biceps.	Musculocutaneous, C5–6.
Brachialis anticus.	Brachioradialis.	Musculocutaneous, C5–6.
	Pronator radii teres.	
Brachialis anticus.	Flexor profundus dig.	Musculospiral, C5–6.
Triceps.	Flexor longus pollicis.	Musculospiral, C6–8.
Outer head.	Flexor sublimus dig.	Musculospiral, C7–8.
Middle and inner head.	Extensor of wrist in pronation.	
	Extensors:	
	Triceps.	
	Anconeus.	
	Extensors of wrist and fingers (in supination).	

ARM MUSCLES, NERVE SUPPLY AND SPINAL SYNAPSE SEGMENTS.

Posterior muscles of the forearm:	
Brachioradialis (supin. rad. long.).	Musculospiral, C5–6.
Extensor carpi radialis longior.	
Extensor carpi radialis brevior.	
Extensor communis digitorum.	Posterior interosseus C6–8. (Musculospiral.)
Extensor minimi digiti.	
Extensor carpi ulnaris.	
Anconeus.	
Supinator radii brevis.	Musculospiral, C6–8.
Extensor ossis metacarpi pollicis.	Posterior interosseus, C6–8.
Extensor brevis pollicis.	
Extensor longus pollicis.	
Extensor indicis.	

Anterior muscles of the forearm:	
Pronator radii teres.	Median nerve, C6.
Flexor carpi radialis.	
Palmaris longus.	
Flexor sublimis digitorum.	
Flexor carpi ulnaris.	Ulnar, C8, D1.
Flexor profundus digitorum.	Ulnar and anterior interosseus, C7–8, D1.
Flexor longus pollicis.	Anterior interosseus, C7–8, D1.
Pronator quadratus.	
Muscles of the hand:	
Abductor pollicis.	Median, C6–7.
Opponens pollicis.	
Flexor brevis pollicis (superf.).	
Flexor brevis pollicis (deep).	Ulnar, C8, D1.
Adductor obliquus pollicis.	
Adductor transversus pollicis.	
Lumbricales, 1 and 2.	Median, C6–7.
Lumbricales, 3 and 4.	Ulnar, C8, D1.
Interossei.	
Flexor brevis minimi digiti.	
Abductor minimi digiti.	

The actions of these groups are multiform and are best considered separately. Single muscles are not capable of being isolated from the group actions, as a rule. The elbow-joint action has been given. Actions of pronation and supination are important.

Pronation:	Supination:
Pronator radii teres.	Supinator radii brevis.
Pronator quadratus.	Biceps.
Brachioradialis.	Brachioradialis.
Flexor carpi radialis.	Extensors of thumb and fingers.

Action at the Wrist-joint.

Flexion:	*Extension:*
Flexor carpi radialis.	Extensors of wrist.
Palmaris longus.	Extensors of thumb.
Long flexors of thumb and fingers.	Extensors of fingers.
Adduction:	*Abduction:*
Flexor carpi ulnaris.	Flexor carpi radialis.
Extensor carpi ulnaris.	Extensors of wrist.
	Extensors of thumb.

Finger Action.

Flexion:	*Extension:*
Flexor sublimis digitorum.	Extensor communis digitorum.
Flexor profundus digitorum.	Extensor indicis.
Lumbricales. On metacarpo-phalangeal joints.	Extensor minimi digiti.
Interossei. On metacarpo-phalangeal joints.	Lumbricales. Acting on interphalangeal joints.
Flexor brevis minimi digiti.	Interossei. Acting on interphalangeal joints.
Adduction:	*Abduction:*
Palmar interossei (to the middle line of middle finger).	Lumbricales.
	Flexor brevis and Opponens minimi digiti (from inner side of hand).
	Dorsal interossei (from middle line of middle finger).

Thumb Action.

Flexion.	*Extension:*
Opponens pollicis (carpo-metacarpal).	Extensor ossis metacarpi pollicis (carpo-metacarpal).
Flexor brevis pollicis. Carpo-metacarpal and metacarpophalangeal.	Extensor brevis pollicis (carpometacarpal and metacarpophalangeal).
Adductor pollicis. Carpo-metacarpal and metacarpophalangeal.	
Abductor pollicis. Carpo-metacarpal and metacarpophalangeal.	
Flexor longus pollicis (all joints).	Extensor longus pollicis (all joints).
Adduction:	*Abduction:*
Adductors of the thumb.	Abductor pollicis.
Flexor brevis pollicis.	Extensors of thumb.
Opponens pollicis.	
Interossei (1 dorsal).	

The Muscles of the Trunk.—The erector muscles of the spine are examined by requesting the patient to lie face downward and asking him to raise the head and shoulders without assistance from the hands. Unless paralyzed the erectors become clearly visible during the attempt. The abdominal muscles are tested in a similar manner, except that the patient lies on the back while making an effort to raise the head.

MUSCLES OF THE THIGH AND BUTTOCK, NERVE SUPPLY AND SPINAL SYNAPSE SEGMENTS.

Pectineus.		
Sartorius.		
Iliacus.		L1–3.
Psoas.		L1–3.
Quadriceps extensor.		L2–4.
Vastus externus.		Anterior crural, L2–4.
Rectus femoris.		L3–4.
Crureus.		
Vastus internus.		
Tensor fasciæ femoris.		
Gluteus minimus.		Superior gluteal, L4–5 S1.
Gluteus medius.		
Gluteus maximus.		Inferior gluteal, L5, S1–2.
Biceps (s. h.).		Peroneal, L5, S1–2.
Pyriformis.		Sacral plexus, S1–2.
Adductor longus.		L2 3.
Gracilis.		Obturator, L2–4.
Adductor brevis.		L3–4.
Obturator externus.		L3–4.
Adductor magnus.		Obturator, L3–4.
Semimembranosus.		L4–5, S1.
Semitendinosus.		Sciatic, L4–5, S1.
Biceps (l. h.).		L5, S1–2.
Quadratus femoris	Sacral plexus.	L4 5, S1.
Gemellus inferior.		
Gemellus superior.		
Obturator internus.		S1–3.

Most of these muscles act upon the pelvis and on the hips and knee-joints.

HIP-JOINT MOVEMENTS.

Flexion.		*Extension:*
Sartorius.		Gluteus maximus.
Iliacus.		Gluteus medius.
Psoas.		Gluteus minimus.
Rectus femoris.		Biceps.
Pectineus.		Semitendinosis.
Adductor longus.		Semimembranosis.
Gracilis.		Adductor magnus.
Obturator externus.		
Adduction:		*Abduction:*
Pectineus.		Tensor fasciæ femoris.
Adductor longus.		Gluteus medius.
Adductor brevis.		Obturator externus.
Adductor magnus.		Pyriformis.
Gracilis.		Obturator internus.
Quadratus femoris.		Gemelli.
Gluteus maximus (lower fibers).		Sartorius.
		Gluteus maximus (upper fibers).
		During flexion.

Internal rotation:
- Tensor fasciæ femoris.
- Gluteus medius (anterior).
- Gluteus minimus (anterior).

External rotation:
- Obturator externus.
- Gluteus maximus (lower).
- Quadratus femoris.
- Gluteus medius. } Posterior.
- Gluteus minimus. }
- Pyriformis. }
- Obturator internus. } During extension
- Gemelli. }
- Sartorius.
- Iliopsoas.
- Pectineus.
- Adductors.
- Biceps flexor cruris.

TEST OF FUNCTION OF HIP-JOINT MOVEMENTS.

Test movement.	Muscles involved.	Innervation.	Synapse.
Bend thigh up, single standing	Iliacus, psoas	Lumbar	L1–L4
	Sartorius: rectus femoris	Femoral	L2–L4
Bend thigh up, double lying	Tensor fasciæ lata	Femoral	L2–L5
Extend whole leg backward	Gluteus maximus	Inferior gluteal	L5, S1
Abduct right and left leg	Gluteus medius	Superior gluteal	L4, S1
	Gluteus minimus		
Adduct right and left leg	Pectineus	Obturator	L1–L3
	Add. longus, med. and magnus	Obturator	L2–L4
	Gracilis: obturator ext.	Obturator	L2–L4
	Pyriformis	Superior gluteal	S1–S2
Rotate and bend thigh out	Quadratus femoris	Sciatic	L4, S1
	Gemelli	Sciatic	L4, S1
	Obturator internus	Sciatic	L5, S2
Rotate thigh in	Gluteus med. and min.	Superior gluteal	L4, S1
Rise to sitting position from back	Iliacus, psoas	Lumbar	L1–L5
	Abdominalis	Lumbar	D5, L1
Fix thigh on pelvis, front	Gluteus maximus	Inferior gluteal	L4, S1
Fix thigh on pelvis, side	Gluteus med. and min.	Superior gluteal	L4, S1

The Lower Extremities.— *The muscles of the lower extremities* are first roughly tested with the patient standing and then more in detail with the patient lying on the back. Note wasting, tremors, twitchings, tonus and temperature (see Figs. 31 and 32):

Flexors of the Thigh.—The patient lying upon his back, ask him to raise the leg from the bed, against resistance, the knee being kept straight. This determines the strength mainly of the iliopsoas, partly of the quadriceps.

Extensors of Thigh.—The leg being kept straight and the patient lying upon his back, raise the foot and ask him to bring it down upon the bed against resistance. This determines the strength of the gluteus maximus and partly of the hamstring muscles.

Abductors of Thigh.—With the leg across the middle line ask the patient to carry it toward the outer side against resistance, thus testing mainly the gluteus medius.

Inrotators of Thigh.—With the patient on his back, flex the knee to a right angle, grasp the foot, and oppose resistance while he inrotates the thigh, testing mainly the gluteus minimus.

Outrotators of the Thigh.—Similarly test the power of outrotation, thus determining the condition of the obturators, pyriformis, gemelli, and quadratus femoris.

Flexors of the Knee.—The patient lying upon his back, request him to bend the knee while the examiner resists the movement by grasping the ankle, thus ascertaining the power of the biceps, semimembranosus, and semitendinosus.

KNEE-JOINT MOVEMENTS.

Flexion:
Sartorius.
Gracilis.
Semitendinosus.
Semimembranosus.
Biceps.
Gastrocnemius.
Plantaris.
Popliteus.

Extension:
Quadriceps extensor.

Internal rotation:
Sartorius.
Gracilis.
Semitendinosus.
Semimembranosus.
Popliteus.

External rotation:
Biceps flexor cruris.

TEST OF MUSCLES OF KNEE-AND-ANKLE JOINT MOVEMENTS.

Test movement.	Muscles involved.	Innervation.	Synapse.
Bend and flex knee	Biceps femoris	Sciatic	L5, S2
	Semitendinosus	Sciatic	L4, S2
	Semimembranosus	Sciatic	L4, S1
	Gracilis	Obturator	L2–L4
	Sartorius	Femoral	L2–L3
Extend knee, kick	Quadriceps femoris	Femoral	L4, S1
Lift toes medially	Tibialis anticus	Peroneus	L4
Lift toes laterally	Peroneus longus	Peroneus	L5, S1
	Peroneus brevis		
Push toes down	Gastrocnemius	Tibial	L5, S2
	Soleus	Tibial	L5, S1
Abduct foot medially	Tibialis posticus	Tibial	L5, S1
Abduct foot (laterally)	Peroneus brevis	Peroneus	L5, S1
	Peroneus longus		
	Extens. long. digitorum	Peroneus	L5, S1

Extensors of the Knee.—With the patient on the back, flex the knee, and by pressure on the sole of the foot resist his endeavor to extend the knee. The quadriceps femoris is the principal muscle concerned.

Plantar Flexors (Extensors) of the Foot.—With the leg straight resist, by pressure upon the sole of the foot, the patient's endeavor to bring the tarsus in a line with the leg, thus testing the gastrocnemius, soleus, peroneus longus and brevis. Have patient stand on toes.

Dorsiflectors of the Foot.—With the leg straight, resist the patient's attempt of dorsal flexion of the foot, thus testing the tibialis anticus and the peroneus tertius. Marked paralysis of these muscles causes "foot-drop." Have patient stand on the heels.

Muscles of the Foot.—The flexors, extensors, interossei, and lumbricales of the toes are examined in a similar manner to those of the fingers. There is a sort of claw-foot analogous to the claw-hand.

MUSCLES OF THE LEG AND FOOT WITH NERVE SUPPLY AND SPINAL SYNAPSE SEGMENTS.

Muscle.	*Nerve Supply and Spinal Segment.*	
Tibialis anticus.	Anterior tibial.	L4–5, S1.
Extensor proprius pollicis.		
Extensor longus digitorum.		
Peroneus tertius.		
Peroneus longus.	Musculocutaneous.	
Peroneus brevis.		
Extensor brevis digitorum.	Anterior tibial.	
Plantaris.	Tibial.	L4–5, S1.
Popliteus.		
Gastrocnemius.		S1–2.
Soleus.		
Flexor longus digitorum.	Posterior tibial.	L5, S1.
Tibialis posticus.		L5, S1.
Flexor longus hallucis.		L5, S1–2.
Abductor hallucis.	Internal plantar, L4–5, S1.	
Flexor brevis digitorum.		
Flexor brevis hallucis.		
First lumbricales.		
Second, third, fourth lumbricales.		
Flexor accessorius.	External plantar, S1–2.	
Adductors hallucis.		
Interossei.		
Flexor brevis minimi digiti.		
Abductor minimi digiti.		

MOVEMENT OF THE ANKLE-JOINT.

Flexion:	*Extension:*
Tibialis anticus.	Gastrocnemius.
Extensor communis digitorum.	Plantaris.
Extensor proprius pollicis.	Soleus.
Peroneus tertius.	Tibialis posticus.
	Peroneus longus.
	Peroneus brevis.
	Flexor longus digitorum.
	Flexor longus hallucis.
Inversion:	*Eversion:*
Tibialis anticus.	Peroneus tertius.
Tibialis posticus.	Peroneus longus.
	Peroneus brevis.

TEST OF FUNCTION OF MUSCLES OF TOES, 2–4, BIG AND LITTLE TOE.

Test movement.	Muscles involved.	Innervation.	Synapse.
Flex proximal phalanges	Interossei lumbricales	Tibial	
Flex terminal phalanges	Flex longus dig.	Tibial	L5, S2
	Flex brevis dig.		
Extend the toes	Extens. long. dig.	Peroneus	L4, S1
	Extens. brev. dig.		
Spread (abduct) toes	Interossei dorsalis	Tibial	S1–S2
Squeeze (adduct) toes	Interossei volares	Tibial	S1–S2
Bend first joint of big toe down	Flex hallucis brevis	Tibial	S1–S2
Bend terminal joint of big toe	Flex hallucis longus	Tibial	L5, S2
Extend up first phalanx	Extensor hallucis brevis	Peroneus	L4–L5
Extend up last phalanx	Extensor hallucis longus	Peroneus	L4–L5
Pull big toe to midline (adduct)	Adductor hallucis	Tibial	S1–S2
Pull big toe out (abduct.)	Abductor hallucis	Tibial	L5, S1
Flex little toe	Flex dig. min. brevis	Tibial	S1–S2
Abduct little toe	Abductor dig. min.	Tibial	S1–S2
Adduct little toe	Opponens digit. min.	Tibial	S1–S2

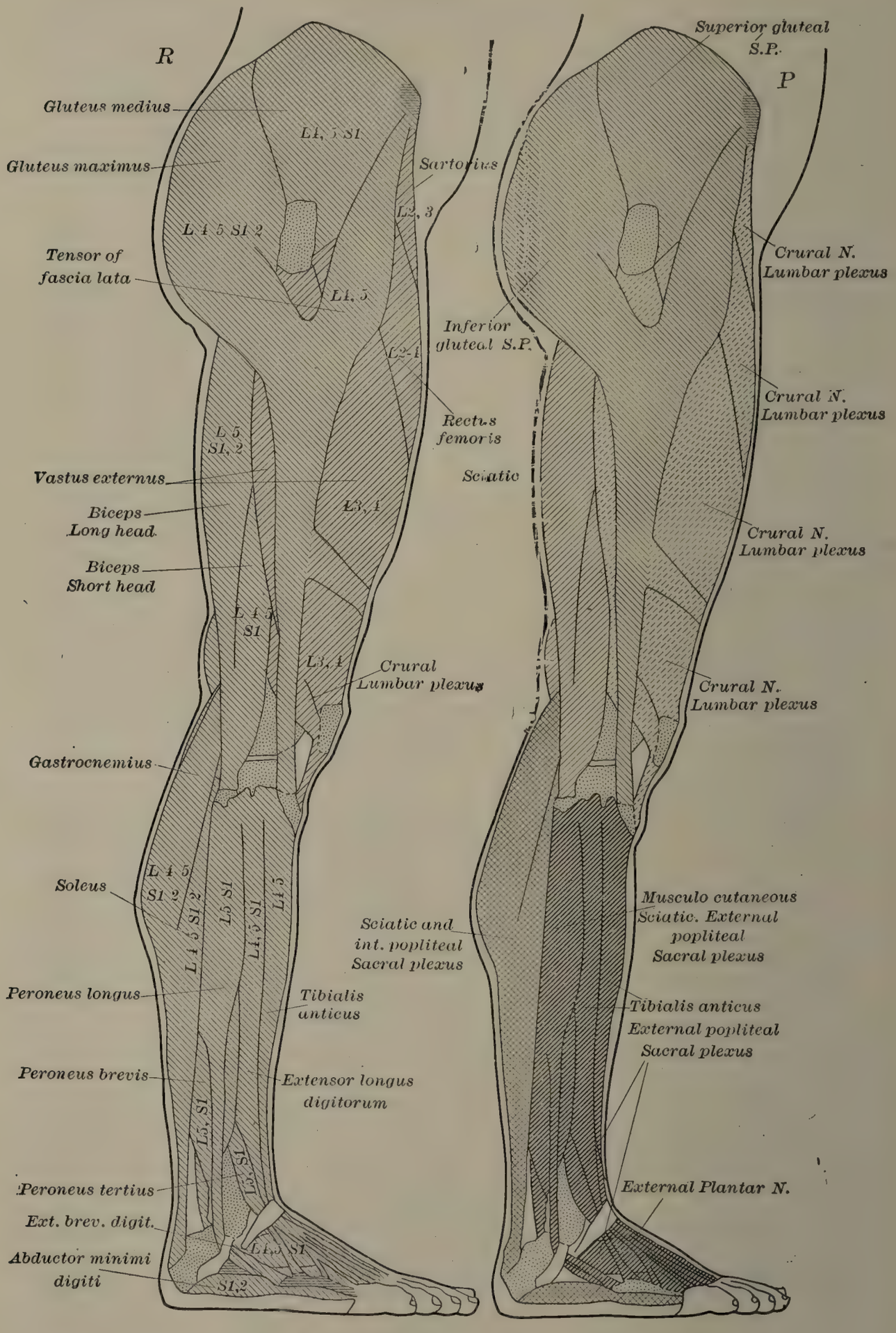


FIG. 31.—*Radicular* (*R*) (to left) and *peripheral* (*P*) (to right) innervations of the muscles of the external side of the lower extremity. Letters and abbreviations as in preceding figures. (After Dejerine.)

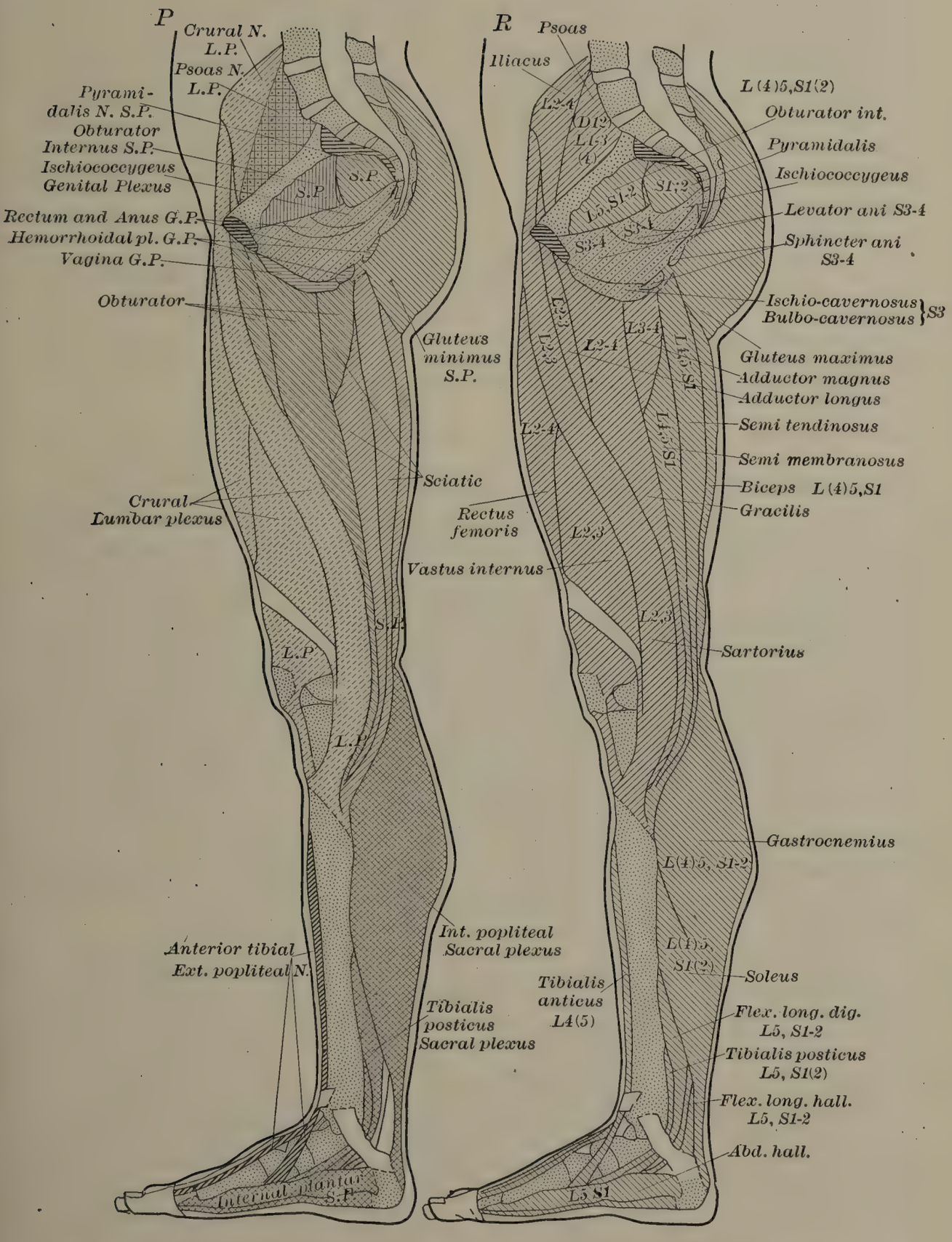


FIG. 32.—*Radicular* (*R*) (to right) and *peripheral* (*P*) (to left) innervations of the muscles of the internal side of the lower extremities. (After Dejerine.) *G.P.*, genital plexus.

Movements of the Toes at the Metatarso-phalangeal Joints.

Flexion:
Flexor longus digitorum.
Accessorius.
Lumbricales.
Flexor longus hallucis.
Flexor brevis hallucis.
Flexor brevis digitorum.
Flexor brevis minimi digiti.
Interossei.

Extension:
Extensor longus digitorum.
Extensor brevis digitorum.
Extensor proprius hallucis.

Abduction:
Abductor hallucis.
Dorsal interossei.
Abductor minimi digiti.

Adduction:
Adductors hallucis.
Plantar interossei.

Movements of the Toes at the Interphalangeal Joints.

Flexion:
Flexor brevis digitorum.
Flexor longus digitorum.
Flexor longus hallucis.

Extension:
Extensor longus digitorum.
Extensor brevis digitorum.
Interossei.
Lumbricales.
Extensor proprius hallucis.

Reflexes.—As this work is not one upon physiology no effort at defining what reflexes are will be attempted. The interested student will find complete details in Bayliss' General Physiology; Sherrington's, Integrative Action of the Nervous System Minkowski's study (l. c.). Magnus, Körperstellung, and Herrick's, Introduction to Neurology. One fact alone needs emphasizing. Since the entire body is all bound together by the nervous system through phylogenetic and ontogenetic experiences there are no simple reflexes. Even the simplest spinal reflexes (knee jerk, etc.) are extremely complicated, and the diagrams in the books are artificial abstractions. The entire body is involved in every reflex; physico-chemical, sensorimotor and symbolic systems all operate more or less.

Reflexes of Upper Extremities.[1]—*Superficial and Deep.*—These are then taken up. Those of the *cranial nerves* have been considered, but the important ones are here gathered together in tabular form:

Reflex.	Test.	Result.	Synaptic association.
Supraorbital.	Tap above supra-orbital foramen.	Contraction of orbicularis.	v–vii.
Conjunctival corneal.	Touching cornea or conjunctiva.	Contraction of orbicularis. Tears.	v–vii, vegetative.
Naso-mental.	Tap side of nose.	Contracture levator menti. Mucous.	v–vii, vegetative.
Palatal.	Touching palate.	Contraction palate	ix–x, vegetative.
Pharyngeal.	Irritation posterior pharyngeal wall.	Hypertonus of pharynx. Nausea.	Complicated, v–vii, ix, x, vegetative.
Pupillary skin.	Pinch side of cheek or neck.	Dilatation of pupil	v, vegetative.
Oculo-cardiac.	Pressure on globe of eye.	Slowing of pulse.	Sympath. v–x.

[1] Minkowski, M.: Zum gegenwärtigen Stand der Lehre von den Reflexen. Neur. *v.* Psych. Abhand. a. d. Schw. Arch. f. N. u. P., 1925, Heft. 1.

Reflex.	Test.	Result.	Synaptic association.
Auditory orbicularis.	Loud noise behind patient. Unknown.	Tightening of orbicularis; may be dilatation of pupil.	Vegetative sympath. viii.
Pupillary.	Exposure to light direct.	Pupils contract.	Complex pathway see p. 62.
Pupillary-consensual.	Exposure to light indirect.	Pupil opposite eye slighter contraction.	Complex pathway see p. 62.
Pupillary accommodation.	Look at near and far object.	Contracts and dilates.	Complex pathway see p. 62.
Pupillary hemiopic.	Irregular illumition.	Irregular contract.	Complex pathway see p. 62.
Winking.	Movement as if to hit face.	Closure of eyelids.	Complex pathway
Labyrinthine.	Movements of body balancing, etc.	Various positions and compensating symmetrical movements.	Complex pathway
Oculogyric.	Movements of eyeballs.	Compensatory head, neck and body adjustments.	Complex pathway

The important superficial and deep reflexes of the upper extremities are:

Elbow or Triceps Jerk.—This is best tested by supporting the patient's arm at the elbow by allowing the forearm to hang flaccidly over the edge of a chair. The stroke is made just above the olecranon, and the reaction consists in an extension of the forearm due to the contraction of the triceps muscle.

Radius Periosteal Reflex.—The radius periosteal reflex consists in a slight flexion of the arm on the forearm when the radius is tapped just three or four inches above the external condyle.

Supinator Jerk.—The supinator jerk is obtained by striking the muscle about midway between the elbow and wrist, the arm being supported at the wrist. It consists in a slight extension of the pendant wrist.

Reflex.	Test.	Response.	Synapse.
Scapular.	Pinch skin over scapula.	Shoulder blade rotates.	C5 D1.
Biceps.	Tap biceps tendon.	Biceps contracts.	C5 and C6.
Triceps.	Tap triceps tendon.	Triceps contracts.	C5, 6 and 7.
Supinator longus.	Tap radial styloid.	Supinator longus contracts.	C5 and C6.
Wrist.	Tap flexor tendons at wrist.	Fingers are flexed.	C6 to C8.
Carpometacarpal.	Tap back of wrist.	Fingers are extended.	C6 to D1.
Palmar.	Pinching skin of palm.	Fingers flex.	C8 D1.
Forearm (Levi).	Flex passively fingers and wrist.	Invol. contr. biceps flex. forearm.	
Mayers finger-thumb.[1]	Flex ring finger in palm.	Opposition of thumb extension of distal phalanges.	

Jacobsohn's radius reflex consists in a slight flexion of the fingers, particularly of the terminal phalanges when the radius of the extended

[1] Mayer: Neur. Centralbl, 1916, No. 1.

outstretched hand supported by the observer's hand is suddenly tapped with a hammer.

Other less studied reflexes are: Böttiger's stroking reflex; Trömner's finger phenomenon; Bechterew's metacarpophalangeal reflex; Stembos' palmar reflex; Gordon's finger phemonenon; Holzinger's hypothenar reflex.

Abdominal Reflexes.—The superficial and deep reflexes of the trunk should next be tested. Both sides should always be tested and recorded; O if absent, + if present; R = L or R > L, R > L, or R, o, L+, or *vice versa*, as the case may be.

Reflex.	Test.	Response.	Synapse.
Epigastric.	Stroke downward from nipple.	Epigastrium dimples.	D7 to D9.
Abdominal.	Stroke down from costal margin.	Abdominal muscles contract.	D11 to L2.
Cremasteric.	Stroke inner side of thigh.	Testicle is pulled up.	L1 to L2.
Gluteal.	Stroke skin over buttocks.	Gluteal muscles contract.	L4 to L5.
Bulbocavernosus.	Pinch dorsum of glans penis	Compressor urethræ contract.	S3 to S4.
Superficial anal.	Prick skin of perineum.	External sphincter contracts.	S5 and Conus
Pilomotor reflexes (See Thomas, loc., cit.)			
Thoraco-abdominal sup., med. and inferior.	Stroking chest, nipple, etc.	Pilomotor response.	D4, 5.[1]
Cutaneo gastric.	Stroking costal border.	Sounds of gastric contraction.	Vegetative.

Other less known reflexes here are Prevels heart sign; Livrerato abdominal heart reflex[2] (stomach punch), solar reflex (epigastric pressure causing feebleness of pulse).[3]

Reflexes of the Lower Extremities.—The *knee-jerk* (Erb-Westphal's sign) is one of the most familiar.[4] The knee-jerk may be tested in a variety of ways. One of the best is to have the patient sit upon a table, which permits the limbs to hang freely, then tell him to look at the ceiling, or divert his attention, the tendon just below the patellar is tapped; or the patient is directed to cross one leg over the knee of the other leg and the tendon is struck in a similar manner. Exaggerated, active, normal, sluggish, or absent responses should be recorded. Another method is to have the patient sitting, and the heels upon the floor but the legs comfortably extended. The tendon is then tapped. Here a simultaneous tapping of both tendons may be tried.

In certain patients the attention must be diverted, otherwise the leg is held rigidly which destroys the reflex. The patient may be

[1] See Sonderbergh: l. c. Barkman: Act. med. Skan., **57**, **61** and Laignel-Lavastine, l. c., for the abdominal sympathetic reflexes, also L. R. Müller: Lebensnerven. Glaser: Therapie d. Gegenwart, November, December, 1924. Goldflam: Rev. de Med., 1921.

[2] Brazil Med., November 29, 1924.

[3] Claude: Jour. d. Phys. et Path. Gen., 1924, **22**, No. 4.

[4] Hoffmann: Untersuchungen über die Eigenreflexe, Berlin, 1922, for extended discussion of tendon reflexes. Also Lewy: Tonus v. Bewegung, Berlin, 1923. Many papers by Wachholder and Altenburger: Pflüger's Arch., 1924, vol. **203**, et seq.

directed to repeat the Lord's Prayer, or compute small sums in arithmetic, or converse with an assistant, thus diverting the attention from the testing. Jendrassik thought of the expedient of utilizing a forced muscular act in the upper extremity to reinforce the knee-jerk. This may be carried out by having the patient make hard fists at a given signal, when the tendon is tapped, or by having him grasp his hands and pull at the given signal. By reinforcement a very weak knee-jerk may be made very evident.

Achilles Jerk.—This is best tested by having the patient kneel upon a chair, the feet being just free of the edge. The Achilles tendon is then tapped. The heel is pulled up.

For patients in bed, the leg should be everted, slightly flexed, and the foot extended to put the tendon on slight tension. The Achilles jerk is reported as absent without ascertainable pathology in about 1 in 500 individuals.

Ankle-clonus.—To elicit ankle-clonus requires some care. It is best obtained by supporting the patient's leg along the under side, the patient coöperating by thorough relaxation, then the free hand grasps the foot, and makes a sudden upward, dorsal flexion, holding the foot fairly firmly flexed at the end of the movement when a series of alternate extensions and flexions take place. The leg should be slightly everted, and the knee somewhat flexed. A false clonus consists in half a dozen flexions and extensions; true clonus continues for some time.

Plantar Reflex.—By stroking the sole of the foot, either at its external or internal border, a quick plantar flexion of all the toes including the great toe takes place. This is normal plantar flexion. As many individuals are ticklish, there is frequently a sudden jerking of the whole foot, or such a protective movement is manifested only in the tendons of the great toe. This should be distinguished from true dorsal extension or the *Babinski reflex.*[1] This consists in the comparatively slow dorsal extension of the great toe when the plantar reflex is tested and at the same time there is a slight spreading apart of the other toes.

If the patient is lying down—as he should be for testing—a fully developed Babinski sign as originally demonstrated by Babinski, consists in the slow dorsal raising of the great toe, the spreading of the other toes, a slight rotation of the thigh on the hip and a contraction of the fascia lata of the thigh.[2] In order to develop the whole test *the*

[1] By the term *dorsal extension* is here meant rising of the toes. By Babinski the term *plantar extension* was used. We are using the words in the ordinary sense, disregarding the fact that by some plantar extension is made synonymous with dorsal flexion, and plantar flexion with dorsal extension. See Babinski: Comptes Rend. Soc. d. Biol., February 22, 1896. Z. Bychowski: Presse Méd., 1922, **30**, 447.

[2] Consult Walshe, F. R. M.: Medical Science, 1920, and Brain, 1924, **37**, 269, for a discussion of this phenomenon following the Sherringtonian principles of reflex activity. Also see Elliott-Walshe: Lancet, January 10, 1925, for toxic causes of Babinski response. Rudolf: Phylogenetic Significance of Plantar Response, Jour. Neur. and Psychopath., 1922, **2**, 337. Of much importance, Brouwer. The Significance of Phylogenetic and Ontogenetic Studies for the Neuropathologist, Jour. Nerv. and Ment. Dis.. 1920, **51**, 113. Clauss, Babinski Reflex: Münch. med. Wchnschr., April 30, 1926, p. 733, Trömner: Deut. Zeit. f. N., 1923, **77**, 189.

feet should be warm, the thigh slightly rotated externally, the knee slightly bent, and the stroke made either on the outer or inner border of the sole by either a fairly sharp instrument, the finger-nail, or a blunt-pointed instrument. As there is great variation in the thickness of the skin of the soles of the feet, the various ways of bringing out a Babinski phenomenon should be tried in each case. Particular attention should be directed to the dangers of confusing the protective, pulling-away motion on tickling, which causes a marked quick dorsal raising of the great toe, with a true Babinski phenomenon. Monrad Krohn has called attention to the rare occurrence of a typical plantar response (Babinski type) in poliomyelitic cases. Hence rarely one must be on one's guard concerning the usual validity of the Babinski response as an *exclusive* pyramidal sign.[1]



FIG. 33.—Extension of the great toe on irritating the sole. The Babinski plantar extension phenomenon. (Chaddock.)

Careful record should be made of the irregularities in plantar response. In some patients there will be plantar flexion of the small toes but no reaction of the big toe. At times this may be as significant as a true dorsal extension. It is to be borne in mind that dorsal raising of the great toe is normal in infants and children up to the age of successful walking. See Mathieu and Cornil (*Paris méd.*, May 22, 1926) for variations of sign in infants.

A number of closely related reflexes have been described, having much the same significance as the Babinski sign, but being less constant, and at times contradictory. These are:[2]

Strümpell Reflex.[3,4]—This follows forceful pressure over the anterior tibial region with a resultant dorsal extension of the great toe. It is found in a number of conditions other than those of functional disturbance of the pyramidal tracts.

Oppenheim Reflex.—Here the inner surface of the leg is sharply and deeply stroked by the thumb from the middle to the ankle behind the malleolus. It brings out a great toe dorsal extension.

[1] Norsk Mag. f. Laeg., November, 1921, p. 805. Also see Hawthorne: Babinski Reflex in coma, British Med. Jour., September 12, 1925, p. 477.

[2] See Walshe, F. R. M.: Med. Sci., 1920.

[3] Noica. Sur le phenomen de Strümpell. Revue Neurologique, July 7, 1919.

[4] Feldman, W. M.: Nature of the Plantar Reflex in Early Life. Am. Jour. Dis. Child., January, 1922, also Mathieu et Cornil: Paris Med., May 22, 1926.

The Paradoxical Reflex.—Called by a variety of names, as described by Gordon,[1] it consists in a dorsal exension of the great toe following the grasping of the deep muscles of the calf and making a forceful indention along their external border.

Mendel-Bechterew.—This reflex consists of the dorsal extension of the toes, especially the second and fifth, when the dorsum of the foot is tapped about at the base of the middle toes. In reflex irritability this reflex is augmented. If plantar flexion takes place the authors regard it as a sign of organic affection.[2]

Chaddock.—This produces a toe extension by stroking the side of the ankle (Fig. 34).



FIG. 34.—The Chaddock modification of the Babinski, causing great toe extension on stroking beneath the external malleolus. (Chaddock.)

Other Lower Extremity Reflexes.—Analysis of the numerous war lesions of the spinal cord have added a large number of reflex anomalies to those previously known. These cannot all be mentioned here, but among the more valuable of these newer findings are the medio-plantar, tibio-femoral, perineo-femoral, non-pyramidal clonus, contralateral adductor, defense reactions (Marie Foix type).[3]

Contralateral-adductor Reflex (*Balduzzi*[4]).—Patient lying with separated legs one percussed the midportion of the sole of the foot. A positive reaction consists of a contralateral inner rotation and adduction of the opposite leg. Occasionally a slighter contraction on homolateral side. It is only rarely found in healthy individuals and chiefly in those with active knee-jerks. It is constant in pyramidal

[1] Goldflam: Valeur clinique du signe de Gordon Rev. Neur., 1925, vol. **32.**

[2] Krug: Leipzig Dissertation, 1911.

[3] These are best summarized in Guillain et Barré's, Travaux Neurologiques de Guerre, Masson et Cie, Paris, 1920.

[4] Balduzzi: Un nouveau réflexe contralatéral des muscles adducteurs, Rev. Neu., 1926, **33**, 243.

lesions from various causes. L1,S2 is given as its segmental spinal localization by its describer.

Medio-plantar Reflex.—The patient lying on the face, the leg flexed on the thigh at a right angle. The midplantar region between the calcaneum and metatarsus is struck with the hammer. Or in the kneeling position, as for the testing of the Achilles the same region is percussed. A double movement results. There is an extension of the foot from the leg and a flexion of the four digits. The former is a true spinal reflex, the latter a local muscular reaction. The spinal reflex is a normal constant. It is absent in tabes, lumbo-sacral radiculitis, polyneuritis and often during epileptic seizure. It is exaggerated in hemiplegia, spasmodic paraplegic cases of many origins (multiple sclerosis, tumor, Pott's, etc.). and in many irritative neuritides. It seems to behave like an Achilles reflex but is independent of this reflex Guillain and Strohl have found these reflexes dissociated. Its synaptic site seems to be S1-2.

Posterior Tibio-femoral Reflex.[1]—This is a reflex involving the semitendinosus and semimembranosus muscles first suggested by Dejerine. The patient in recumbent position, the leg is abducted and partly everted and relaxed. The left hand pressing slightly above the knee, the insertion of the muscles is percussed a few inches below the knee on the internal aspect. A tendon and a periosteal reflex are obtainable. They are dissociable. A contraction visible to the eye and to feel occur in these muscles. This reflex is abolished in cord lesions which destroy L4-5 S1., as in tabes, radiculitis (4–5th lumbar roots, first sacral root) and certain neuritides. It is not infrequently conserved in mild sciaticas, but in certain severe sciaticas it is abolished, with the Achilles and medio-plantar reflexes. It may afford with its preservation, and abolition of these later, localizing significance. It is exaggerated in pyramidal tract lesions.

Posterior Perineo-femoral Reflex.[1]—This is a reflex of the quadriceps femoralis, also suggested in Dejerine's Sémiologie. Here the patient lies on the sound side, lightly flexing the thigh and the leg. The lower end of the tendon is percussed, or on the head of the peroneus, causing a contraction of the quadriceps. The point percussed is a little higher than that for the posterior tibio-femoral reflex. Often the two reflexes are mingled. The synapses involved lie in L5 S1-2. This reflex is lost in cord lesions involving these areas in tabes, radiculitis, neuritides and sciatic lesions situated above the emergence of the biceps nerve. In most simple sciaticas it is conserved. It is often lost with loss of Achilles and medio-plantar reflexes, but may be dissociated. It is exaggerated in pyramidal tract lesions.

Defense Reflexes (Marie-Foix).—This general type of reflex is of much significance. It is a very complex phenomenon and only its chief manifestations can be mentioned. It has in certain of its manifestations been observed by numerous clinicians. There are many modes

[1] Guillain et Barré, loc. cit.

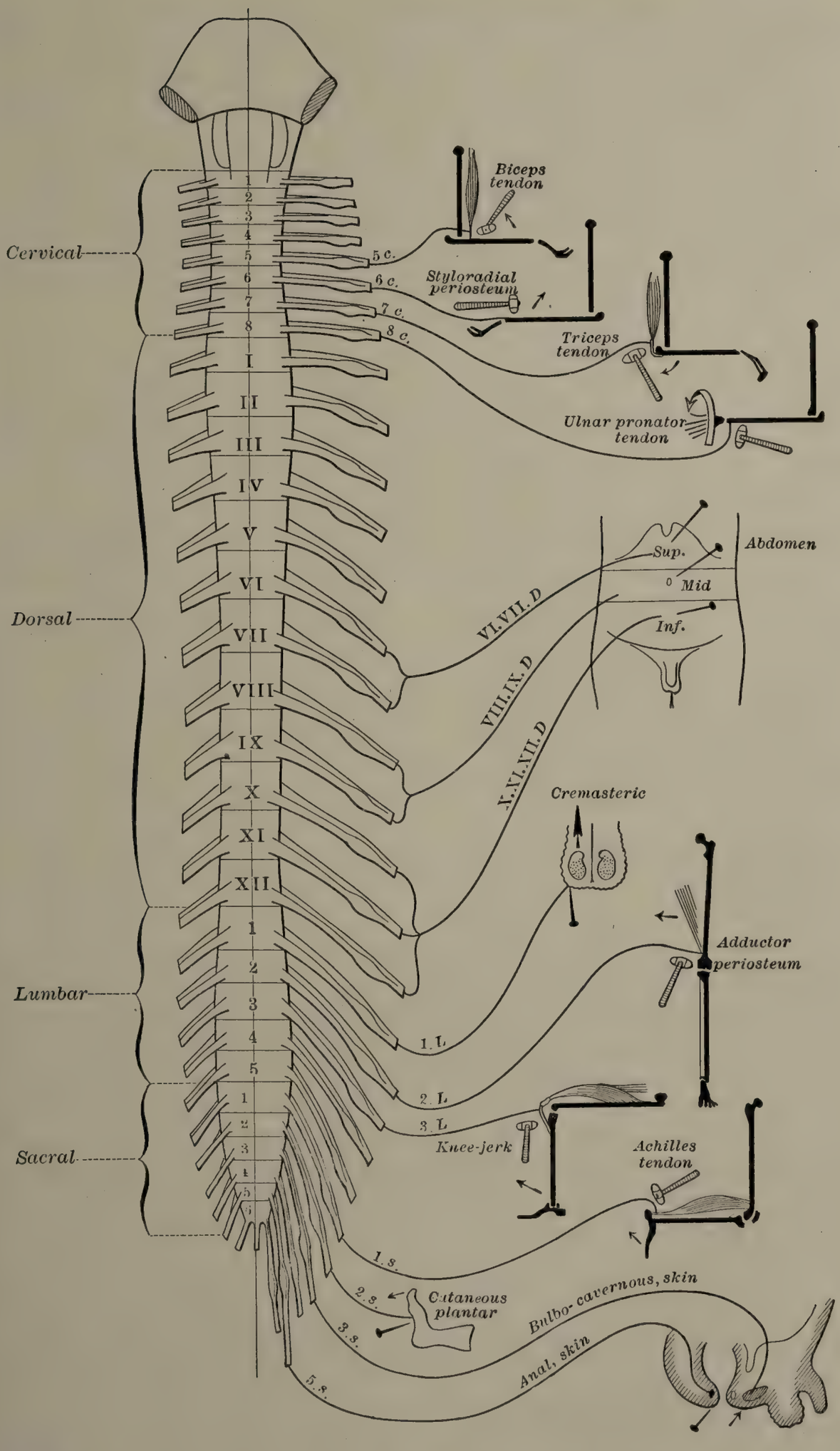


FIG. 35.—Scheme of principal spinal reflexes, (after Lévy Valensi)[1] with pathways involved.

[1] J. Lévy Valensi. Valeur diagnostique des reflexes. Paris Méd., July 22, 1922.

of eliciting certain of its manifestations. Originally Marie and Foix brought out a contraction of defense by squeezing the toes or by strongly flexing the toes on the foot. In comatose patients not only the leg of the affected side drew up but the sound side showed marked defense movements. Later it was found by strongly pinching the skin of the foot, or the skin of the abdomen, or even of the skin of the chest by an artery clamp, these defense movements were induced. These consist chiefly in an extension of the spastic or paralyzed leg, with similar though less marked extension of the spastic, or paralyzed extremity when the skin of the sound side is pinched.

Reflex.	Method of obtaining.	Result.	Location.
Knee.	Tap patellar tendon.	Leg extended.	L3 and L4.
Achilles.	Tap tendo Achillis.	Plantar flexion foot.	S, S4.
Ankle-clonus.	Sudden dorsiflexion foot.	Quick up-and-down movements.	Pyramidal tracts (L3, S2).
Plantar.	Stroke soles of feet.	Plantar flexion all toes.	L3, S2.
Adductor foot reflex.[1]	Stroke inner border foot.	Raise inner border, extend ankle.	
Babinski.	Stroke soles of feet.	Dorsal extension great toe.	Pyramidal tracts (L3, S2).
Oppenheim.	Stroke inner side of calf.	Dorsal extension great toe.	Pyramidal tracts (L3, S2).
Paradoxical.	Deep pressure in calf.	Dorsal extension great toe.	Pyramidal tracts (L3, S2).[2]
Medioplantar.	Percussion of midplantar.	As Achilles	S 1–2.
Contralateral adductor.[3]	Strike sole of foot. Adductor mid sole.	Inner rotation opposite leg.	L4, S2.
Posterior tibio-femoral.	Percussion semitendinosus.	Semimembranosus contract.	L4–5, S1.
Posterior Peroneo-femoral.	Percussion tendon quadriceps.	Contract.	L5, S1–2.
Piotrowski.	Percussion ent. malleoli.	As Babinski.	Extend ankle.
Cerebellar knee-jerk.[4]	Knee tap.	Pendular swing on ipsilateral side.	Cerebellum.

Back Reflexes.[5]—Little has been made of the reflexes of the muscles of the back in disease. In previous editions of this work practically only the pilomotor posterior reflexes has been considered. Bechterew (1900) early described a *lumbo-femoral reflex*. Tapping of the sacral and lumbar region induced a contraction of the erector spinæ and superficial gluteal regions.

Noica in 1912 showed a shoulder elevation reflex. Galant in 1917 showed in children how stroking alongside the spinal cord caused a bending of the entire sacral column. This type of reflex disappears after about one year and is present only in idiots or imbeciles in later

[1] Hirschberg: Rev. Neur., August 15, 1903. Marie et Meige: Rev. Neur., 1916, p. 420.

[2] For a complete discussion of reflex action see Sherrington: Jour. Physiol., 1910, vol. **4**. The complicated problem of medullary reflexes is amplified in a thesis of A. Stahl, Paris, 1913.

[3] Balduzzi: Rev. Neur., 1926, **33**, 243.

[4] Holmes, G.: Brain, 1917, **40**, 507.

[5] Ketrorozeff, A.: Ueber die Rückenreflexe des Menschen. Zurich Dissertation, Vogel. Leipzig, 1926 (see for literature).

life. v. Monakow showed it in certain cerebral pyramidal lesions. Veraguth in 1917 first attempted a systematic study from which study the following briefly noted reflexes upon the back are developed.

The back dermatomeres, D1-12; L1 S3-4, constitute reflexogenic zones as truly as the chest or abdomen. In infants these reflexes are striking. Veraguth has termed them perisacral back reflexes. They are not limited to the lower sacral regions, however, but may invade the neck. Care must be exercised not to confuse them with the head and neck righting position reflexes of Magnus and de Kleijn. In certain bilateral hemiplegics, "Little's" cases they are easily evoked. For often a child in order to get away from a stroke along the spine will curve away from the irritatant; the new bodily position now sets in motion the Magnus phenomena. Thus the arm and neck movements are not true back reflexes in Galant's sense. The Bechteref, Galant and Noica reflexes may be provisionally accepted as back reflexes. Pathological reappearances of these strong infantile reflexes occur in spinal injuries, in cerebral lesions, in deep back muscle lesions, cerebellar lesions, Little's disease in particular. Their study in the hyperkinesias of encephalitis should offer interesting material.

Types of Vegetative Reflexes.—The clinical relationships and interdependencies between vegetative reflexes, sensations of pain, and emotional stimuli are extremely intricate, and up to the present time have not been codified into an orderly series. Here only a few empirical data are recorded. The student is referred to special works upon the general subject.[1] Mackenzie's reflex or *referred* phenomena it is to be remembered are clinical signs and not exact expressions of an anatomical or physiological reflex arc activity.

Mackenzie speaks of the viscera as showing sensory, motor and visceral reflex activities.

I. The *sensory reflex* which serves for protection begins as a sensory stimulus in the zone of the organ concerned. This is either internal or splanchnic and is felt as pain, though all authors are not agreed upon this, or external or somatic, where pain is felt in hyperesthetic reflected zones. The following are the characteristics of these sensations of pain:

(*a*) Splanchnic pain usually is in the midline, even if the organ by which it is caused lies on one side or partly so (esophagus, stomach, liver, intestines, kidneys).

(*b*) Contrary to the pain of hyperesthetic zones, splanchnic pain is not relieved by moderate chloroform narcosis.

(*c*) The radiating external pains are never precisely defined.

(*d*) The radiating external pains are felt in deeper structures (muscles, breast) not in the superficial layers of the skin.

(*e*) Artificial stimuli as alcohol, mustard plasters, hot applications, cantharides and the galvanic or faradic currents prevent viscerofugal

[1] Vasomotor Reflex. Ogata and Vincent: Jour. Comp. Neur., 1919, **30**, 355, Mackenzie Symptoms and their Interpretation; Pottenger: The Vegetation Nervous System and Internal Medicine; Higier: The Vegetative Nervous System and the large work of Laignel-Lavastine and L. R. Müller (l. c.).

stimuli from passing through the spinal roots to the hyperalgesic zones. They act as quieting, pain-relieving agents (derivantia et revulsiva).

(*f*) Both types of pain, visceral and radiating, are increased by, if not originating, in intense emotions, as fear and anger.

II. The *motor reflex* usually consists in some stimulation in that part of the external musculature corresponding to the organ of stimulation. Examples are contraction of the chest muscles, particularly the large spinal muscles "*signe des spinaux*," which occurs in pleuritis, and contraction of the abdominal muscles in diseases of the stomach, intestines and liver. Sherrington was one of the first to demonstrate the role of the efferent sympathetic nerves in these reflexes. He cut the abdominal visceral nerves and stimulated their central ends. There resulted a distinct contraction of the abdominal musculature corresponding to the nerve which was stimulated. This visceromotor reflex stimulation gradually disappeared on cutting the posterior roots which carried the stimuli from the visceral nerves.

III. The *organ reflex* is usually a secretory, peristaltic or antiperistaltic activity in the organ which is stimulated. Examples are salivation, gastrosuccorrhea, vomiting, accumulation of mucus in the bronchial tree and hiccough. In all these reflexes the primary sensory irritation plays the main role (Head, Mackenzie, Forster).

Tabetics often do *not* show these reflex phenomena because the visceral reflex fiber pathways are interfered with. (Visceral anesthesiæ). Such failures to respond normally occur as follows: cremaster reflex after stimulation of the testicle; pain and abdominal contractions during parturition, abdominal pain, vomiting and muscle spasm in appendicitis.

Toothache often gives a vicerosensory hyperesthesia of the cheek.

The pain in the shoulder of the same side as in pneumonia is comprehensible because the sensory innervation of the shoulder is related to the same spinal segments (C4, C5) as the phrenic nerve supplying the pleura.

The severe pain of pleuritis caused by deep breathing is not due to direct irritation solely, but is also of a referred visceromotor reflex of the intercostal and spinal muscles.

In certain heart disease pains felt in the arm and thoracic region the connection is to be found in the same spinal segments (D3, D4) as certain vegetative cardiac synapses. The rare occurrence of pain in the skin of the neck and the left neck muscles is made possible by the afferent vagus fibers which carry the stimuli over into the second and third cervical segments. A more significant visceromotor heart reflex is the feeling of oppression which results from a radiating spasm of the intercostal muscles. A cardiac organ reflex of less significance is the salivation and profuse urination which occurs in angina pectoris, both symptoms due to reflex shunting through the vagus synaptic junctions (nuclei) located about the floor of the fourth ventricle.

The feeling of cold in the stomach after drinking cold water is attributed to contraction of the skin bloodvessels and is accompanied by a

circumscribed area of goose-flesh. Thus two visceral reflexes, one pilomotor, the other vasomotor are observable. The visceromotor reflex contraction of the upper left rectus abdominis in gastric ulcer is explained by the synaptic switching of the sympathetic gastric fibers, D6, D7. The heartburn which results from regurgitation into the esophagus localizes itself one segment higher. Pain of gastric origin rarely directly locates an ulcer or carcinoma. It is usually in the median line, and in cardiac, fundal and pyloric disease is found in the upper, middle and lower part of the epigastrium.

Cutaneo-gastric Reflex.—Described by Mitchell[1] consists in reflex movements of the stomach, heard by phonendoscope over abdomen as the skin is stroked along the left costal margin. Bubbles of gas are expelled through the pylorus, or later crunching sounds or still later the rush of fluid into the duodenum.

A very interesting referred organ reflex in appendicitis is difficulty of urination which is not infrequently associated with the hyperesthesia over McBurney's point.

Pain in the upper arm in liver disease is explained by the associations in the same spinal cord segment, C4, of the nerves of the arm and the phrenic nerve to the liver.

In disease of the kidney pelvis and ureters the viscerosensory reflex is referred to the testicles; the visceromotor to the cremaster and transverse muscles since the nerve supplies are associated in the eleventh and twelfth dorsal and first and second lumbar segments. For this reason the testicles and the scrotum are sensitive in cases of nephrolithiasis.

Mass Reflex.[2]—In complete spinal cord severance segmental activity may persist. The mass reflex consists in bladder, and rectal contraction, involuntary, with sweating and flexion of the lower extremities. (See more fully under Spinal Cord.)

Since the upper portion of the bladder is developed from the allantois (L2) and the lower from the cloaca (S2,) the reflex pain depends upon the site of the disease. Sometimes it is above the pubic bone, sometimes in the penis and perineum. The visceromotor reflex causes spasm of the sphincter ani.

Reflex pain from the uterus and ovaries lies in the lower abdomen.

Pain in the knee in hip-joint disease is due to the association of the spinal segment supplying fibers to these two joints, L4. The synovia are supplied by vegetative nerve fibers just as is the peritoneum.

Tremors.[3]—The tremors of the upper extremities first claim attention. Those of the head may be fine and oscillatory, nodding, rhythmical, or jerky. The rapidity of the tremors should be noted.

[1] Mitchell, P.: Lancet, January 28, 1911.

[2] Head and Riddoch: Brain, 1918, **40**, 188; Riddoch: Lancet, 1918, ii, 829.

[3] Extremely complex situations: Advanced students are referred to special works. Pelnar, Gorden Homes, Especially F. H. Lewry: Tonus u. Bewegung, Berlin, 1923, for complete bibliography and specially paralysis agitans tremors. Barker: Monographic Medicine, vol. **12**. Purves Stewart: Diagnosis of Nervous Diseases. Dejerine: Semeilogie d. Systeme Nerveux.

Tremors of the hand and wrist should first be tested with the pendant hand supported at the wrist. Then with hands extended and fingers wide apart *static* tremors come out. Or with the hands in motion *locomotor* tremors become evident.

Static tremors are either fine and rapid (eight to twelve per second), or coarse and slow (four to six per second). They may be irregular. When involving the whole arm one speaks of movement rather than tremors.

Intention tremors are those increasing by voluntary action. Ataxias are brought out best by having the patient bring his index fingers from any position slowly to the end of the nose, first, with eyes open, then with eyes closed, test both sides (finger-nose test—F. N. T.), or the index fingers should be brought to touch each other (finger-finger test —F. F. T.). Here coarse irregular movements (ataxias) may be brought out. The patient's finger may overshoot the nose (dysmetria). The patient's ataxia increases markedly as the nose is reached (intention tremor), or increases only just as the object sought is arrived at.

Ataxia in the lower extremities is tested by the knee-heel test (K. H. T.), the patient, on his back, is directed to touch the left knee with the right heel, and *vice versa.*

Athetoid movements are coarse, slow, sinuous, progressive, rhythmical movements in the fingers, arm, or trunk.

Choreic movements are irregular, coarse, or fine movements, non-rhythmical and non-coördinated—they are jerky movements.

Facial Associated Movements.[1]—A description and discussion of various associated movements of the facial muscles, evoked mainly by lateral ocular deviation. Six main associated movements are described: (1) The oculo-aural movement, whereby lateral ocular deviation is accompanied by a bending back of each pinna particularly on the contralateral side. (2) The oculo-nasal movement; here wide dilatation of the nostril is evoked by turning the eyes laterally. (3) The oculo-mandibular movement; this comprises protrusion of the chin and deviation to the opposite side on lateral ocular deviation. (4) The oculo-lingual movement, in which the tip of the tongue moves over to the same side as that to which the eyes are turned. (5) The oculo-frontalis movement, whereby the ipso-lateral eyebrow is elevated and the contralateral one depressed on lateral ocular deviation. (6) The possibility of contraction of the stapedius being associated with screwing up of the eyelids or with turning of the eyeballs to left or right, is discussed. The nature of these movements is discussed from the anatomical, phylogenetic and embryological standpoints; reasons are given for not regarding these as phenomena of a reflex nature. A study of various animals reveals their presence in certain species, depending chiefly upon the mode of life adopted by these animals. The nature of these movements would therefore appear a device

[1] Carmichael, E. A. and Critchley, M.: Jour, Neurol. and Psychopath., London, 1924, **5**, 124.

whereby the senses of sight, smell and hearing are simultaneously brought to bear upon an object.

The authors furthermore describe various examples of "facial nystagmus" evoked by lateral ocular deviation. Most of the patients showing this phenomenon were suffering from disseminated sclerosis and it is thought that it may be a point of diagnostic importance as ocular nystagmus was absent in most of these cases. Two main varieties are described: (1) Nystagmus of the external ear; (2) nystagmus of the eyebrow.

Associated Reflexes.—Among the simpler of the complicated associated reflexes the so-called Kernig, Bikeles' and Brudzinski's neck signs may be mentioned.

Kernig's Sign.—The patient lying upon the back, the thigh is slowly or abruptly flexed upon the trunk to a right angle. A reflex contracture of the hamstring muscles causes *resistance to this procedure.* It is usually an indication of meningeal invitation.

Bikeles' Sign.—Raise the arm up and back, the presence of *passive* resistence to extension to passive movement, is analogous to the resistence in Kernig's sign. It also is a partial indication of meningeal irritation but may also be present in neuritis.

Brudzinski's Neck Sign.—Of similar import to Kernig's sign. It consists in partial flexion of the hip and knee-joints when the head is flexed passively upon the chest.

A much more complicated series of reflexes have been described by Magnus and de Kleijn known as position-posture reflexes, labyrinthine head and neck reflexes, etc. These are reflexes which have been determined by physiological experimentation with lower animals and are related to complicated proprioceptor impulse equilibrium. The study of the pathology of the striatum, as in Wilson's disease, postencephalitic Parkinsonism, paralysis agitans, chorea and postural disorders, in general, has brought them into prominence. Two general groups may be distinguished: an *elementary* series of posture reflexes, more exactly delimited by Foix and Thévenard, Delmas-Marsalet;[1] and a secondary *compound* series studied in great detail by Magnus[2] and his school. The most outstanding of these are the *righting reflexes* so valuable for all equilibrations.

The Righting Reflexes.[3]—The righting reflexes (first defined anatomico-physiologically by Magnus and De Kleijn of Holland), are reflex movements operating to keep the animal in its optimum posture whenever there is a departure from it. The stimuli activating this this neural mechanism come from the labyrinths, eyes, body surface acting on the head and body, and the neck-axial muscles and tendons. The *midbrain* and interbrain contain the centers.

[1] Delmas-Marsalet: Études cliniques sur les reflexes de posture elementaire, Jour. d. Med. de Bordeaux, November 10, 1926, p. 819.

[2] Magnus: Körperstellung, Berlin, 1924, Rademaker: Berlin, 1925. Monographs of Kinnier Wilson, Lewy, Jacob, C. H. Vogt (striatum).

[3] Brock, S. and Wechsler, I.: Loss of the Righting Reflex in Man with Special Reference to Paralysis Agitans, Arch. Neur. and Psych., 1927, **17**, 12,

In analysing paralysis agitans Brock and Wechsler state that tremor may be absent, that paralysis of voluntary movement is wanting and that even rigidity may be missing. On the other hand, these authors stress the importance of the ever-present loss of associated, automatic movement and the "flexion" posture and believe that these are the *kinetic* (or motile) and *static* phases of a disturbance of one underlying righting mechanism in man. This loss of a particular righting response causes a "dissolution of erectness" (W. M. Kraus) on the *static* side. These writers interpret the lack of "reactive movements" (O. Foerster) of flight, fright, defense, surprise, etc., in paralysis agitans as *kinetic* losses in the sphere of the righting reflexes. The various pulsions and gestinations seen in this disease illustrate the same defect brought out by the simple stimulus of a shifting center of gravity.

Going further, Brock and Wechsler believe that with man's assumption of the upright posture and his newer evolved capacity to give motor facial expression to his emotions, the facial mimetic motor patterns became an added affective component to his righting reflexes, which in the lower animals are almost purely somatic responses. In this way, they account for the loss of emotional expression in paralysis agitans.

In most cases the coexistent rigidity "strangles" the musculature preventing the investigation of these reflexes.

Analysis of certain signs described by Souques and von Sarbo is clarified by interpreting them as disturbed righting responses. Souques suddenly tilts backward the chair in which a patient is seated. Normally there is sharp extension of both lower extremities at the knee-joints—an attempt to restore erect posture. In paralysis agitans the knees remain flexed—a loss of this righting adjustment. von Sarbo described a sign, "hyptokinesis" consisting of retropulsion on strong dorsiflexion of the head. In some cases this induced backward swaying, in others it was necessary to close the eyes as well. He felt that the sign was caused by a lesion in the nucleus ruber system. Brock and Wechsler explain the sign as follows: Tilting of the head backward displaces the center of gravity enough to produce a loss of righting in certain cases. In others, shutting the eyes eliminates the optic righting reflexes and intensifies or first elicits this loss of righting. The familiar Romberg sign is mentioned as a loss of an optic righting reflex.

These authors conclude that "dissolution of erectness," loss of "reaction movements," loss of associated and automatic movements (J. R. Hunt) may be reinterpreted in the newer physiologic terms of righting reflexes in man. Further investigations are necessary to define the exact anatomical pathways and physiological basis for this new contention.

Tics.—These are involuntary, uncoördinated movements of complex origin. They often resemble *myoclonic* movements which are more properly twitch-like movements in parts of a muscle.[1]

[1] Kinnier Wilson, Tics: Jour. Neur. Psychopath, 1927.

Diadokokinesis is a term used to define the ability to perform alternate rapid coördinated movements, of antagonistic muscles. Certain patients show a loss of this ability (adiadokokinesis). The tests most frequently applied are quick pronation and supination of the semiflexed hand; piano-playing movements or quick flexion and extension of the forearm on the arm. The term is applicable only in the absence of motor paresis or gross anesthesia.[1]

Apraxia.—This, speaking generally, consists in the loss of ability to perform purposeful movements. The tests are to have the patient throw a kiss, make a salute, a beckoning gesture, a threatening gesture, or to go through an imaginary act; such as taking a match out of a box and lighting it or blowing it out. It is also elicited by testing the movements necessary to use objects correctly.

EXAMINATION OF SENSORY NERVOUS SYSTEM.

The most important of the tests of the sensory nervous system are for: (1) light touch, (2) pain, (3) thermal sensations, and (4) deep sensibility. Head has suggested the terms epicritic, protopathic, and deep sensibility for three types of sensibility which he differentiates. While there are many excellent reasons to refer Head's protopathic sensibility to the vegetative (affective) systems, the original point of view of this investigator is here maintained with reservations, until this intricate problem of sensibility becomes more definitely formulated.[2]

1. Epicritic sensibility is that which recognizes light touch, distinguishes small differences between the points of a compass, and recognizes small variations in the temperature of objects.

2. Protopathic sensibility recognizes pain and extremes of heat and cold. It and the following function partly at least through the vegetative system.

3. Deep sensibility recognizes deep pain and muscle and joint sense. Bony sensibility is included here.

Epicritic Sensibility.—To test epicritic, or gnostic sensibility the *light touch* is employed. Touching the skin with the end of the finger is not a light touch test. Such a test is too coarse. It is a test for deep sensibility because it distorts the skin surface. Either a wisp of cotton-wool should be used or a fine camel-hair brush. For most purposes the cotton-wool is to be preferred. In accurate testing, special esthesiometers are indispensable. Those mostly in use are von Frey's hairs or Franz's simple esthesiometer.

The patient's body should be explored systematically. He is asked to say "Yes" every time he is touched, and then asked to localize the spot touched. The testing should be made with the patient first lying down, and due attention should be given to the character (thickness, etc.), of the individual patient's skin in drawing conclusions from the

[1] Plastic tonus and proprioceptive reflex, Sherrington: Quart. Jour. Physiol., 1909, vol. 2. See chapter on Cerebellum.

[2] Byrnes, J.: Jour. Nerv. and Ment. Dis., 1919; ibid., 1922.

tests. In going over the extremities care should be taken to circle the limb with the touches as well as going up and down. It is specially desirable to avoid suggestive questions, such as, Do you feel this?

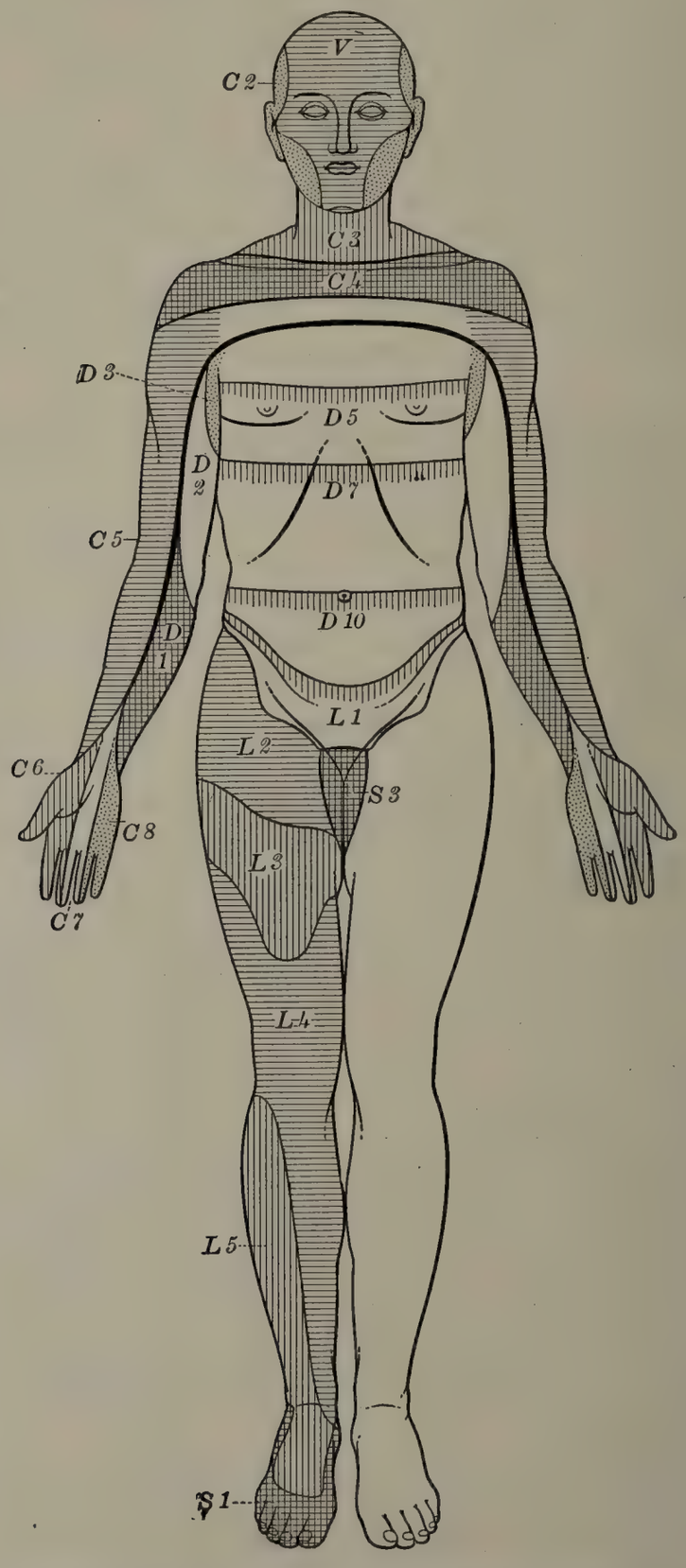


FIG. 36.—Illustrating the segmental spinal sensory areas. Front view.

What do you feel? etc. In certain conditions in which symbolic factors are paramount the prompt response "No" is obtained at each touch over the so-called anesthetic area. This is a finding pointing to such symbolic or psychogenic factors.

Modifications of light touch should be charted and marked on the skin with an anilin pencil. Such indications are very useful as landmarks for localization. Anesthesia or hyperesthesia to light touch

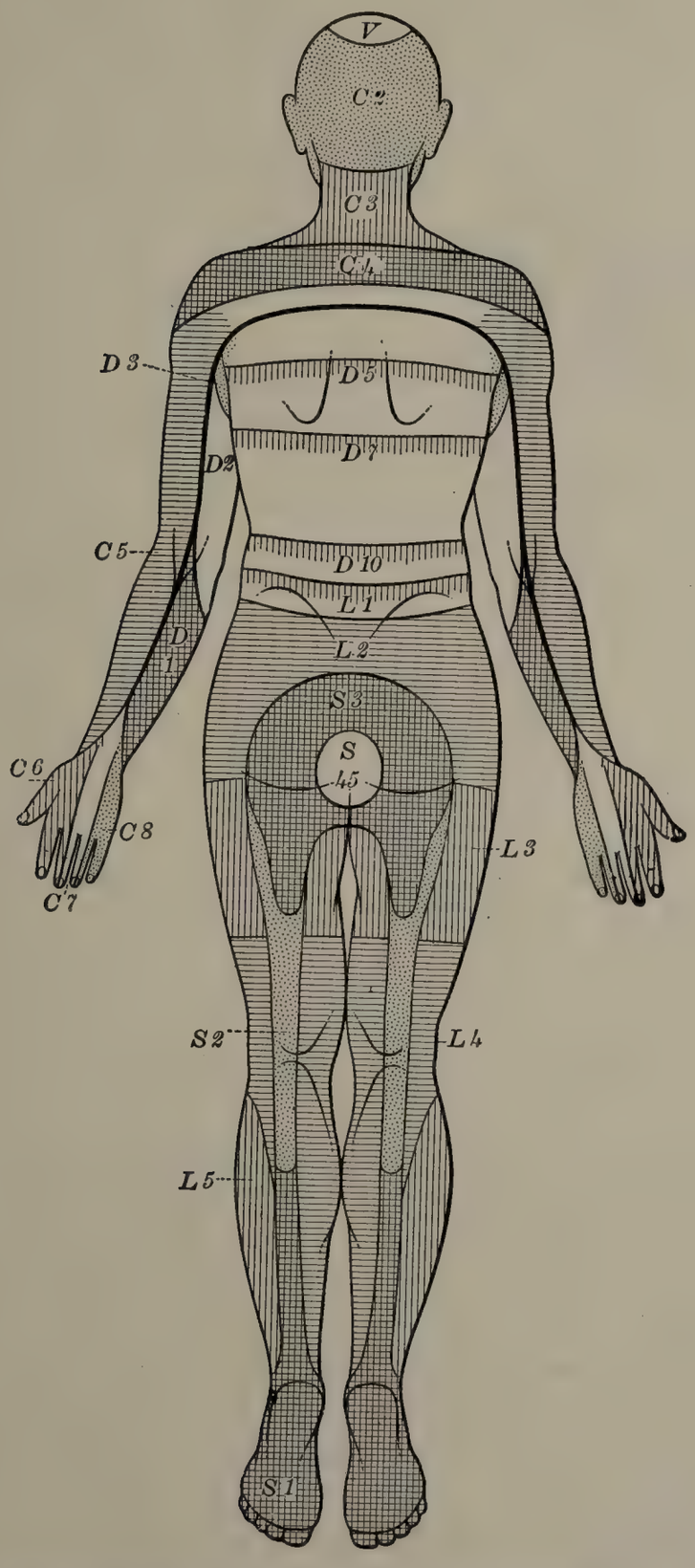


Fig. 37.—Illustrating the segmental spinal sensory areas. Rear view. These or similar sensory charts are useful in plotting sensory changes.

should be charted. In all hairy parts the skin should be shaved for accurate testing, otherwise cotton-wool drawn across the skin by bending the hairs will deform the surface and so give a deep sensibility

response to test for light touch. (See Plates X and XI for the pathways involved.)

A Weber compass is useful for testing the individual capacity for recognizing one or two points. One devised by Gordon Holmes is most practical. There is a great variability in individual discrimination and in sensibility in different regions. Some of the available

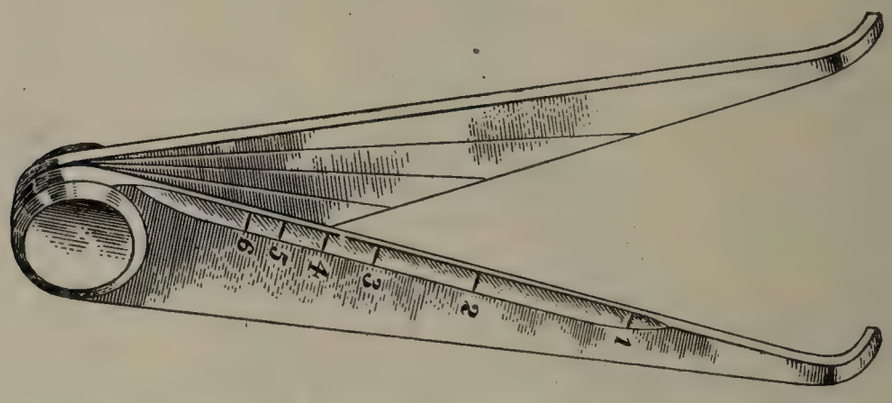


FIG. 38.—Holmes's compass for testing touch discrimination.

figures for the minimum separation distance recognized as two points are as follows:

Tongue	1.5 mm.
Finger-tips	2.3 mm.
Lips	3.4 mm.
Dorsum of fingers	5.6 mm.
Forearm	9.1 mm.
Forehead	10 to 15 mm.
Neck	23 to 30 mm.
Leg: back of foot	30 to 40 mm.
Back	50 to 60 mm.
Arms and thigh	70 to 80 mm.

Further tests may be made of gnostic light touch by placing variously shaped objects on the skin. Such tests are very valuable in special cases.

Epicritic thermal sensibility. This is most easily tested by use of the back of the finger for warm, and the metal head of a percussion hammer for cool. Gnostic thermal sensibility recognizes differences as small as 2 to 5 degrees of temperature, while affective sensibility is unable to recognize differences between 40° and 20° C. Loss of gnostic sensibility for heat, with preservation of affective thermal sensibility, is not uncommon. The reverse is also found.

In research work an electrical thermometer or thermophile is useful. In ordinary routine work test-tubes with ice water and hot water may be used, or metal tubes which have been plunged in cold or hot water. It is not sufficient to test patients for extremes of heat and cold alone; minute differences should be tested for as well.

Protopathic Sensibility.—*Pain.*—This is quickest tested by pinching the skin between the nail and the finger. A sharp-pointed pin with a round glass head is also useful. The patient is asked to distinguish between head and point. Absence of pain (analgesia) should be care-

fully charted as well as increased pain sensibility (hyperalgesia). The limbs should always be tested in their circumference as well as in their length, care being taken not to overlook thin strips of analgesia from radicular lesions. Hair sensibility should also be tested by pulling the hair. Painful faradic stimulation is at times of value in determining the value of an existing analgesia.

Deep Sensibility.—Here deep pressure pain, muscle and joint sense and bony sensibility are to be tested. Deep pressure with the thumb and fingers, or a special instrument

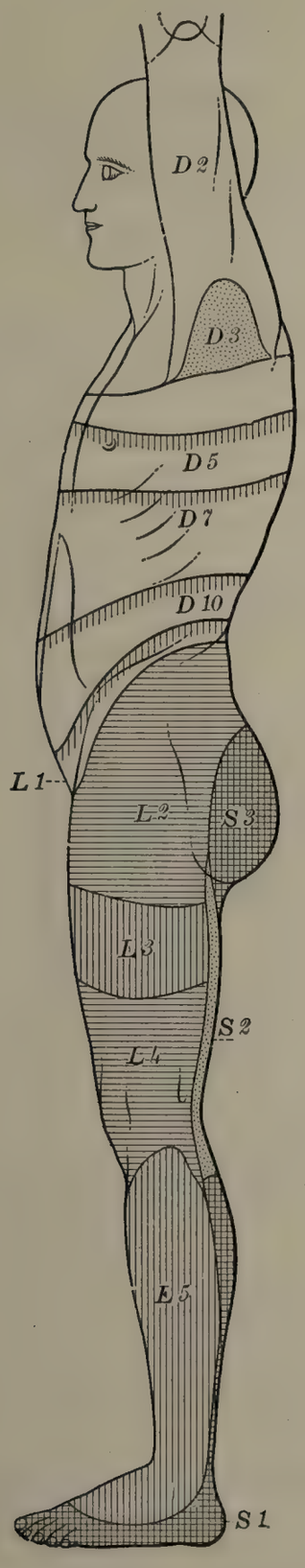


FIG. 39.—Illustrating the segmental spinal sensory areas. Side view.

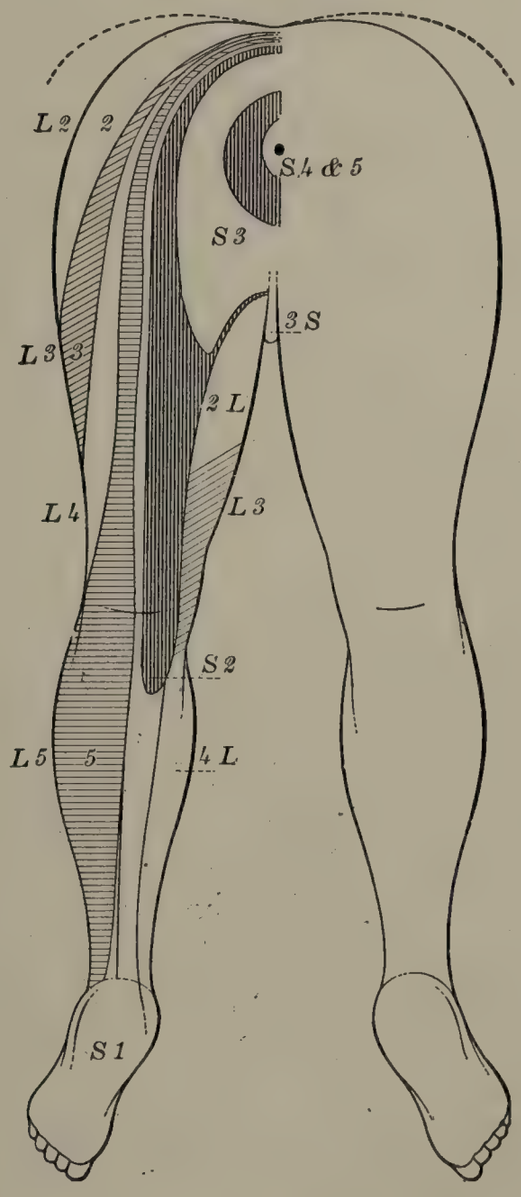


FIG. 40.—Illustrating the segmental spinal sensory areas of the lower extremities.

(baresthesiometer), is used. The pressure should be sufficient to cause pain.

Muscle and joint sense are tested by first showing the patient that one moves the thumb and big toe up or down—and then repeating

movements up or down while the eyes of the patient are closed. Further, weights may be used on the supported and unsupported hand, and the ability to estimate differences observed; or the patient is requested to imitate with one hand a definite position of the other hand with closed eyes.

Bony sensibility is tested by a tuning-fork of low vibrating capacity. This is placed still vibrating, upon bony surfaces, and sensation is intact when the patient feels the thrill. It is a highly important method, particularly in lesions of the peripheral nerves, spinal cord and thalamus.

Sensibility of the *nerve trunks* to direct pressure should then be tested. In the upper arm the brachial plexus branches in the neck and under the arm are palpable and along the inner arm and elbow-joint one may reach the median, radial, and ulnar. Anesthesia of the ulnar (Biernacki) is a frequent finding in tabetic patients.

The radicular and peripheral sensory distributions are shown in Figs. 27, 28, 29, 30, 31 and 32.

In the lower limb the sciatic, anterior crural, cutaneous femoris, tibialis, and superficial peroneus are palpable.

Lasègue's Test.—This consists in flexing the extended leg on the abdomen, keeping it extended by pressure on the knee, when in neuritic processes a sharp pain (popliteal space) is brought out. It is an indispensable test in the presence of suspected alcoholic neuritis.

The distribution of *pain* in neuralgic or neuritic affections should be carefully charted. With the sensory examination, gnostic and praxic tests should be carried out.

Stereognosis signifies the ability to recognize objects by touch. Astereognosis, first described by Puchelt in 1844, is its absence. In a wider sense the tests indicate the perception of spatial and quality relations through the sense of touch. Objects should not only be named but their qualities described—shape, margins, density, etc. A lump of sugar, thimble, match-box, marble, knife, pencil, scissors, etc., are useful test objects.

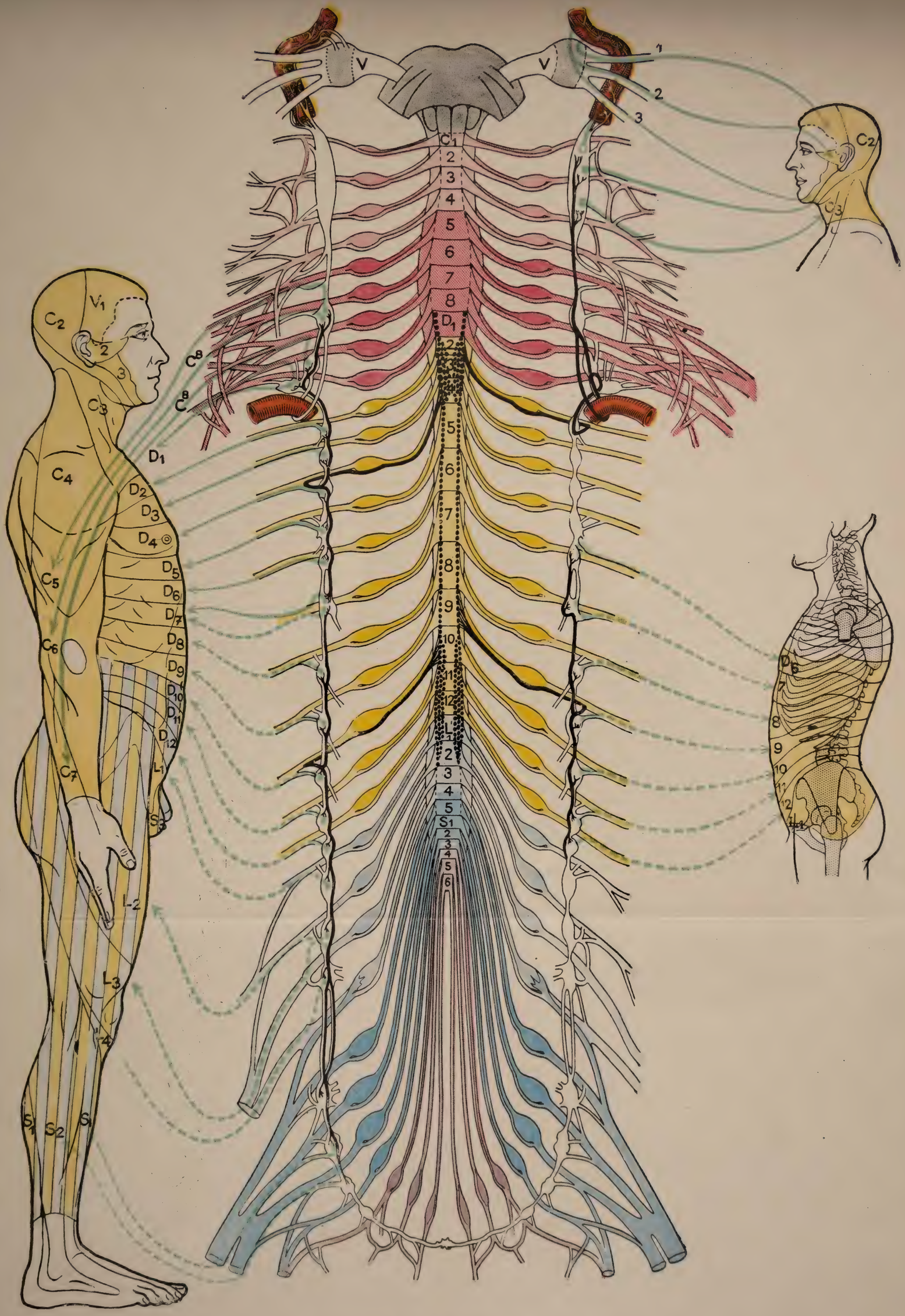
Apraxia consists in[1] the loss of ability to carry out a purposeful movement, not dependent on a palsy. The most useful tests have been referred to. The student is specially referred to Plates X and XI for the interpretation of his findings and their anatomical foundations.

Pilomotor, Vasomotor and Secretory Disturbances. — These should always be tested. André Thomas'[2] work should be consulted by the student. (See Plate II.)

The recent researches of André Thomas upon the pilomotor, sudoral and related skin reflexes, made possible by the many spinal cord, midbrain, and higher cerebrospinal lesions, have opened up a large territory in neuropsychiatry with far reaching applications. Only a brief summary of his more important conclusions can be given here.

[1] Gans: Zeit. f. d. g. N. u. P., 1916, vol. **31**.
[2] André Thomas: Le Reflexe Pilomotor, Paris, 1921.

PLATE II



PILOMOTOR PATHWAYS. (THOMAS.)

Distribution of the Preganglionic fibers of D^2, D^4, D^9, D^{10}, according to anatomical-clinical observations. D^2, Case Mart—Cerebral Reflex. D^4, Case Pin—Cerebral Reflex. D^9, Case Charp—Cerebral Reflex. D^9, Case Chatel—Spinal Reflex. D^{10}, Case Le Mas—Cerebral Reflex. D^{10}, Case Orthl—Spinal Reflex. Preganglionic fibers in black ——. Postganglionic fibers in green.

Goosefleshing is evoked by the application of cold, friction or the electric current. To stimuli on one side the response is homolateral and may extend to the whole of the stimulated half of the body or be restricted to one or more segmental areas. Certain parts of the skin are specially receptive, the nape of the neck, the shoulders and the lower part of the axilla. Stimulation of the neck area must be definite. The goosefleshing descends. On the limbs the extensor are more receptive than the flexor surfaces. If both sides of the body are stimulated the response is symmetrically bilateral.

When the spinal cord is injured the goosefleshing response is exaggerated immediately below the upper level of the lesion. In total division of the cord, as is also shown by the work of Lhermitte, two types of response are seen. One above the level of the segmental region. This is the "Cerebral Pilomotor Reflex" of Thomas; the other is below. This is known as the Spinal Pilomotor Reflex of Thomas. This change in the character of the reflex is an indication of the severity of the lesion. The reflex appears when spinal shock subsides, that is, simultaneously with the mass reflex of Head and Riddoch, and it is elicited by the same stimuli as is the mass reflex, for instance, passive movements of the leg. Hence the governing factors in distribution are the same as those for reflex sweating, that is, the distribution of the thoracico-lumbar white rami. In partial lesions and in various diseases of the spinal cord, the responses vary and are difficult to interpret. In peripheral nerve lesions the reflex response is abolished over the area of sensory loss.

The cerebral reflex varies greatly in definiteness and distribution. It descends to the level of the sympathetic distribution of

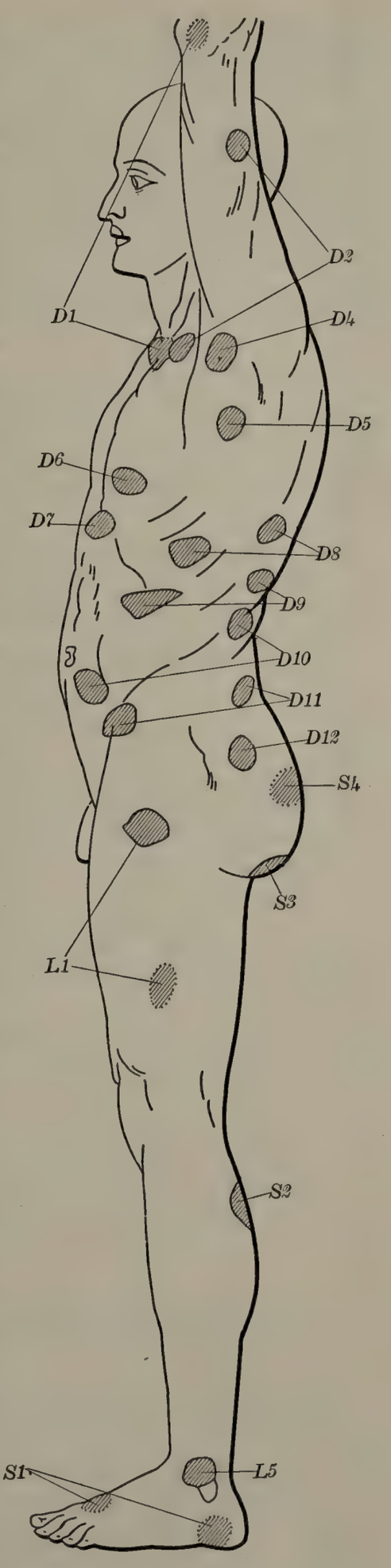


FIG. 41.—Cutaneous reflex zones of hyperalgesia, showing their relations with the spinal root segments and their vegetative nervous-system connections. The dotted areas are to be referred to the internal surfaces. (After Dejerine.)

the isolated cord. In the normal subject the reflex may be obtained by peripheral stimuli and by various emotional factors. In the case of the former means of excitation the sensations aroused are affective and unpleasant. Accordingly from anesthetic skin no response is elicited. The emotions which produce the reflex are usually of unpleasant quality. The most efficient stimuli are fright and horror, but any profound emotion suffices. The reflex stirred up by emotion is bilateral, that by peripheral irritation may be unilateral. The pilomotor reflex, therefore, may be looked upon as containing an affective component.

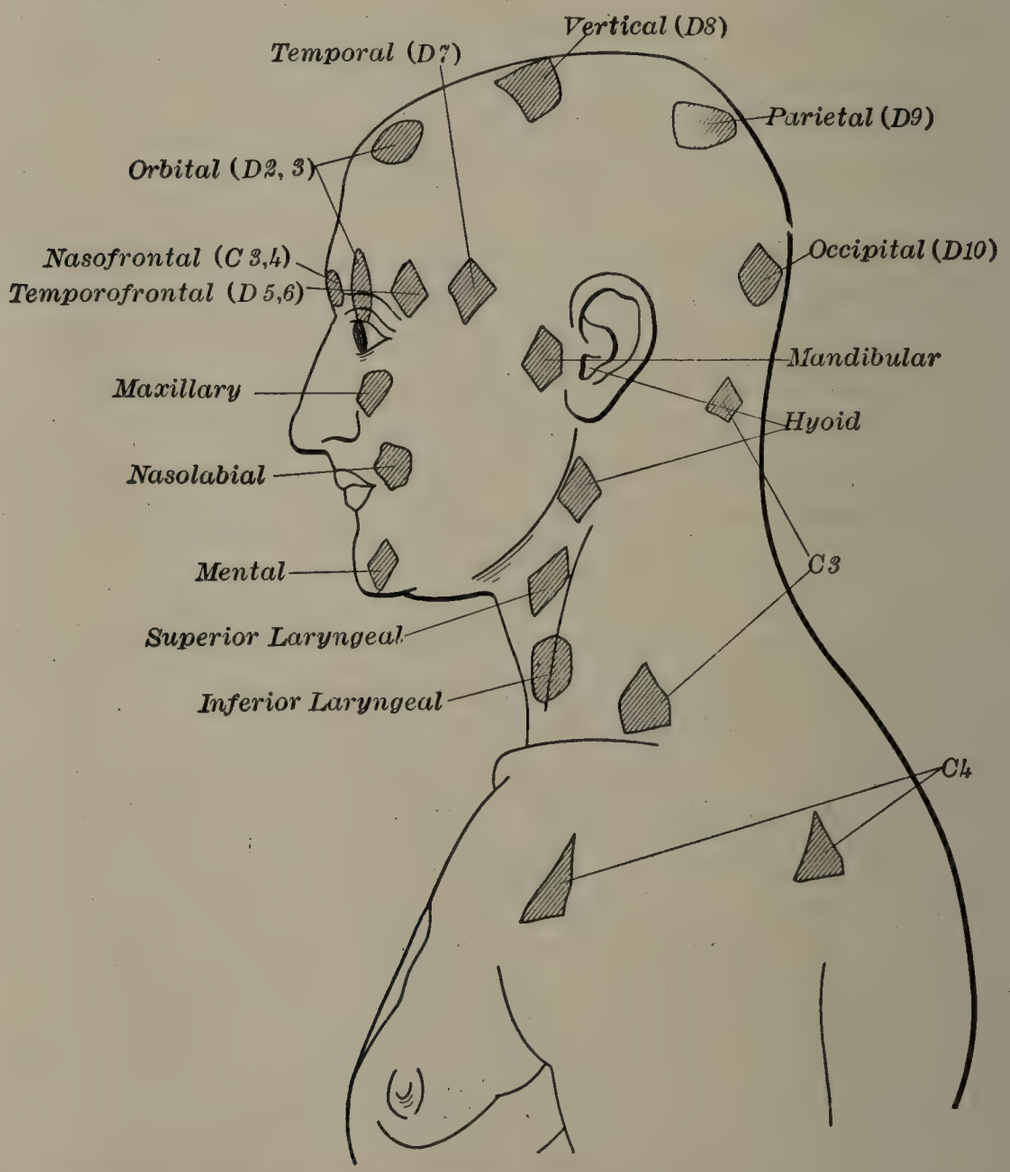


FIG. 42.—Cutaneous reflex zones of hyperalgesia of the head, neck, and shoulders in their relations to vegetative nerve (somatic) disturbances. (After Dejerine.)

With reference to the synaptic associations Thomas states that these are found in the fourth to seventh dorsal segments for the upper limbs and for the lower limbs the ninth to twelfth dorsal segments, the first and probably the second lumbar segments are located in the first, second and third dorsal segments, the synapses for face, neck, and upper thorax pathways, pass through the cervical plexus. Incoming and outgoing pathway disturbances may be located. In hemiplegics without thalamic involvements the cervical reflex persists unmodified.

Lesions of the central gray disturb the reflexes, as seen in syringomyelia, hematomyelia, intramedullary spinal tumor, spinal cord, gunshot wounds, etc.

Cauda equina lesions do not usually disturb the pilomotor reflexes, but if the sympathetics be severed below the synaptic connections the reflex is lost. In herpes the reflexes may be lost, and in sympathetic chain lesions modifications occur in relation to the kind of injury. In divided nerves the reappearance of the spinal pilomotor reflex is indicative of beginning regeneration. For further details see the monograph of Thomas, loc. cit.

Trophic Disturbances.—The presence of dermographia, of blushing, of redness, or blanching of the skin should be looked for. Ulcers, thickness of skin, dryness, or other trophic disorders should be charted. Reflex hyperalgesias (referred pains) should always be inquired for. Patients refer to them chiefly as "sore spots." (See Figs. 41 and 42.)

Scheme for Testing Sensibility.—The following scheme for testing sensibility is advised:

A. *Spontaneous Sensations:* Pain, numbness, tingling, position of the limb, idea of the limb, hallucinations or illusions.

B. *Loss of Sensation:*

1. Touch.
 (*a*) Light touch, cotton-wool on hairless and shaved hair-clad parts; threshold with von Frey's hairs.
 (*b*) Pressure touch, threshold with pressure esthesiometer.
2. Localization: Naming the part touched. Henry's or Head's method, target, etc.
3. Roughness: Threshold with Graham-Brown's esthesiometer. Sand-paper tests, discrimination of relative roughness.
4. Tickling and scraping: Tickling on soles and palms. Cotton-wool rubbed over hair-clad parts. Light scraping with finger-nails.
5. Vibration, tuning-fork: Loss or diminution of sensibility. Alteration in the character of the sensation evoked.
6. Compass points: Points simultaneously applied. Points successively applied.
7. Pain:
 (*a*) Superficial pain: pin-prick; threshold with algesimeter; reaction to measured painful stimuli.
 (*b*) Pressure pain: threshold with the algometer; reaction to painful pressure.
8. Temperature: Thresholds for heat and cold. Effect of adaptation on threshold. Discrimination of different degrees of heat and cold. Affective reactions: (*a*) to extreme degrees, (*b*) to warmth.
9. Position: By imitating with the sound limb the position of the affected limb; by pointing with the sound limb; measurement of defect by Horsley's method.

10. Passive movement: Appreciation of movement. Recognition of the directions of movement. Measurement of the angle of the smallest movement which can be appreciated; falling away of the unsupported limb when the eyes are closed.
11. Active movement: Imitation of movement by the sound limb; ability to touch a known spot; measurement of the defect by Horsley's method.
12. Weight:
 (*a*) With hand supported: Recognition of differences in weights applied successively to one hand. Appreciation of increase or decrease of weight. Comparison of two weights placed one in each hand.
 (*b*) With hand unsupported: Comparison of two weights placed one in each hand. Recognition of differences in weights applied successively to one hand.
13. Size: Difference; threshold. Distinction of the head from the point of a pin.
14. Shape (two dimensional).
15. Form (three dimensional): Recognition of common objects by their form.
16. Textures.
17. Dominoes: Ability to count points by touch.
18. Consistence.
19. Testicular sensibility:
 (*a*) Light pressure.
 (*b*) Painful pressure.
20. Sensibility of glans penis to measured prick.

Status Corporis.—A systematic physical examination is a *sine qua non.* The main facts to be noted in the questionnaire are the condition of the heart, the presence of murmurs, the character of the arteries (hard, tortuous), blood-pressure, the lungs, presence of tumor in abdomen, enlargement of liver, and the condition of the urine, the blood with special reference to leukocytosis as an index for hidden sources of infections, roentgen-ray explorations and the cerebrospinal fluid. (See scheme of cerebrospinal fluid findings, pp. 128–131)

MENTAL-EXAMINATION METHODS.

In no department of medicine is a complete examination of the patient more important than in that of psychiatry. This examination must not only include the symptoms that the patient may present when seen, but must also include the most detailed obtainable anamnesis. It must be borne in mind that a psychosis is a new condition in an individual who was previously well. Above all it is not a something that comes from without, which attacks and seizes on the patient like, for example, a pathogenic microörganism, but is rather to be considered as a type of reaction of the individual to certain inimical conditions. In order, therefore, to understand a particular case it is

of the highest importance to have, as fully as possible, a conception of the individual before he became afflicted, so that the symptoms which are the expressions of this reaction may be understood.

No scheme of mental examination which has been devised is comparable in definiteness or fixity to the procedures in the field of internal medicine. The symbol is a much more elusive object of inquiry than the heart beat, the urine, or the test meal. Mental examination cannot be in accordance with a fixed, unvarying scheme and method of procedure. Constantly during any psychological examination, situation will develop which, if followed will throw any set scheme out of gear. It is desirable, nevertheless, to have some accepted course to follow, no matter how roughly formulated, but any such method is capable of a richness of results of which its outline on the printed page gives not the slightest indication. The reply to the simplest question is not only informing as simple response but much more so when the general attitude and affective signs are also taken into consideration. The spoken word is therefore, not only the symbol of a certain ideational contact but also, in its utterance and the accompanying signs is indicative of the affective content. The word as symbol thus is of importance precisely in proportion to one's ability to see its meanings. A simple question, for example, may be answered promptly or slowly, with a rising or falling inflection, in a high or low pitched voice, clearly or inaudibly, certainly or stutteringly, rightly or wrongly, easily or with evident effort and perhaps showing perplexity, doubt, or embarrassment; the answer may be accompanied by pallor or flushing, by trembling or marked increase of muscular tensions, there may be marked respiratory disturbances and variations of pulse rate, etc. Such a simple scheme as that here outlined, used in this way by a competent and ingenious examiner may easily be the wedge that opens the way to many things.

The scheme of examination which follows is directed primarily to elucidating the mental state. It is taken for granted that the student is familiar with the various methods of physical examination. The omission of specific directions as to the physical examination is not, however, to be taken as an indication that it is considered unimportant. On the contrary, a physical examination in minute detail is of the utmost importance and unless it is made the risk is bound to be run that the key to the whole situation will be overlooked.

The principal value that a scheme of examination may have, however, is in formulating tests that call for an actual record of the patient's reaction and not the conclusions of the examiner. Hospital records are filled with such remarks as "the patient shows lack of judgment" or is "disoriented" or has "failure of memory." All of these are conclusions and are not—records of facts. Such histories are useless to anyone except perhaps the persons who wrote them. The reader of a history is entitled to a statement of the facts on which the conclusions are based and then he is at liberty to form his own conclusions from the identical premises. How much better and more accurate

CEREBROSPINAL FLUID IN DIFFERENTIAL DIAGNOSIS*

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Neurological Laboratory, Massachusetts General Hospital, Boston, Mass.

Disease.	Initial pressure, mm. of spinal fluid, horizontal position.	Rise on jugular compression.	Appearance.	Cells per c.mm.	Globulin.	Protein, mg./100 cc.	Sugar, mg./100 cc.	Chlorides (as NaCl), mg./100 cc.	Non-protein nitrogen, mg./100 cc.	Gold sol.	Comment.
Normal Lumbar Cisternal Ventricular	70–190	Prompt	Clear Colorless No clot	0–5 0–5 0–3	0	15–45 10–25 5–15	50–75 50–75 55–80	720–750	12–18	0000000000	Sugar and chloride values apply to fasting non-febrile individuals.
Blood plasma	...	...		...	...	70–110	570–620	18–30	6200–8200		
Meningismus	+	N	N	N	0	**N or low**	N or +	**N or low**	N	N	**Large amount of fluid with relatively slight drop in pressure.**
				INFECTIONS OF THE CENTRAL NERVOUS SYSTEM.							
Tuberculous meningitis	✢	N	Opalescent to turbid faint yellow ±; **delicate fibrin web**	+; mononuclears	+	✢	**Low**	**Very low**	N	Variable	**Excess fluid; excess polys very early and in infants; chloride usually below 640 mg./100 cc.; progressive fall in sugar which may be high at outset, to under 30 mg./100 cc. or lower; tubercle bacilli found in clot or sediment, guinea-pig inoculation positive.**
Brain abscess	✢	See brain tumor	Clear and colorless to turbid clot ±	✢	±	**Slight increase**	**N or ✢**	**N**	N	Variable	**Polys nearly always present;** if complicated by septicemia or high fever chlorides may fall to low levels (brain abscess represents one form of "aseptic meningitis").
Acute syphilitic meningitis	✢	N	Clear to turbid faintly yellow fibrin clot ±	+	+	+	**N or slightly low**	**Slightly low**	N	**Strong reaction zone variable**	**Wassermann reaction nearly always positive.**
Acute purulent meningitis	✢	N	**Clear to purulent faint yellow ✢ clot ✢**	+	+	+	**Low**	**Low**	N	Variable	Sugar may be but slightly decreased at outset; often falls rapidly to under 10 mg.; chlorides rarely below 640 unless septicemia is present; meningococci and influenza bacilli found in smear and culture with difficulty; pneumococci, streptococci and staphylococci easily; indol present in influenza meningitis.

Acute anterior poliomyelitis	✢	N	Slight opalescence, rarely turbid and faint yellow ±; delicate fibrin web ±	+	Slight +	**Slight ✢**	**N or slight increase**	**N or slight decrease**	N	Variable	**In preparalytic stage polys may exceed 80%; rapid change to mononuclears; with gradual decrease in cells protein increases for two tothree weeks.**
Tuberculous meningitis	✢	N	Opalescent to turbid; faint yellow ±; **delicate fibrin web**	+; mononuclears	+	+	**Low**	**Very low**	N	Variable	**Excess fluid; excess polys very early and in infants; chlorides usually below 640 mg./100 cc.; progressive fall in sugar which may be high at outset, to under 30 mg./100 cc. or lower; tubercle bacilli found in clot or sediment, guinea-pig inoculation positive.**
Encephalitis lethargica	**N**	N	N	**N or slight increase; no polys**	±	**N or slight increase**	**N or slight increase**	**N**	N	Variable	**Sugar normal unless blood sugar is elevated; over 50 % of cases have normal cell count, which rarely exceeds 60 cells; protein increase when present is slight, rarely reaching 100 mg./100 cc.**
				Central Nervous System Syphilis.							
Acute syphilitic meningitis	+	N	Clear to turbid faint yellow ±; fibrin clot	+	+	+	**N or slightly low**	**Slightly low**	N	**Strong reaction zone variable**	**Wassermann reaction nearly always positive**
"Meningovascular syphilis"	±	N; rarely delayed	N; rare fibrin clot	+	+	+	N	N	N	Variable	**Wassermann reaction nearly always positive.**
Progressive parenchymatous syphilis	+	N	N; rare fibrin clot	+	+	+	N	N	N	**Paretic or luetic**	**Wassermann reaction always strongly positive** (includes tabes, paresis and optic atrophy).
Late inactive forms	**N**	N	N	**N**	**N**	±	N	N	N	Weak luetic ±	**Wassermann reaction weakly positive or negative.**
						Brain Tumor.					
Above Tentorium — Not involving wall of ventricle — Lumbar	✢	**N**	N	N	N or slight increase	**N or slight increase**	N or +	N	N	Variable	Tumors involving optic chiasm may give increased cells (mononuclears); lumbar puncture in patients with choked discs may be dangerous unless ventricle is tapped at the same time.
Above Tentorium — Not involving wall of ventricle — Ventricular	✢	**N**	N	N or slight increase	N	**N**	N or +	N	N	N	Ventricular fluid normal.
Above Tentorium — Involving wall of ventricle — Lumbar	✢	**N**	N; occasionally faintly yellow	N or slight increase	+	✢	N or +	N	N	Variable	Protein increase here comes from ventricle.
Above Tentorium — Involving wall of ventricle — Ventricular	✢	**N**	N; occasionally faintly yellow	N or slight increase	+	✢	N or +	N	N	Variable	Protein increase occurs in lateral ventricle invaded by tumor; other ventricle may have normal fluid.

* Presented before the American Neurological Association, June, 1926. By permission of Dr. James B. Ayer.

CEREBROSPINAL FLUID IN DIFFERENTIAL DIAGNOSIS.*—(*Continued.*)

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Neurological Laboratory, Massachusetts General Hospital, Boston, Mass.

	Disease.	Initial pressure, mm. of spinal fluid, horizontal position.	Rise on jugular compression.	Appearance.	Cells per c.mm.	Globulin.	Protein, mg./100 cc.	Sugar, mg./100 cc.	Chlorides (as NaCl), mg./100 cc.	Non-protein nitrogen, mg./100 cc.	Gold sol.	Comment.
						BRAIN TUMOR.—(*Continued.*)						
Below Tentorium — Intracerebellar and fourth ventricle	Lumbar	✢	N or delayed	N	N	Slight increase or N	Slight increase or N	N or +	N	N	Variable	Fourth ventricle tumors usually do not cause any protein increase; **the protein increase below intracerebellar gliomas is slight, usually under 100 mg./100 cc.**
	Ventricular	✢	N	N	N	N	N	N or +	N	N	N	Ventricular fluid normal; **subtentorial block** best demonstrated by combined ventricular and lumbar puncture.
Below Tentorium — Extracerebellar	Lumbar	✢	N or delayed	N or faintly yellow rare clot	N	+	✢	N or +	N	N	Variable	**Protein increase in these cases well marked; usually 150 mg./100 cc. or higher.**
	Ventricular	✢	N	N	N	N	N	N or +	N	N	N	Ventricular fluid normal.
				SPINAL SUBARACHNOID BLOCK (TUMOR, POTT'S DISEASE, MENINGITIS, ETC.)								
Partial block	Lumbar	N or low	Delayed	N or faint yellow clot rare	±	✢	✢	N	N	N	Variable	Partial block best demonstrated by combined cistern and lumbar puncture.
	Cisternal	N	Prompt	N	±	N	N	N	N	N	Variable	Lipiodol arrest, but some passes by the block.
Complete block	Lumbar	Low or N	Absent	Colorless, with sl. clot, to deep yellow, with massive coagulation	±	✢✢	✢✢✢	N	Slightly low	N	Variable	Little fluid obtainable; rapid drop in pressure to zero.
	Cisternal	N	Prompt	N; faint yellow, or clot rare	±	±	±	N	N	N	Variable	Cistern puncture not necessary to demonstrate block; lipiodol completely arrested.
Tumor of cauda equina	Lumbar (below tumor)	Low	Absent or delayed	Colorless, with sl. clot, to deep yellow with massive coagulation	N	✢✢	✢✢✢	N	...		Variable	
	Lumbar (ab. tumor)	N	Prompt	Yellow or colorless; clot ±	N	✢	✢✢	N	...	...	Variable	
	Cisternal	N	Prompt	N; yellow and [illegible]	N	[illegible]	[illegible]	N	[illegible]	[illegible]	[illegible]	

equina; also numerous cases of acute polyneuritis					to ++	to +++		low		Variable	to distinguish from tumor, unless puncture is below lesion and block can be excluded; lipiodol valuable.
					MISCELLANEOUS.						
"Bloody tap" (normal fluid)	**N**	**N**	**Blood-tinged or bloody supernatant fluid colorless or pink clot**	**No excess of WBC**	±	**Slight increase**	N	N	N	Variable	**Progressively less bloody in consecutive tubes; supernatant fluid never yellow (at original puncture) even after standing; if much blood present clot will form.**
Early subarachnoid or ventricular hemorrhage (within 24 hrs.)	**+**	**N**	**Bloody + supernatant fluid faintly yellow, no clot**	**Sl. excess of WBC; usually polys; many crenated RBC**	Slight increase	**Slight increase**	N	N	N	Variable	**All tubes equally bloody;** yellow tint to supernatant or centrifuged fluid appears with 2 to 4 hours after hemorrhage occurs and deepens.
Late subarachnoid or ventricular hemorrhage (up to 2 wks.)	±	**N**	**Bloody ± supernatant yellow; no clot**	**Excess of mononuclears; RBC crenated**	Slight increase	**Slight increase**	N	N	N	Variable	Yellow color will increase for 8 to 12 days, when all RBC usually have disappeared; compare with Froin syndrome.
Original "Froin syndrome"	**Low or N**	**Absent**	**Deep yellow, clear massive coagulation**	+	**++**	**+++**	N	Slightly low	N	Variable	See **"complete spinal subarachnoid block"**—above; distinguish from late subarachnoid hemorrhage by high protein content.
Multiple sclerosis — **Clinically active**	**N or low**	**N**	N	**Slight increase (rarely over 30)**	Slight increase	**Slight increase**	N	N	N	**Strong reaction, paretic or luetic**	A strong paretic or luetic gold-sol reaction in presence of negative Wassermann in patient not previously treated for syphilis is strong evidence for multiple sclerosis.
Multiple sclerosis — **Clinically inactive**	**N or low**	**N**	N	**N**	N	**N**	N	N	N	N; weak paretic or luetic	
Lateral sinus thrombosis	**+**	**Delayed or absent to jugular comp. on side affected side**	N to turbid; slight yellow or clot rare	Usually slightly increased	N	**N or low if uncomplicated**	Increased or N	**Low or N**	N	Variable	Considerable normal variation on the two sides to unilateral jugular compression in many normal individuals; a prompt response on each side excludes complete lateral sinus thrombosis.
Uremia	+	N	N	N	N	**N or low if uncomplicated**	+	May be increased or decreased	**Increased**	**Variable; may give strong paretic curve**	

* Presented before the American Neurological Association, June, 1926. By permission of Dr. James B. Ayer.

MENTAL-STATUS QUESTIONNAIRE.[1]

General Demeanor:

Dress: Hair:

Hands:

Reaction: Free: Reserved:

Respectful: Hostile:

Friendly: Puerile:

Evidences of struggle: Bruises:

Accessibility:

Natural: Free:

Alert: Happy:

Elated: Exuberant:

Dull: Apathetic:

Somnolent: Depressed:

Gloomy: Worried:

Fearful: Suspicious:

Perplexed: Disdain:

Scornful: Sarcastic:

Motor Activity:

Average: Excited:

Diminished: Slow:

Resistive: Frigid:

Queer: Bizarre:

Speech Activity:

Average: Talkative:

Noisy: Quiet:

Slow: Mute:

Disordered: Divertable:

[1] See interesting study by Courbon: Séméiologie de la mise des Aliénés. L'Encéphale, 1919, ix–xiv, p. 359.

MENTAL-STATUS QUESTIONNAIRE—*Continued.*

Stream of Thought:

Clear: Logical: Relevant:

Jumping: Coherent: Incoherent:

Retarded: Irrelevant:

Disconnected: Queer:

Fragmentary: Jumbled:

Examples: Stenograms should be recorded:

Content of Thought:

Worry: Insight:

Apprehensive: Anxiety:

Delusional: Phobias:

Hallucinatory: Character:

Peculiar Experience:

Comparison, former states:

Attitude of Friends:

Suicidal Ideas:

Dreams:

General Orientation:

Full name:

Age: When Born:

Present Year Month Day

City: Place:

When here: How Long:

What here:

People about:

Are you sick:

Why here:

Description:

MENTAL-STATUS QUESTIONNAIRE—*Continued.*

Subjective Idea of Memory:

Attention:

Thinking:

Capacity:

Memory—Combination:

[7 × 8]	[9 × 8]
972864]	45319628]
516724]	97125684]
$x + 19 = 36$]	$x - 27 = 15$]
$x \times 7 = 84$]	$x \div 8 = 7$]

Change:

2 eggs cost 10—how much 3]

School Knowledge:

History:

Geography:

Countries:

Rivers:

Capitals:

Natural Objects: Name five of each:

Birds:

Fish: Postage:

Animals: Local:

Flowers:

Masselon: Hunter—Forest—Gun—Dog—Rabbit]

Description:

MENTAL-STATUS QUESTIONNAIRE—*Continued.*

Finckh—People who live]

Too many cooks]

Ebbinghaus:

Differences:

Ox / Horse	Box / Basket
Ice / Water	Bird / Butterfly
Steps / Ladders	Dwarf / Child
Lie / Mistake	Shrub / Tree

Continuous Sentence:

If it rains]

Because]

Nevertheless]

If the soup be burned]

Because]

Nevertheless]

Backward Associations:

Name] Week Days]

Months] 75826]

Story:

Reproduction of Picture:

Name—Writing: Eyes open and closed:

Description:

than the statement "defective memory" would be this test: The patient in the course of the examination is given the address 375 Oxford St. After five minutes he is asked to recall it. He gives the number 176, but cannot give the name of the street. Here is a definite fact. A multiplicity of such facts gives one a basis for conclusions about the patient. The record of an examination should be composed of data of exact nature.[1]

Mental Examination.—*Orientation:* time; place; persons.

General memory: family; school; occupation; marriage; children; diseases.

Emotional status: insight; sleep; dreams.

Hallucinations: auditory; visual; tactile; visceral, etc.

Speech: voluntary; writing (name, date, the United States of America, the Commonwealth of Massachusetts); auditory; visual; test-phrases. (Statistical, perturbation, Third Riding Artillery Brigade.)

Stories ("Cowboy," "Gilded Boy," "Polar Bear," "Shark," "Good Girl").

Special memory: Civil War; names of two generals; three European countries; capital of native State; President; repeat numbers, such as 45319628; 35984271; 487631; 955217; 7368; 487; 352; 375 Oxford Street (after three to five minutes).

Masselon (hunter, dog, gun, forest, rabbit); (man, wood, coal, stove, dinner); (needle, thread, button, vest); (pipe, match, smoke); (pen, ink, letter). Make simple sentences of these.

Ziehen.—What is the difference between (horse and ox; dwarf and child; lie and mistake; water and ice)?)

Combinations (7 x 6; 56 — 18; 23 — 14; 81 — 9; X — 5 = 17; X — 8 = 13; have 50c; buy cherries 12c, butter 7c, bread 10c; how much change?

Forward and Backward associations (month; days of week; 752186, 25729, 6418, 265, 497).

General information: cost of postage; color of stamps; holidays and meaning (Christmas, Easter, Fourth of July).

Finckh ("The early bird catches the worm;" "Lies have short legs;" "Set a thief to catch a thief;" "Burn a candle at both ends"). What is the significance of these proverbs?

Ethical questions:

Drawing diagram (after five seconds' exposure).

NOTE.—Here especial caution is needed to avoid recording conclusions. For example: Under orientation the patient's actual answers to such questions as, When were you born? How old are you? What day is this? etc., should be put down.

The stories which are named are as follows:

"Cowboy Story."—A cowboy from Arizona went to San Francisco with his dog, which he left at a dealer's while he purchased a new suit of clothes. Dressed finely, he went to the dog, whistled to him,

[1] See White: Outlines of Psychiatry, Eleventh edition, 1926.

called him by name and patted him. But the dog would have nothing to do with him in his new hat and coat but gave a mournful howl. Coaxing was of no effect, so the cowboy went away and donned his old garments, whereupon the dog immediately showed his wild joy on seeing his master as he thought he ought to be.

"Gilded Boy Story."—It is related that at the coronation of one of the popes, about three hundred years ago, a little boy was chosen to act the part of an angel; and in order that his appearance might be as gorgeous as possible he was covered from head to foot with a coating of gold foil. He was soon taken sick, and although every known means was employed for his recovery, except the removal of his fatal golden covering, he died within a few hours.

"Polar Bear Story."—A female polar bear with two cubs was pursued by sailors over an ice field. She urged her cubs forward by running before them, and, as it were, begging them to come on. At last in dread of their capture she pushed, then carried and pitched each before her, until they actually escaped. The polar bear is a wonderful swimmer and diver. In the capture of seals lying on the ice, it dives some distance off and swimming underneath the water, suddenly comes up close to the seals, shutting off their retreat to the sea.

"Shark Story."—The son of a governor of Indiana was first officer on an oriental steamer. When in the Indian Ocean the boat was overtaken by a typhoon and was violently tossed about. The officer was suddenly thrown overboard. A life-preserver was thrown to him, but, on account of the heavy sea, difficulty was encountered in launching the boat. The crew, however, rushed to the side of the vessel to keep him in sight, but before their shuddering eyes the unlucky young man was grasped by one of the sharks encircling the steamer and was drawn under the water, leaving only a dark streak of blood. (Adapted from Ziehen.)

"Good Girl Story."—Once upon a time there was a girl whose father and mother were dead, and who was so poor that finally she had nothing but the clothes on her back and a little piece of bread in her hand. She was deserted by everybody, but since she was good and honest she went into the world with confidence in God. As she went along she was met by a poor old man who said, "Give me something to eat, I am hungry." The girl gave him the piece of bread and went on farther. Soon afterward she encountered a little girl freezing and almost naked, who begged for her clothes. The good girl gave the poor child the warmest of her garments. Night came on, the good girl was tired, cold and hungry. She traveled into the woods, and, wandering off the road, she knelt and prayed to God. As she knelt she saw the stars falling all about her, and when she looked she found they were many bright gold dollars. (Adapted from Ziehen.)

These stories which are used have been selected with great care. They are especially valuable. It is remarkable the amount of information that one can obtain from getting a patient to repeat one or two. Defects of memory and attention show immediately, while the

manic tendency to elaborate is characteristic. They should never be omitted. The cowboy story is usually the easiest, while the good girl story is hard, because of the great amount of detail. The emotional feature of the "streak of blood" in the shark story is particularly impressive and may be about the only feature of the story reproduced.

In the special memory test, of course, different people will have to be treated differently. A Polish immigrant just landed would hardly know about the Civil War. The important thing, however, is to record actual question and answer.

In the Masselon tests the patient is asked to incorporate such words as pen, ink, letter, into a sentence.

In the Ziehen test the patient is asked to tell the difference between horse and ox, dwarf and child, etc.

The problem of calculating the change left from 50c after making certain purchases is an excellent example of the usefulness of standard questions. Everyone on the hospital staff knows that the answer is 21c; and although this is a little thing, when multiplied many times it makes a great deal of difference in the ease with which one can go over a history or appreciate it when read.

The forward and backward associations are valuable as roughly quantitative. The average person should be able to give six numbers forward and five numbers backward. This test will disclose just how many the patient can give and is one of the valuable tests for repeating from time to time during the course of the psychosis. It is also very valuable in detecting the malingerer. A definite intention to blunder is usually readily distinguishable from a natural blunder.

In the Finckh test the patient is asked the meaning of sayings or proverbs such as, "The early bird catches the worm."

Such ethical questions can be asked as, What would you do if you saw a man drop a $10 bill?

In addition to the tests given in the table frequent use is made of the Ebbinghaus test which consists of having the patient complete a sentence in which certain words have been left out, such as: I got up in the . . . and after washing my . . . went to . . . Or better often is Ziehen's modification of this test. The patient is asked to complete such a sentence as this: If it rains . . . because in spite of . . . The Bourdon test is very valuable as a measure of attention. It consists of getting the patient to strike out certain recurring letters or numbers in a standard page and timing the result. A similar test is the tapping test—timing the number of taps that can be made in a given time, say thirty seconds.

The cases will be numerous in which it will be found desirable or necessary to pursue the examination further in some direction. No scheme can cover all possibilities and would be useless if it did, because impossible to carry out. Much must of necessity be left to the judgment of the examiner. By following this plan, however, it is believed that the general and important features necessary for a case record will be covered in the large majority of cases.

It is useful, after completing the examination, to accent the significant features in a short summary, which might include a provisional diagnosis if the facts warranted.

The tests here described are for the most part *intelligence* tests. Even such questions as might be propounded under the head of "ethical questions" may very easily have only the value of intelligence tests because the patient will quite likely answer in accordance with the conventional ideas with which he is perfectly familiar rather than answer in accordance with the way in which he feels. While the intelligence tests are important and while by taking them in a routine manner one can get a good deal of information from the patient, often information of matters that lie deeper than mere questions of intelligence, still they are by no means all-sufficient. The intellect is after all only superficial as a guide to conduct. The deeper motives that move men to action come from the realm of feeling, and if the symptoms are to be explained or understood the emotional springs of conduct must be fathomed.

The same criticism may be made of the Binet-Simon and other tests devised to determine the degree of mental defect expressed in terms of the psychological (I. Q.) age. These tests are valuable in skilled hands for the more pronounced degrees of defect, but as development proceeds they become progressively more unreliable in proportion to the increased psychic mass, the impossibility of standardization because of the wide individual differences, and the greater probability of distortion from unknown emotional sources.

More recently a definite attempt has been made by Hoch and Amsden[1] to formulate a scheme of examination, directed more especially to the affective aspect of the psyche. To use such a scheme as this, however, and in general to gain any real insight beneath the surface of the psychic life, the technic of psychoanalysis needs to be used.

PSYCHOANALYSIS.

Psychoanalysis is a method by which the human mind is dissected so to speak, and by means of which unconscious motives of conduct are sought. If anything like a complete understanding of patients is to be had the methods of psychoanalysis must be used. This is hardly the place to discuss these methods at length. It would require more space than a text-book of this character could properly give it. The student is referred to special works. Here only the briefest suggestions will be given.[2] (See also in this connection the Introduction to Part III and the Treatment of the Neuroses.)

[1] Guide to the Descriptive Study of the Personality with Special Reference to the Taking of Anamneses of Cases of Psychoses, Rev. of Neurol. and Psych., 1913, 11, 577.

[2] Freud: A General Introduction to Psychoanalysis, New York, 1920. Freud: The Ego and the Id, Freud: Inhibition, Anxiety and Symptoms, New York, 1927. Levin: The Unconscious, Macmillan, N. Y. Jelliffe: Technic of Psychoanalysis, Nervous and Mental Disease Monograph Series, No. 26, for important bibliography. White: Mechanisms of Character Formation, Macmillan & Co. Rank and Forenczi: Development of Psychoanalysis, Nervous and Mental Disease Monograph Series, No. 40. Freuds' Collected Works, 12 cols. Four vols, in English, Hogarth Press, London. Ferenczi: Technique of Psychoanalysis, New York, 1927.

The Complex.—Mental systems should not be conceived as made of or containing ideas which are deposited here and there, helter-skelter, without order, as the scraps of paper that are thrown carelessly into a waste basket. Quite the contrary. Ideas are grouped about central experiences, constellated one may say, built into coherent and harmonious structures not unlike the way in which bricks and stones are brought together to form buildings and these buildings are again grouped to form the larger whole—the city. The significant fact in this connection is that the cement that holds the bricks and stones together, the binding substance, is *feeling*.

This orderly arrangement of ideas upon a background of feeling which serves to unite them is what gives character, individuality to the personality. The creating of the proper feeling-tone about things and events is one of the main functions of education.

Now it so happens that in certain individuals a constellation of ideas, grouped about a central event that conditions a highly painful emotional state, is crowded out of clear consciousness—*repressed*—into the unconscious and so tends to lead an existence which is relatively independent and in so doing gives origin to various symptoms. Such a constellation is termed a "complex" in psychoanalysis.

The complex, crowded out of relation with the personal consciousness, seeks for expression notwithstanding; and because it is not synthetized with the conscious ego system its expression cannot be controlled and guided into the usual channels. Thus are created inhibitions, anxiety and symptoms.

The extreme difficulty in locating and uncovering the complex is due to the symbolic forms in which it usually manifests itself. The painful memories of disagreeable experiences, unethical, unconventional, and otherwise impossible and hateful wishes while crowded out of mind by what Freud has so aptly termed the "censorship of consciousness" (super ego systems) struggles to find outlet. The complex cries for recognition, the censor will have none of it—the fight is on, the conflict wages, until finally a sort of compromise is reached by permitting the complex to come into clear consciousness but only on pain of not disclosing its true self, that is, it is permitted to appear under the cloak of a complete disguise.

For example, Freud's case of Elizabeth. She was engaged in nursing her sick father who afterward died. One evening, spent away from home at the solicitation of the family, she met a young man of whom she was very fond and he accompanied her back home. On the walk home she quite gave herself up to the happiness of the occasion and walked along oblivious of her duties. On reaching home she found her father much worse and bitterly reproached herself for forgetting him in her own pleasure. She immediately repressed this disagreeable thought from her consciousness. Now she had, each morning, to change the dressings on her father swollen leg. To do this she took his leg upon her right thigh. The repressed complex seized upon the feeling of weight and pain of her father's leg upon her

thigh as a handy and efficient means of expression and so the repressed wish comes into consciousness under the disguise of a painful area of the right thigh corresponding in extent and location to the place upon which she rested her father's leg.

This is the sort of mechanism that accounts for many unusual and strange experiences that otherwise appear to be without reason. Unexplained forgetting, slips of the tongue, certain mental attitudes, moods, and even the dominant traits of character are due to the activity of submerged complexes while the phenomena of dreams are explained in the same way.[1]

The unconscious methods are very logical. The complex often expresses itself symbolically (*symbolism*), often by the transfer of an emotion from a painful event to a less painful or indifferent event (*displacement*), often, as in hysteria, by the conversion of the conflict into a physical symptom (*conversion*). In the phobias, obsessions and compulsive type of disturbance, the repressed affect undergoes a *substitution*. In this way the symbol carrier of the repressed wishes, the symptom, is a most ingenious disguise.

Dreams.—The analysis of dreams is for the purpose of determining the presence and nature of complexes which are exercising a controlling effect upon the patient's conduct and feelings. The dream appears as a quite senseless experience to the patient and upon the face of it it would appear also to be senseless. A very little effort, however, will show that there is a certain rough meaning to the dream. For example, the scenes or the dream will be representations, usually more or less fragmentary, of things which have happened in the life of the individual during the previous twenty-four hours and may easily be associated in his mind with events of some moment to him. As soon as this is pointed out the patient will acknowledge it, if he does not know it first himself, and will think that the dream has been explained. This however, is only the *manifest content* of the dream. Behind this manifest content there lies a deeper, a more profound, a more important meaning or series of meanings. The superficial experiences only serve to hide the *latent content*, which contains the real material of importance in the dream.

The latent content will at most invariably show that the dream in its deeper meanings is dealing with repressed, unconscious material, with material that the patient is not willing to acknowledge to himself. One object of the dream is to conserve sleep, and to that end it does not permit the real dream thoughts to become known, and so builds up a structure that upon its surface appears utterly without meaning and nonsensical, and therefore is hardly deserving of attention. Therefore the dreamer usually passes the dream by without any particular notice, and in the course of the day it is generally forgotten.

The mechanisms by which this disguise is effected have already been spoken of in discussing the complex. The mechanisms are those of

[1] Freud: Psychopathology of Every Day Life.

distortion. These mechanisms of distortion are first the mechanism of *displacement,* which effectuates the transfer of the principal affect from the situation where it belongs to some other situation. The feeling of anxiety, or fear, or disgust, if it were retained attached to that portion of the dream to which it is normally attached, would disclose to the patient the real thing about which he was anxious, fearful, or disgusted, but if displaced upon some indifferent part of the dream serves the purpose of distortion. *Overdetermination* is a mechanism which secures the appearance in the dream of a particular figure, for example because of its likeness not to any one experience in the patient's life, but to many. All of these experiences, as it were, focus in one direction and produce the result; for example the face of a dreamed-of person may appear to be entirely unknown to the dreamer, but when it is, so to speak, dissected it is discovered that each portion of the face, the nose, the lips, the eyes, the hair, each of them, have well-defined associations in the mind of the dreamer and that they have all condensed into this composite to serve the purpose of the dream formation. *Symbolism* is another mechanism which aids distortion so that instead of things being dreamed of as they are in reality, they are dreamed of as being represented symbolically, and so the dream becomes increasingly difficult to read, and to the dreamer quite unintelligible. Further, the mechanism of *secondary elaboration* is the mechanism by which the dreamer, after he is awake, goes on, as it were, with the dream material and explains to himself by a process of rationalization what certain features of the dream mean, and still further distorts it and places it beyond the pale of his power to recognize it.

Here in the dream is seen one of the most exquisite of defense mechanisms of the human being at work. One is accustomed to think of the human animal biologically as defending itself from all sorts of attacks upon its integrity. The various infections by microörganisms are met by vital processes of defense which are frequently fully successful, and the whole theory of immunity is the theory of a defense mechanism of the human animal. The dream is just such a mechanism, and if what is going on in the mind of the patient would be known, what the patient is defending himself from, what are the disintegrating factors at work at the psychological level, the easiest access to the knowledge of these factors can be found if the meanings of the dream can be learned. Dream analysis is a most important tool for the unravelling and treatment of all of the neuroses and psychoneuroses and for the understanding of the psychoses.

Technic.—The technic of psychoanalysis is an art more successful in some hands than in others. However, the general methods of procedure may be briefly outlined.[1]

In the first instance the physician must be fully imbued with a

[1] Jelliffe: Technic of Psychoanalysis (Important Bibliography), Nervous and Mental Disease Monograph Series No. 26, New York, 2d Edit. Brill: Psychoanalysis, New York, 3d Edit., 1920. Psychoanalytic Review, New York and Washington. International Jour. of Psychoanalysis, London.

profound belief that mental symptoms, each and every one of them, have a meaning and a meaning which can be brought to light and will show them to be logical and understandable in each instance. He must then have patience to listen to the story of his patient, and not only listen to it, but listen to it attentively for the purpose of trying to find the meaning in it, for the purpose of trying to find out where the vital points are which can be attacked to best advantage.

It is true that the dream analysis is the main avenue to the understanding of the unconscious motives of action, but all sorts of hints may come from other sources. For example one of the authors was recently listening to the story of a patient. In the course of that story the patient misspoke and said quinine when he intended to say calomel. Realizing that a "slip of the tongue" of this sort must have its meaning and is not an accidental occurrence, because nothing mental is accidental; the slip of the tongue was analyzed and led directly back to one of the most important emotional events in the life of the patient, an event which threw a flood of light upon his psychoneurosis.

Nothing is too trivial to be worthy of analysis, nothing but may throw light upon the situation. All the little slips of the tongue, forgotten incidents, points at which two recitals of an occurrence do not agree, even witticisms, are necessary to trace out besides the analysis of the dream life, and offer an abundance of material in the course of the analysis.

The method of procedure is the method of free association. Whether it be the analysis of some component of a dream or of a slip of the tongue, or what not, the method of free association is the one employed. The patient should be alone with the physician. It is practically impossible to conduct an analysis, at least beyond the surface, in any other way. Under circumstances of quiet and freedom from interruption, as far as possible, the different points which are to be analyzed are taken up. The patient is instructed to take a certain element of the dream which he has just recounted, for example, and hold it in his mind, and then tell freely all of the ideas that come to him. He is told to tell all of the ideas without any effort on his part of selection, no matter whether the ideas appear to him to have any relationship with the portion of the dream that he has been told to keep in mind or not, and no matter whether they appear ridiculous or have other qualities that incline him to lay them aside. He must tell them all just as a man might sit at the window of a railroad train and jot down, as far as possible, everything that he sees pass the window as the train speeds on. Preferably the patient should be lying down, unable to look at the physician.

The theory of this procedure is that if the patient does not direct the thought in any way every idea that comes must of necessity have some relation to the event held before the mind about which enlightenment is sought. This is the method of unravelling the tangled network of the mental life and while it may be supplemented by word association or other means, still it would seem as experience increases,

that no other method is needed, that this answers all the purposes. It takes a long time, as a rule, however, to effect an analysis—weeks, months, perhaps longer. It must be remembered that what has taken years to form cannot be unravelled in an hour.[1]

It will probably occur to many to wonder how it is that one can expect to find memories reaching back for years sufficiently well preserved to be helpful. As a matter of fact the memories of all repressed experiences are perfectly clear no matter how old. The explanation for this is that being repressed they are dissociated from the every-day events of life, they are kept in their original form, they have not been subjected to the attrition and amalgamation with the intricacies of associational life. They do not fade out by this process of absorption as do the memories of indifferent events, but remain where ever after they may be brought to light by analysis and used as helps for cure.

It will be seen from this short description what a far-reaching method this is. A method of analysis from which no event of life, no matter how apparently trivial, is free. A method that in its results lays bare not only the immediate antecedents and causes of the symptoms, but the whole innermost life of the patient, reaching back even to the period of early childhood. This of course takes time. A case of any complexity and difficulty quite generally takes several months, of at least three séances each week, to reach a final result.

The object of psychoanalysis is not merely a dissection of the psyche and the discovery of the roots of the psychosis or neurosis, as the case may be, but is distinctly therapeutic. The physician tries to show the patient to himself as he really is. The patient is thus enabled to see how his symptoms are the results of hanging on to infantile ways of pleasure-seeking, self-indulgences, which are repulsive to his better self. When he has seen this the path is pointed along which he must go toward the effective sublimation, socialization, of his infantile tendencies in activities that are useful and which meet with conscious approval. The object of psychoanalysis then is to liberate the psychic energy which is bound up in infantile ways of pleasure-seeking and set it free for socially useful ends.

[1] Freud: Interpretation of Dreams. Stekel: Die Sprache des Traumes.

PART I.

THE PHYSICO-CHEMICAL SYSTEMS.

THE NEUROLOGY OF METABOLISM.

CHAPTER II.

VEGETATIVE OR VISCERAL NEUROLOGY.

THE AUTONOMIC AND SYMPATHETIC NERVOUS SYSTEMS—THE ENDOCRINE SYSTEM.

Neither structural neurology (anatomical aspect) nor functional neurology (physiological aspect) have as yet adequately dealt with the primitive network which in lower animals from Paramœcium onward came into being as a tangible medium through which coördination of function of separate organ units might take place.

Life, in its gradual advance toward greater control of the cosmic forces which surrounded it and of the accumulated urges within has constructed newer and more advanced forms with definite structures which serve as heritages of its past efforts and as forecasts of its future possibilities.

Here is no place to enter into a discussion of the achievements and difficulties by which the gradual synthesis of that master spirit in evolution, the nervous system, has advanced from Paramœcium to Homo. Such a series, however, can be assumed for the purposes of this work. It is outlined that a certain progression can be roughly demarcated into two large divisions, which intimately related, can in a certain sense be separated. We purpose dealing with these as the splanchnic, or vegetative nervous system, and the somatic, or sensori-motor, or projicient systems. This division offers certain very definite pragmatic advantages, which in the present state of our primitive knowledge, seem to reflect better models of analysis of biological behavior than any other.

It is the endeavor to formulate such phenomena in as simple a form as possible. Hence the conception that the human organism, like all other living organisms, is an energy system with an immeasurable experimental heritage, which captures, transforms, and delivers the energy of the cosmos which surrounds it. The nervous system is

engaged in this work. It is the great organizer of life's experiences. It binds every cell of each living organism into a correlated whole, and makes possible the behavior of the organism as a whole. It has already been outlined that the chief function of life is the continuation of life. Life everlasting is its goal, and the entire organization of the organism is bent on the carrying out of this program. This is largely what Aristotle meant by Entelechy; what biologists call "patterns." "Constitution" thus meaning hereditary patterns.

The simplest formula which a limited intelligence has devised is that such a program may be roughly sketched in the function of preservation of the individual, the species, and the still more insistent goal (entelechy) of the propagation of the race.

The poet Schiller has spoken of these as the instincts of Hunger and of Love. In a rough analogy, they are here discussed as the *mechanisms of metabolism*, and the *mechanisms of socialized creation*. That which is here dealt with as the vegetative or visceral nervous system is primarily, though not exclusively, engaged with the former; while the somatic, or sensorimotor system deals chiefly, though in the same divided manner, with the latter.

It is here assumed, not without the recognition of definite debatable problems, that the visceral or vegetative nervous system is a gradual evolution of certain aspects of the primitive network of lower animals, while the sensorimotor system gradually grew out of later arriving aspects of this same primitive network.[1]

Turning now to a more didactic form of statement it may be said that two characteristic types of nervous system structures are found in all higher animals, namely, a vegetative and a sensorimotor system. Anatomically they are combined in close and complex interrelationships; functionally they in a measure serve different individual ends though integrated in the conduct of the animal as a whole.

The *vegetative nervous system* relates the various organs of the body for the purpose of the upkeep of the machine. The activities of

[1] The authors of this text-book assume that whereas the so-called "student" is supposed to be best served by telling him what is established in neurological science as "facts," he is also badly served by not telling him that all so-called "facts" are but stepping stones, constantly in revision. They are but provisional abstractions, to be scrapped with the advance of observation and supplanted by better working hypotheses. The student who wishes to benefit by the recorded data of the problems which are here but thumb-sketched is referred to the following collections of provisional "facts." Herrick: Introduction to Neurology, latest edition The Brains of Rats and Men, 1926. Gaskell, W. H.: The Involuntary Nervous System, 1916; Gaskell: Origin of the Invertebrates, 1908; Parker, G. H.: The Elementary Nervous System, 1919; Fortuyn: Die vergleichende Anatomie der Wirbellosen Thiere, 1920; Kappers: Die vergleichende Anatomie der Wirbelthiere, 2 vols., 1921–1922; Higier: The Vegetative Nervous System, 1919; Müller, L. R.: Lebensnerven, 2d Edit., 1923; Guillaume: Le Systeme Sympathique, 1921; Laignel-Lavastine: Pathologie d. Sympathique, Paris, 1923; Dresel: Die Erkrankungen d. veg. Nervensystems, Berlin, 1922, also Ergeb. d. ges. Med. II, 1921; Zeit. f. d. g. i. Med., 1923, vol. **37**; Greving, Lewy, Glaser, Spiegel and others. Various contributions of Langley. Carpenter, F. W., Recent Contributions to the Physiology of the Autonomic Nervous System, Psychological Bulletin, April 15, 1917. Papers by Raymond Dart, See Jour. Nerv. and Ment. Dis., December, 1924. The voluminous studies on the problems here involved are found in the many literature citations found in these volumes and also in part indicated here.

the vegetative nervous system permit the satisfaction of organic bodily needs and thus may be said to be the media through which the psyche acts thus constituting the affective or emotional attitudes of the various types of well-being in separate segments of the body.

The *sensorimotor system* has developed as an effector mechanism to satisfy the needs registered, conscious as well as unconscious, by the vegetative system. Its general functions and their special disturbances are taken up in later sections of this work.

This section would treat of the structure and the functions of the vegetative nervous system. A number of differing terms have been used for this system. The oldest was the Sympathetic Nervous System. It is also called the Autonomic Nervous System, also the Neuro-visceral, or the Splanchnic System. The name is more or less immaterial if the work done by it be kept in mind.

The vegetative nervous system consists of those nervous structures which supply, by afferent and efferent pathways, impulses to and from the special sense organs, smooth and striated muscle fibers, and all those automatically working organs such as the heart, lungs, intestines, genital apparatus, bloodvessels, excretory glands, skin and organs of external and internal secretion, such as the liver, stomach, pancreas, intestinal gland, and the thyroid, thymus, adrenals, parathyroid, hypophysis and epiphysis, etc., respectively.

Certain of these are closely related histologically and functionally to nerve structures, hypophysis (anterior, and pituitary, posterior lobes), epiphysis, thyroids, parathyroids, suprarenals, and blood glands, and have been variously brought together as the endocrinous glands or glands of internal secretions—while others are distinctly non-nervous, liver, pancreas, testicles, interstitial bodies, thymus, ovaries, uterus, lungs stomach, heart, etc., but their functions, like those of the endocrine glands, are automatically controlled and interrelated by one or other of the two portions of the vegetative system, the *sympathetic proper,* and the *parasympathetic* or *autonomic.*[1]

All of these structures are functioning all of the time, twenty-four hours every day, and their *disturbances* are manifold. These result from (1) affect stimuli such as fear, rage, jealousy, pain, as seen in many neuroses and psychoneuroses represented as palpitation of the heart, anorexia, fainting, crying, diarrhea, mydriasis, eosinophilia, etc., or (2) from infections or toxemias, causing reactions such as reddening, swelling, gooseflesh, tachycardia, dryness of the mouth, stenocardia, gastric or visceral crises, Argyll-Robertson pupil, etc.,

[1] See Higier, Vegetative Neurology, Tr. by Kraus, Nervous and Mental Disease Monograph Series, 1919, No. 27. Müller, L. R.: Die Lebensnerven, 1924. Eppinger and Hess: Vagotonia, Nervous and Mental Disease Monograph, No. 20, New York, 1915. Gaskell: Involuntary Nervous System, 1916. Nöel Paton: Nervous Regulators of Metabolism, 1913. Giovanni: Patologia del Simpatico, 1876. Castellino e. Pende: Patologia del Simpatico, 1915. Pottenger: Symptoms of Visceral Disease and Their Relation to the Vegetative Nervous System, 1922. Onuf and Collins: Experimental Research Sympathetic Nervous System, Arch. Neurol. Psychopath., 1900, vol. **3**, and the works cited on p. 146. Schilf: Das Autonomic Nervensystem, Leipzig, 1926.

or (3) from constitutional defect states showing such syndromes as myxedema, acromegaly, scleroderma, dwarfism, mongolism, eunuchoidism, myopathies, disordered blood states, etc.

It has required a long time for orthodox medicine to accept what has been known empirically for centuries that emotional factors are capable of producing acute as well as chronic structural alterations (so-called organic disease), as well as being constantly operative in causing so-called, and badly so-called, *functional* disease. The study of the vegetative system has enabled this gap in knowledge to be bridged by showing the exact mechanisms by which these structures, acting upon hormone production, may produce structural alterations.[1]

The role of this system in its reactions to mental stimuli has been studied recently and for a period of years by Pavlof, Cannon and others by laboratory methods and has helped to give an experimental status for empirically known events. Starting with the general thesis that the vegetative nervous system makes important synaptic connections in the cranial-cervical, thoracic-lumbar and sacral regions of the cerebrospinal axis, these studies have attempted to correlate the activity of these portions of the system with differing types of emotions. As a result of physiological experiment whenever the innervation from the thoracic-lumbar (sympathetic) portion of the system meets, in a viscus, the innervation from either the cranial-cervical or the sacral portion, there is an opposition in the effects produced. In the eye, the contraction of the pupil is due to impulses received from the cranial autonomic, while the dilatation of the pupil is effected by nerve fibers which belong to the thoracic portion of the sympathetic. Again, the secretory and tonic motor innervation of the stomach come through the vagus nerve and sympathetic neurons which have their origins in various parts of that system. Stimulation of the splanchnic nerves causes an inhibition of both the secretory and motor functions of the stomach. The viscera of the pelvis are likewise doubly innervated. The sympathetic neurons from the thoracic-lumbar segment cause relaxation of the lower end of the intestine. The sacral parasympathetic fibers for the same organs cause contraction. The bladder and reproductive organs are similarly innervated. Corresponding to the three parts of the vegetative system and the organs associated with them, according to Cannon, there are three types of response to emotions. The cranial portion of the system, by regulating the secretion and motion of the digestive organs, is concerned with building up the reserves of bodily strength. The emotions correlated with these physiological activities are bodily satisfaction and well-being. The sympathetic system is largely reinforced by the suprarenal gland hormones. The physiological effects of the secretion of this gland are almost closely related to the effects of stimulating the thoracic-lumbar sympathetic system; the pupil is dilated, the heart is accelerated, the functions of the stomach are inhibited, and the glands of the skin and

[1] Dresel, K.: Die Bedeutung der vegetativen Zentren für die innere medizin. Med. Klinik., 1923, **19**, 1417. Pavlof, Conditioned Reflexes, Tr. by Anrep, Oxford, 1927.

the erector muscles of the hair are excited. Glycogen in the liver is liberated in the form of dextrose, with the result that sugar is available for muscles. The emotions which call for a sudden summoning of muscular energy are rage and fear, since correlated with them are the instincts of fighting and flight. The functions of the pelvic viscera are mainly concerned in emptying accumulated secretions. The emotions depending on these functions are those of satiety and repletion. Cannon's *very crude classification* of emotional reactions *is hardly to be accepted*,[1] but the underlying facts concerning the interrelationship between psychogenic (symbolic), *i. e.*, emotional factors and neuronic mechanistic alterations of physical structures are incontrovertible.[2]

The vegetative nervous system may be conceived as the primitive, archaic portion of the ganglionic or metameric system of the lower vertebrates.[3] The chief central switchboard synapses or centers are located in the inter- and mid-brain.[4] In the evolution of higher animals these vegetative structures have built connections with the relatively more important (*i. e.*, for purposes of civilization and culture) neopallium or cortex, but there has been built up a definite series of cortical associations, as the primitive rhinencephalon (olfactory organs) have had added to them the cortical representatives from all of the visceral components. Thus it has come about that the ganglionic system which in man serves the vegetative functions of the body is represented in the primary metameres, the spinal cord, again in the brain stem, central gray matter and midbrain, optic thalamus (hypothalamus), tuber cinerium, locus niger, hypophysis etc., and finally in the cortex where the different organs of vegetative function have localization as surely as those of the bodily musculature. As yet the cortical areas for visceral associations are vaguely outlined but the brutal vivisections of the World War, when more carefully analyzed, have already done much to make the cortical stations of the visceral reflex arcs more accurately localized. There are cortical representatives for every structure in the body probably. In Petromyzon, and in the lower fishes, Amia, etc., the first beginnings of telencephalization of affective focal organizations are foreshadowed.[5]

The conceptions of anatomists concerning the vegetative system are

[1] In the light of psychoanalytic studies these emotional reactions are much better comprehended.

[2] Cannon: Fear, Rage, Hunger and Pain and the Nervous System, Appleton & Co., New York. For a fuller and more penetrating study of this problem consult Kempf. The Autonomic Functions and the Personality. Nervous and Mental Disease Monograph Series, 1918, No. 28. Higier, loc. cit., Laignel-Lavastine, loc. cit. Renne, O.: Jetzige Stand der Lehr des vegetativen Nervensystem, Deutsch. med. Wchnschr., August 12, 1920. Hess, W. R.: Ueber die Wechselbeziehungen Zwischen psychischen und vegetativen Funktionen, Zurich, 1925. Also Kempf, E., Psychopathology, St. Louis, 1920. Edinger's earlier studies are classics. For the analytic study of these reactions see the enormous Freudian literature.

[3] Raymond Dart: Jour. Comp. Neur., April 15, 1924, Jour. Nervous and Mental Disease, 1925.

[4] Greving: Lage u. tätigkeit der vegetativen Zentren in Zwischenhirn, Ztschr. f. d. g. Neur. u. Pschy, 1923, **83**, 22. Ergebnisse der Anat. u. Entwichlg., 1922, **24**, 348.

[5] See papers of Tilney F. Spiegel, loc. cit. Herrick: The Brains of Rats and Men, 1926. Kappers: Comparative Anatomy, l. c. etc.

rapidly developing. Their detailed consideration cannot advantageously be followed here.[1]

Vegetative Receptors.—Vegetative receptors are widely distributed throughout every organ of the body. Each year newer details are being added as to vegetative receptors in skin, muscle, ovary, testicle, bone, etc. These may be distinct from those utilized by the sensorimotor system[2] or the same receptor structure—probably a compound receptor—may be utilized for different types of stimuli.[3]

Langley's earlier teaching that the vegetative system was not a reflex arc, as the sensorimotor system is incorrect. True vegetative reflex arcs are universal. Many of the afferents however, are mingled with sensorimotor afferents, mostly unmyelinated, hence were overlooked when cruder histological methods were employed. Furthermore many receptors are compound. They function for differing qualities of stimuli. The vegetative afferent path is certainly partly, if not chiefly unmyelinated, and has been neglected in most all physiological and pathological interpretations. Hence a double system is functionally active, one chiefly utilized in the upkeep of the machinery itself, the other serving for an effector mechanism to keep the organs and the individual as a whole going. Many vegetative receptors, lie within the organs themselves, many of which are being discovered by newer technical microscopic methods, and unmyelinated and myelinated fibers carry the primary cosmic energy stimuli, chemical or physical, from the recipient structures to be transformed and recombined for the effector purposes of the body, which may be expressed as growth, or as social conduct.[4]

By way of illustration. The idea here advanced would regard most if not all receptors as pluri potential. Take the retina as a *type*. It receives light stimuli which may be separated into (1) *Waves* per se, (2) waves as of certain length and rapidity (*color*) red, green, etc; (3) waves, colors (*form*), as leaves, flowers, fruits; (4) waves, color, form (*values*) *i. e.*, *good* for food, *bad* as dangerous animals, etc. (*symbolic*). Thus waves, color, form, symbolic values, are all streaming into the receptors. The separate components are synthesized along the optical and related pathways. The primary ones are utilized for metabolic purposes, *i. e.*, the upkeep of the structures that make up the pathway: the later ones are used for effector purposes of conduct. Just where

[1] See Ranson's many papers, particularly Jour. Comp. Neurol., August 15, 1919, for a new but inadequate systematization. Also see Müller, L. R., loc. cit. and Spiegel, loc. cit.; Dresel, loc. cit. (important beginnings of nervous pathways involved in metabolic activities). Laignel Lavastine, loc. cit. Kraus: Die Pathologie d. Person, would speak of "Vegetative System." See v. Bergmann: Funktionelle Pathologie d. vegetativen Nervensystems, Berlin, 1926.

[2] Carpenter: Nerve Endings of Sensory Type in Muscular Coat of the Stomach, Jour. Comp. Neurol., October 15, 1918.

[3] (See Sensory Sympathetic Type in Muscle. See Dart also in Pancreas and other viscera. See Cajal, et al. Bloodvessels, see Dart. Müller Die Lebensnerven, etc.)

[4] See the many studies of Kuntz: Anatomical Review, 1922, **24**, 193, for instance for myenteric and submucous plexus reflex arcs. When Cannon and his pupils speak of separating an organ from its vegetative nervous system they are talking nonsense. It cannot be done. Vegetative receptors and effectors lie within the organs themselves.

the reflex arc patterns are shunted off for the separate functions are problems which anatomists and physiologists are commencing to analyze. The problems are intricate but they are being resolved.[1]

Spinal Synapses.—For many of the reflex activities of the vegetative system these are located chiefly in the lateral gray of the spinal cord (Jacobsohn) (Fig. 44 and Plate III). Jacobsohn[2] has gone over the old work by examining a complete set of serial sections from a human spinal cord stained by the Nissl method. Malone[3] has carried these studies up into the midbrain.

According to Jacobsohn, there are two columns of vegetative synaptic cells in the spinal cord (Fig. 44).

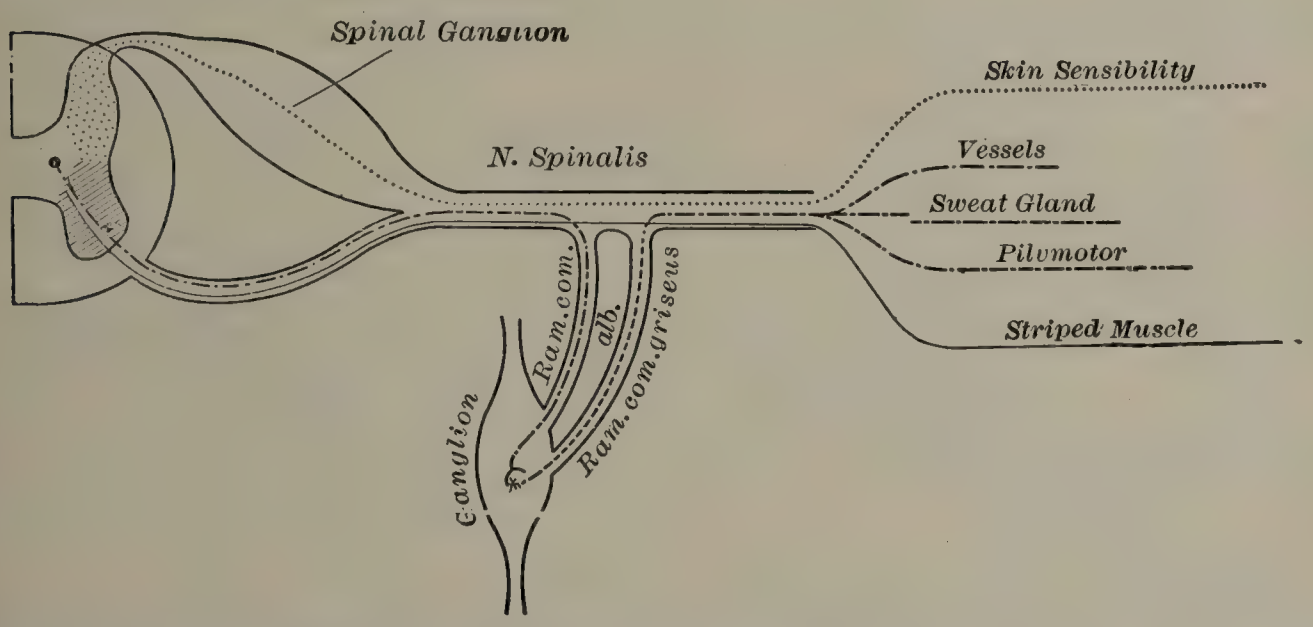


FIG. 43.—Diagram of the central connections and peripheral distribution of the vegetative system,—.—.—; the motor, ----, and the sensory, ..., fibers. (Higier.)

I. The lateral cell-column is composed of two parts: (*a*) An upper column corresponding to Langley's "Sympathetic System." This lies in the lateral horn of the dorso-lumbar cord (C_8—L_3) and is designated the Nucleus sympatheticus lateralis superior s. cornu laterale. (*b*) A lower column lying in the sacral cord, from S_2 caudalward. It is placed between the anterior and posterior horns and is designated the Nucleus sympatheticus lateralis inferior s. sacralis.

The dorso-lumbar column is thickest at the upper dorsal segments and at the upper lumbar segments, that is near the cervical and lumbar enlargements where there are collections of ganglion cells for the extremities.

[1] Consult Winkler, C.: Manuel de Neurologie, 1920–1922–1927. Kappers, A.: Die vergleichende Anatomie des Nervensystems, 1921–1922. In the work of these Dutch investigators these dynamic concepts receive more adequate attention and indicate more essentially the radical departure from the static concepts which have been heretofore in the focus of a neuropsychiatry. Also consult Dresel, K.: Experimentelle Untersuchungen zur Anatomie und Physiologie der peripheren und zentralen vegetativen Nervensystems, Ztschr. f. d. g. Exper. Med., 1922, **37**, 373, and the works of Laignel-Lavastine, Müller and Greving, loc. cit.

[2] Jacobsohn: Ueber die Kerne des menschlichen Rückenmarks, 1908. Ueber die Kerne des menschlichen Hirnstammes, 1919. The early work of Onuf and Collins, loc. cit. should be included here.

[3] Malone: Ueber die Kerne des menschlichen Diencephalon, 1910.

II. The medial cell column lies in the medio-ventral marginal zone of the anterior horn of the lumbosacral cord, from L_4 distalward and is designated the nucleus sympatheticus medialis s. lumbosacralis. Low down in conjunction with the nucleus radialis, it forms an area of groups of cells which takes up almost the entire anterior horn and the space between.

All the cells of these three columns have the following three characteristics: (1) They are always in groups and closely packed together. (2) They are long, round, club-shaped or vesicular, rather small, round cells. (3) They have a homogeneous appearance and are usually stained more darkly than the larger, less closely packed motor and sensory cells.

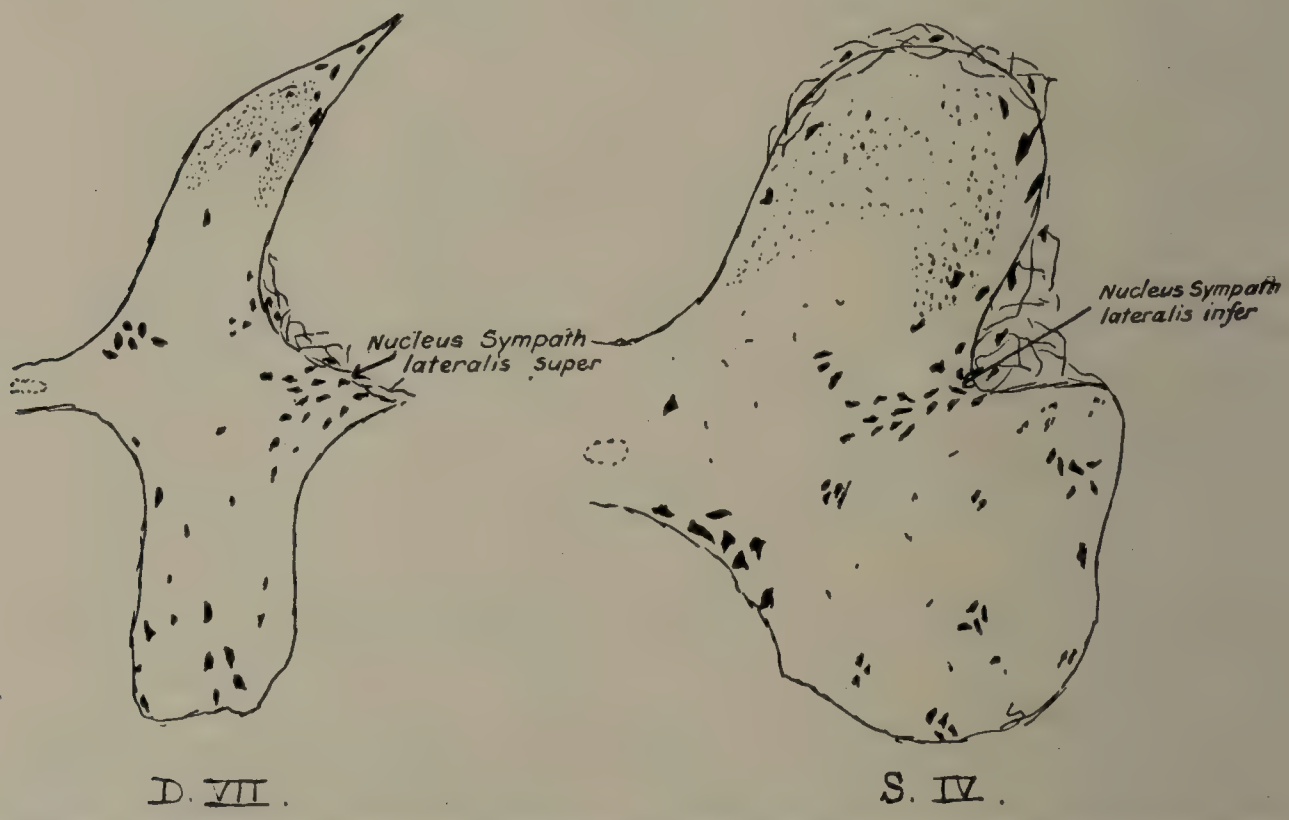


FIG. 44.—Sympathetic nuclei at the seventh dorsal and fourth sacral levels of the spinal cord. (Jacobsohn.)

A cross-section of the *medulla,* at a level in which the most important cranial nerves are placed, shows, in addition to the large multipolar motor cells, small circumscribed groups of oval or pear-shaped unipolar cells (paracentral cells). These are the nuclei from which the preganglionic rami communicantes spring. The nuclei are as follows (see Plate VI):

1. Nucleus pupillaris (Bernheimer)—median to the oculomotor nucleus.
2. Nucleus lacrimalis—median to the facial nucleus.
3. Nucleus salivatorius superior (Kohnstamm)—dorsal to the facial nucleus.
4. Nucleus salivatorius inferior (Kohnstamm)—near the glossopharyngeal nucleus.
5. Nucleus dorsalis vagi—between the motor and sensory vagus nuclei, *i. e.*, between the nucleus ambiguus and the nucleus solitarius vagi.

A closer analysis of the anatomical position of the various vegetative nuclei shows that their relation to sensory and motor nerves is the same as in the spinal cord.

As a paradigm Higier takes the most orally placed ganglion—the ciliary. It is of great clinical significance. For years well known authors have spoken of this ganglion as a spinal ganglion or a mixed ganglion (Schwalbe, Budge, Remak, His, Van Gehuchten, Kölliker, Bach). A cross-section of the brain stem shows the following: the oculomotor nerve is the anterior motor root, the trigeminal nerve is the posterior sensory nerve, the Gasserian ganglion corresponding to a spinal ganglion while the ciliary ganglion is the vegetative ganglion.

The white rami go from the visceral nuclei by way of their corresponding motor nerves. L. Müller and Dahl have tried to establish this on a firm basis.

Afferent and efferent pathways may be differentiated in the cranial ganglia as well as in those of the sympathetic chain. The white rami communicantes pass by way of the anterior motor roots in the cranial as well as in the spinal region. Many rami albi spring from cranial nerves and have been anatomically described, though the part they play was not even thought of.

1. Radix motorica, or R. albus ganglii ciliaris—from the oculomotor.
2. Nervus petrosus superficialis major, or R. albus ganglii sphenopalatini—from the facial.
3. Nervus tympanicus and its process going to the otic ganglion—nervus petrosus superficialis minor, or R. albus ganglii otici—from the motor part of the glossopharyngeal nerve.
4. The chorda tympani, which sends fibers as the R. albus to the submaxillary ganglion—from the motor nervus intermedius.

The post-cellular tracts of the cranial ganglia are unmyelinated like those of the sympathetic chain. They supply smooth muscle and glands exclusively. When they have a long path to follow to reach the organs which they innervate, they join spinal sensory nerves.

After making the first synaptic junctions, where distributions to different levels may take place, the stimuli, which are to serve the vegetative functions travel chiefly by way of the white ramus—ramus communicantes albus to a second synaptic junction. The collection of nerve structures making up this synaptic junction are termed, for the most part, the *sympathetic ganglia* (see Figs. 44, 45, 46, and Plate III). A further redistribution of the stimuli takes place at this level from which the stimuli travel by means of the ramus communicantes griseus (mixed with motor fibers of the sensorimotor system from the anterior horn synapse), and find their next synapse (typically) in the viscus of final distribution. Here the analogue of the motor horn cell is located and the final effector vegetative function, physical (contraction or relaxation) or chemical (glandular—anabolic or catabolic) takes place.

This reflex arc (various types of which are recognized) is shown in the work of Gaskell to differ materially from the ordinary reflex arc

of the voluntary system as is seen in the accompanying illustrations (Figs. 42, 44, 45, 46, and Plate III). This is the original type for each metamere, the symmetry of which, however, has been much distorted. This distortion is shown as an irregular or inconstant localization of the ganglia, or synapses, or as an inconstancy of the communicating branches, irregular distribution of the synapses in the cerebrospinal axis or as an incongruity of the embryonal metameres with the spinal and cranial segments (Figs. 45 and 46).

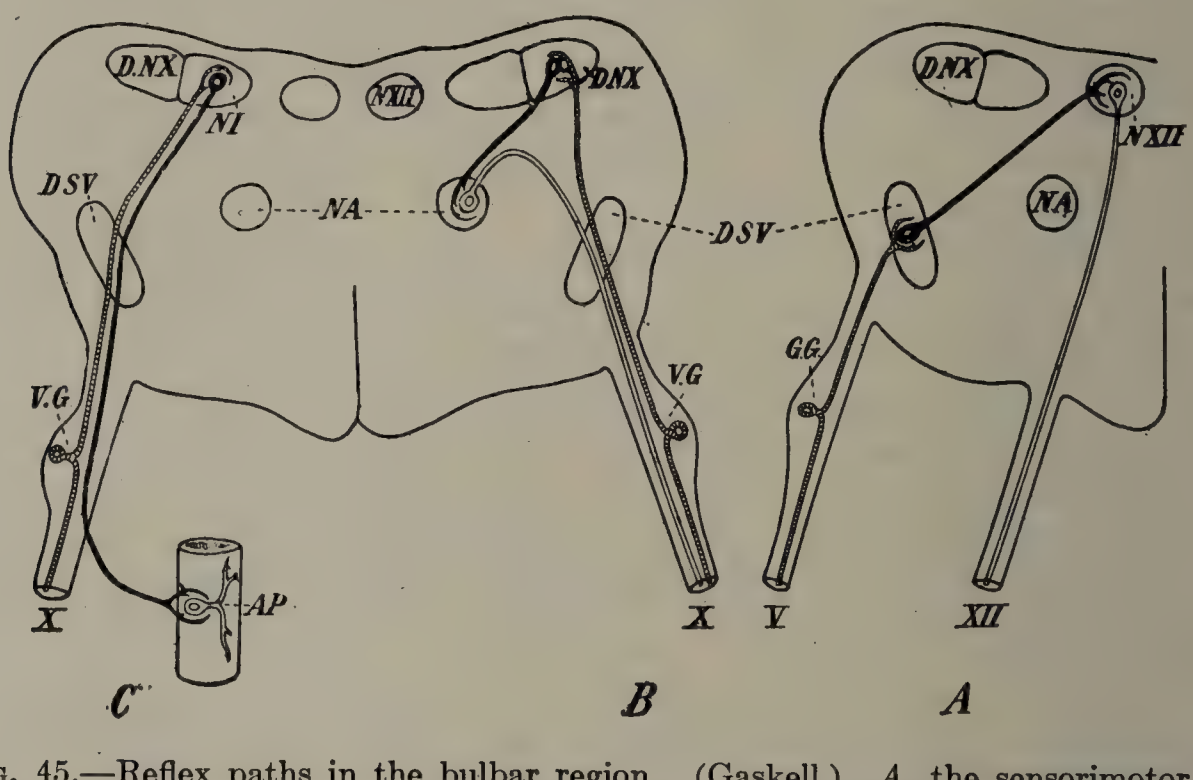
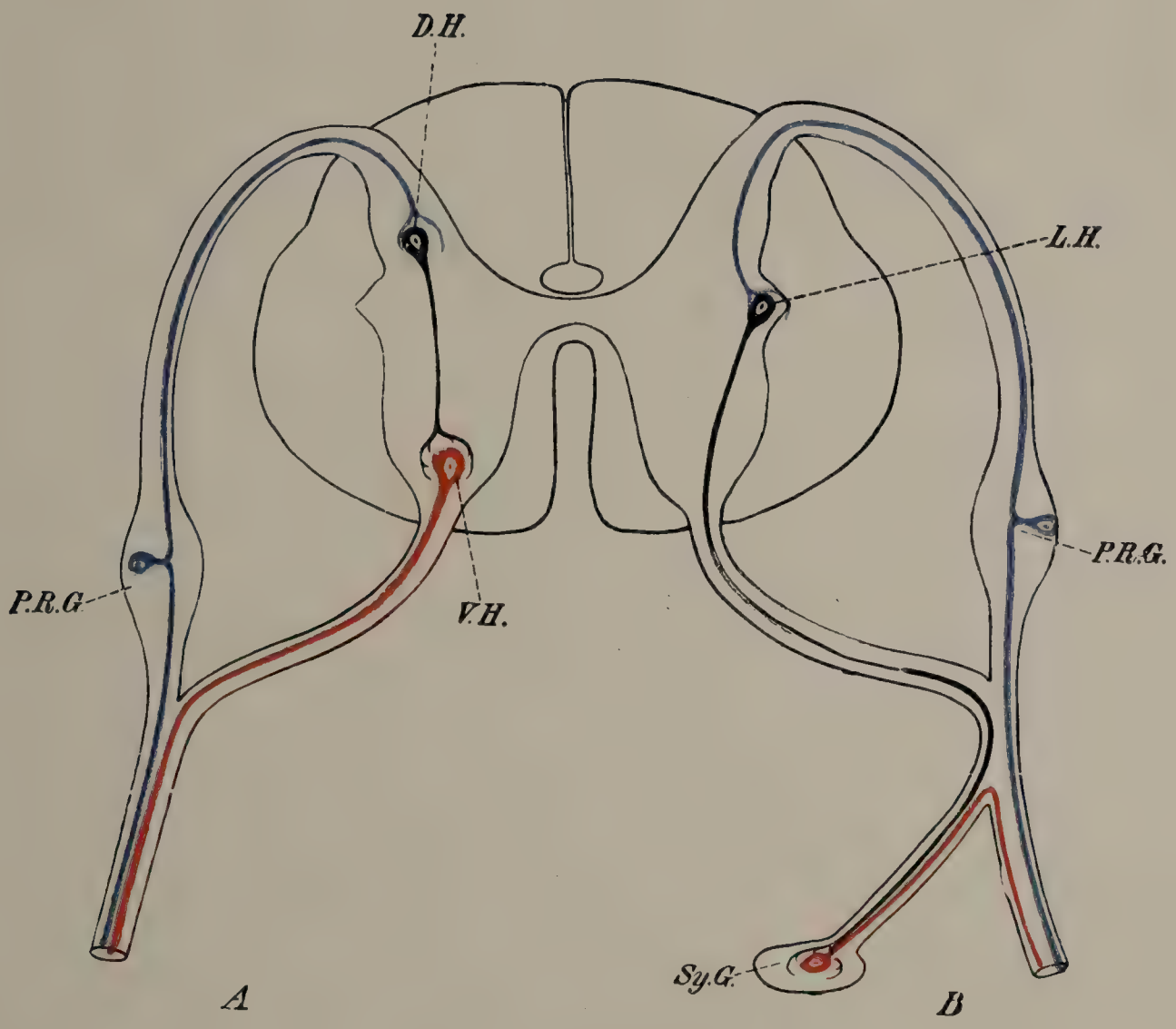


FIG. 45.—Reflex paths in the bulbar region. (Gaskell.) *A*, the sensorimotor system. The sensory neuron runs in the fifth nerve, *V.*, its cell bodies forming the Gasserian ganglion, *G.G.* The connector neuron synapse occurs in the descending root of the fifth, *D.S.V.*, and an effector synapse in the twelfth nerve nucleus, *N.XII.*; *B*, the sympathetic system. The receptor neuron runs in the tenth nerve, *X*, the cell bodies making up the vagus ganglion, *V.G.*, making its connector synapse in the dorsal vagus root, *D.N.X.* The connector fiber makes its synapse with the effector in the nucleus ambiguus, *N.A.*; *C*, the parasympathetic or autonomic system. The receptor neuron runs in the tenth nerve, *X*. The first connector synapse forming the nucleus intercalatus of Staderini, *N.I.*, part of the dorsal vagus synapse group, *D.N.X.* The connector neuron runs out with the vagus, *X*, finally making an effector synapse in some viscus, or plexus. Auerbach's plexus for the intestines, etc.

The chief anatomical features concerning this process of distortion in the head region may be recapitulated as follows: In the skull the distortion is greatest as many metameres are anatomical conglomerates. Many of the ganglia are modified intervertebral ganglia, such as the geniculate and the Gasserian; others are compound ganglia due to the amalgamation of a spinal with a sympathetic ganglion as the jugular and vagus ganglia; other pure intervertebral ganglia are the ciliary, otic, sphenopalatine, submaxillary and sublingual which supply the smooth muscles of the eye, the vessels, the tear, salivary and mucous glands respectively. Some of the chief anatomical features for the head ganglia may be seen in the superb charts from Müller.[1]

[1] Higier, loc. cit.

PLATE III



Reflex Paths in the Cord. (From Gaskell.)

A, of the sensorimotor voluntary system. The receptor neurons run in the posterior root, their cells lying in the posterior root ganglia, *P.R.G.* The connector synapse neurons lie within the dorsal horn, *D.H.*, and make with the effector neurons lying in the ventral horns, *V.H.*, a synapse with the motor neuron, which emerges from the cord as the motor spinal nerve. *B*, of the vegetative nervous system. The receptor neurons run in the posterior root as a sensory neuron, making its first synapse in the cells of the lateral horn, *L.H.* (Jacobsohn's sympathetic nuclei). The connector neuron runs out with the motor nerve as the white ramus communicans, making its synapse with the effector neuron in the sympathetic ganglia, *Sy.G.* The effector neuron runs in gray ramus communicans to the viscus directly or through intercalated connector neurons, making a final synapse within the viscus itself.

The upper ganglion, ganglion cervicale supremum, obtains its precellular fibers from the last cervical (C8) and upper dorsal (D1–3) segments and innervates the vessels, hair muscles and skin glands of the head, the dilator pupillæ and Müller's orbital muscle. The inferior

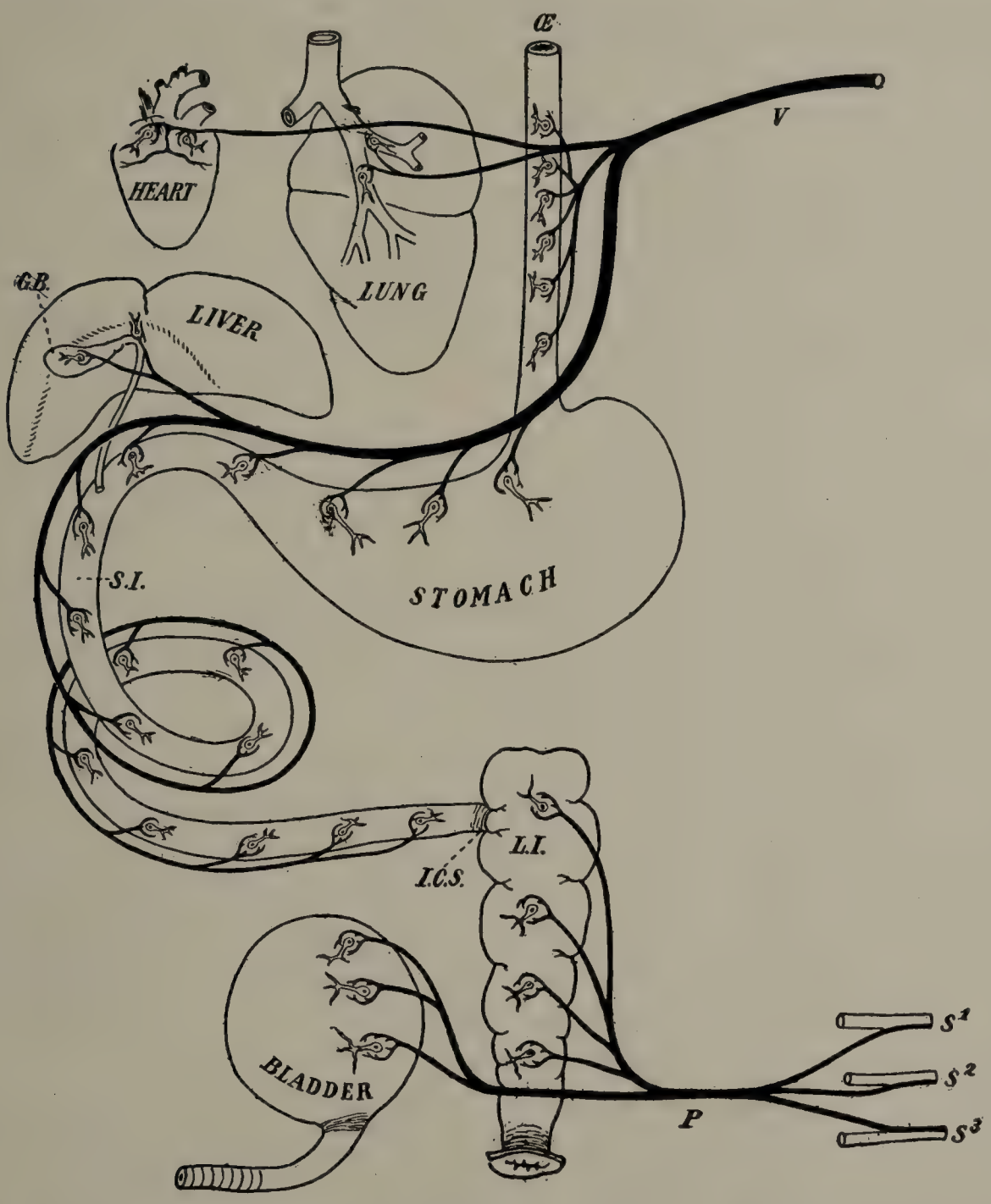


FIG. 46.—The vagus nerve, *V.*, contains connector and effector neurons as far as the ileocolic sphincter. Further effector (sympathetic? and autonomic) neurons lie within the viscera themselves. The pelvic nerve, *P.*, contains connector (and effector) neurons for the sacral outflow, terminal effector neurons lying within the walls of the large intestine and bladder. The vagus thus carries connector neurons to the motor effector cells of the heart, *H.*, which, Gaskell states, have to do with the slow wave-like contractions only (?) found in certain tortoises. The vagus also carries connector fibers to the effectors in the bronchi, *La.*, and also connector fibers to the effectors within the walls of the gall-bladder and bile ducts, *L.I.* (vagotonic icterus), to the walls of the esophagus, *Œ*, the stomach, *St.*, and small intestine, *S.I.* The pelvic nerve, synapses in the sacral root, S^1, S^2, S^3, carries connector fibers to the effectors in the large intestine *L.I.*, and bladder, *B.* (Gaskell.)

cervical ganglion with its closely related stellate ganglion derives its preganglionic fibers from the D1–5, and gives rise to the accelerator nerve of the heart and probably the vasoconstrictor fibers of the pulmonary vessels.

The largest ganglion of the abdomen, the celiac, has its chief root in the celiac plexus in the major and minor splanchnic nerves, the former of which comes from D4–9, the latter from D10–12. As the mesenteric nerves they innervate the stomach glands, liver, pancreas, spleen, kidneys, adrenals, and intestinal glands as far as the ascending colon. The inferior mesenteric ganglion receives its precellular fibers from L1–3 and sends its postganglionic fibers to the colon and as the hypogastric, in part, to the anus, bladder, sphincter of bladder and genitals. In these abdominal ganglia, and the plexus of Auerbach and Meissner, intercalated vegetative neurons are found.

Higier, loc. cit., to whose valuable monograph the student is referred for more detailed study of the vegetative system, follows a group of investigators and includes in the sympathetic system various glands which contain chromaffin cells. These cells all develop from the sympathetic anlage, and are therefore in very close relationship to the ganglion cells. They are found partly separate, partly in small groups in the sympathetic system, in the sympathetic ganglia, or in large nerve networks about bloodvessels. Where they are found as individual bodies they are designated *paraganglia*.

They are for the most part spherical with a connective-tissue capsule and are broken up by large nerves and bloodvessels, between which the chromaffin cells lie in unequal masses.

Of the larger chromaffin bodies the following four may be named: (1) the carotid paraganglion incorrectly spoken of as a gland (carotid gland or epithelial organ). (2) The coccygeal paraganglion incorrectly spoken of as the sacral gland (coccygeal gland). (3) Aortic paraganglion at the bifucation of the aorta. (4) The best studied and largest chromaffin body, the suprarenal body, the medulla of the adrenals, from which the active blood-pressure raising adrenalin is produced, a substance which stimulates the sympathetic system, and plays an enormously important role in the body.

Clinical Considerations.[1]—Insofar as only a general survey of the anatomical data concerning the spinal synaptic stations of the vegetative pathways, is here possible, only a general account of certain clinical correlations will be made. These concern the symptomatology within the vegetative sphere arising from various types of lesions affecting the spinal cord as implicating the synapses (centers) and their segmental connections. They are considered further in the later appearing descriptions of spinal cord disorders, forming but part of the complex syndromes of spinal lesions.

The disturbances in the sympathetic innervation of the eye, sympathetic ophthalmoplegia, betray themselves as well in the triad of the Horner's syndrome, miosis, ptosis, enophthalmia, as also by a single one of these symptoms. This indicates that the tracts coming from the cortex, that is subcortical ganglia, that go to the cilio-spinal

[1] Spiegel, E. Die zentrale Lokalization autonomer Funktionen, Zeit. f. d. g. Neur. u. Psych. Ref. u. Ergeb., 1920, **22**, 142, a most important summary. Journal Nerv. and Ment. Dis., June, 1922.

center, to the cells of the lateral horn at the level of C8 and D1 contain their own separate fibers for the innervation of the pupil of the eyelid and of Müller's muscle in the orbits. These fibers have a common field in the lateral column, and pass in isolation the one from the other. The picture becomes complicated by the fact that near the pupillo-dilatory fibers there seem to be also tracts carrying stimuli for pupillary contraction. Shima has observed that at lesions of the central sympathetic paths of the eye the sensitiveness of the pupil for local adrenalin action is raised so that on the side of the lesion one may obtain a mydriasis. As for the sweat glands the central autonomic paths may influence also the sensitiveness of the peripheral nerve endings. Thus an altered irritability of autonomic organs may be observed in a central affection.

Passing to the lower end of the spinal cord there are many observations relative to the pelvic viscera which are of much clinical importance.

Lesions of the spinal cord lead to more or less permanent functional disturbances in the vegetative system and these changes merit consideration in the segmental diagnosis of spinal cord affections. Down from the third sacral segment the characteristic multipolar cells of the anterior horn which are known as centers of the striated musculature are more and more replaced by peculiar, much smaller bludgeon- or spindle-like thickly arranged cells, that type which Jacobsohn has described as characteristic for sympathetic elements of the spinal cord. In how far the N. sympathicus medialis lumbo-sacralis of Jacobsohn are coördinated with certain organs of the pelvis it is impossible to say either from the comparative anatomical investigations in man and woman at different ages (Bräutigam, Sakai) or from the cases analyzed up to the present time (de Buck, Bruce, Irimesca, and Parhon). In none of these cases was a hollow organ of the pelvis alone affected but always at the same time other organs or the striped musculature, also, so that in the spinal cord there were always several groups of cells in axonal degeneration. It is not to be expected that definite groups of cells are coördinated with definite organs, because researches concerning the distribution of the innervation of the voluntary musculature in the spinal cord have shown that several groups of cells are involved in the innervation of one muscle, respectively of one group of muscles. Thus it comes to pass that the vegetative synapses in the spinal cord for the pelvic organs also extend themselves vertically along the cord. Therefore the statement that a certain hollow organ is represented by a certain segment is to be understood only in the sense, that this segment chiefly but not exclusively sends and receives different fibers to and from the organ concerned.

Thus L. R. Müller, on the basis of the cases set down in literature (I. Kirchhoff, Kocher, Oppenheim, Sarbô, Schlesinger, etc.) and of his own observations originally placed a center for the bladder in the fourth, a center of the M. sphincter ani externus in the fifth sacral segment, but has recently taken his stand against the fixed acceptance of a certain single segment. He also states that the pelvic nerves

which effect the discharge of urine descend from the lower sacral segments, the hypogastric nerves which inhibit it from the upper lumbar segments. The question of the stricter segmental innervation of the bladder loses its importance to the clinician because, as L. R. Müller emphasized at first and the experiences of the late war confirmed (Marburg and Ranzi, Schwarz), in diseases of the conus terminalis or of the cauda equina disturbances of the bladder may arise quite similar to those in transverse lesions in higher segments. Incontinence with dripping of urine and ability of the bladder to discharge its contents as well as retention also, an inability to relax the M. sphincter vesicæ may arise from conus lesions. Further the picture of the automatic bladder described first by Müller and exhaustively analyzed by Head and Riddoch and Lhermitte develops itself, to a state in which the urine is automatically forced out at certain intervals. The fact, experimentally proved by Zeissl, Langley, Anderson and others, that the Nn. erigentes coming from the sacral segments, effect the emission of urine, the Nn. hypogastrici located in the lumbar segments inhibit this function; this fact of antagonistic influence of lumbar and sacral centers has not yet found clinical expression. In order to distinguish between a disturbance involving the conus and a higher transverse lesion the fact might be taken into consideration that a destruction of sacral synapses but not a transverse lesion above the conus can lead to a loss of the reflex control of the bladder which passes over the spinal cord. Thus L. R. Müller observed that in a transverse lesion in the dorsal region an irritation of the skin on the lower extremities effected an emission of urine, a reflex which was absent in an injury of the sacral region.[1]

In the same way in disturbances of the innervation of the rectum a lesion of the centers situated in the conus, S5, may be best distinguished from a lesion of the central path by the fact that only in the first case is the reflex arc passing over the spinal cord interrupted so that the contraction of the sphincters, among which the striped M. sphincter ani externus is certainly to be taken into consideration, fails on touching the mucous membrane of the anus.

So far as the internal genital functions are concerned, the act of birth in human beings can take place even if the sacral synapses are destroyed, as an observation of Higier tends to show. Concerning the masculine genital, L. R. Müller originally held that S2 as center of erection, S3 as center of ejaculation, while he assumes in a more recent work together with Dahl, that the ejaculation reflex passes over the upper lumbar segments, that of erection over S3. Recently on the basis of observations upon dogs, in which, after the removal of the lower sacral region, erection upon friction of the penis ceased, he denies a reflex arc existing outside the spinal cord. Experience in man is, however, contradictory and the existence of a peripheral reflex arc outside of the spinal cord is to be maintained. In complete destruction of the cauda

[1] Dennig: Die Innervation der Harnblase, Berlin, 1926.

equina, histologically controlled, Marburg and Ranzi observed erections that were not caused psychically but could be induced by mechanical irritations of the penis.

This fact, that erection stimuli do not pass merely over a spinal but also over a peripheral autonomic reflex arc is important for estimating the symptom of the "dissociated disturbance of potency" described by H. Curschmann and Boenheim.

Ejaculation in its end phase, the emission of the semen from the pars prostatica urethræ, is known to be caused by the contraction of the striped M. bulbo- and ischiocavernosus. The normal innervation of this process is bound with the intact condition of the reflex arc passing over the spinal cord. Proceeding from the supposition that the ejaculation center lies in S3 under the erection center situated in S2, Boenheim considers the absence or the diminution of the ejaculation while capacity for erection remains as characteristic for a localized affection in the conus terminalis, as far as this affection lies in the spinal cord. Since, further, the orgasm is caused by the sensation of the muscle contractions which take place at ejaculation, a lesion in the conus, which in case of lesion of the ejaculation center interrupts also the centripetal fibers from the genitals, must lead to a reduction or a suspension of the orgasm when eroticism is present. The author means that this symptom picture of the dissociated disturbance of potency, that is, disturbance of ejaculation when erection is maintained and failure of the orgasm when eroticism is retained is of value in the differential diagnosis of conus affections. The fact mentioned that erection may be caused also through the reflex arc outside the spinal cord, as Boenheim himself admits, must limit considerably the value of this phenomenon. Therefore it is possible that as this symptom appears in an affection of S3 alone, so also it may appear in simultaneous affection of the erection center. Besides, it may appear just as well in conus as in caudal lesion since erection may take place also when all the sacral roots are affected.

It is also important for segmental diagnosis in the conus that the feeling of fullness of the bladder and of the rectum may be still preserved at a lesion of the myelin substance in the lowest part of the conus (Kocher) while the motor centers of these organs do not function. Thus the sensory fibers of these organs enter the spinal cord at a higher level than that of the emergence of the motor fibers and therefore the conservation of the sensitivity of the bladder and of the rectum in existing paralysis of these organs speaks for the fact that the process which has destroyed the motor centers did not penetrate the myelin sheath of the higher segments.

Segmental diagnosis of other disorders of the vegetative nervous system in other divisions of the spinal cord are less easily analyzable than for those of the conus terminalis. From numerous investigations in degeneration (Biedl, Laignel-Lavastine, Huet, Anderson, Scaffidi, on the other side Lapinsky and Cassirer) it is held that a certain group of cells of the gray substance, namely, the cells of the

lateral horn, which are distinctly characterized by their *smallness, their coarse form,* their thick stratification, their position in a gelatin-like substance (N. sympathicus lateralis superior of Jacobsohn) are to be considered as synapses of efferent, automatic fibers; that perhaps in addition to this afferent fibers are ending about the cells of Clarke's column, just as small ones do about the groups of cells attached to the central canal (paramedial cells), also the substantia gelantinosa Rolando stands perhaps in connection with the fibers of the autonomic system (T. Sano). Concerning the closer functional importance of these groups of cells it is established only for the centrum ciliospinale, which sends off the sympathetic tract to the smooth musculature of the eye into the cervical sympathicus of the same side, that it is represented by the cells of the lateral horn in the transition between the cervical and the dorsal segments (Jacobsohn). The same or the neighboring groups of cells arranged round the central canal in the dorsal segments apparently control the innervation of the sweat glands and of the bloodvessels. This may be supposed in consequence of the observations upon the disturbances of these functions in lesions of the gray substance (Higier) these functions are not yet attributable to definite groups of cells. The central paths for the segmental centers of the spinal cord probably pass along the lateral column chiefly of the same side, although so far anatomists have not succeeded in delimiting them as isolated systems. It is known that disturbance of the innervation of the sweat glands and the vasomotors can arise on the same side when there are unilateral lesions of the spinal cord.

Oblongata Synapses of Vegetative Functions.—Here cardiovascular, respiratory, enteric (gastric) and metabolic functions are correlated. Complete phyletic analysis is still lacking although numerous researches have accumulated a mass of material that awaits the genius of an organizing intelligence to put it in form for the student. The study of the neurology of metabolism will form one of the largest chapters in the neurology of the future.

Among the cranial nerves arising from the rhombencephalon, clinicians are interested particularly in the vagus nerve, the disturbances of which show themselves above all in alteration of the heart rate. But neurological science it still insufficiently informed concerning the center of the fibers inhibiting the heart. It is known that the nucleus dorsalis vagi which lies directly under the floor of the ventricle has to be taken for the center of the efferent fibers to the intestines (Gaskell, Bruce, Marinesco, Kohnstamm and Wolfstein, etc.), and in regard to the localization in this nucleus it can be said that the smooth musculature of the intestinal tract is represented above all in its posterior part (Kosaka and Yagita, Vermeulen), the plexus pulmonalis in its anterior portion (Hudovernig).

It is in respect to the localization of the heart vagus that opinions still diverge. Some authors consider the nucleus dorsalis X as the center of the inhibiting fibers of the heart (Kohnstamm, L. R. Müller, Molhant). More recent researches on the contrary lead to the admis-

sion that this nucleus is to be sought in the depth of the medulla oblongata in the sphere of the nucleus ambiguus (Kosaka and Yagita, Sturmann). The pathological-anatomical results of the present time are also not uniform. Kato Toyoiro found changes in the nucleus dorsalis X in increase of brain pressure by tumors but at the same time also in the nucleus ambiguus and in the emerging roots of the X cranial nerve. Spiegel failed to find in cases of meningitis changes transgressing the normal limit in the nuclei of the vagus, and what is more, he found the inflammation of the meninges spreading over to the emerging roots of the nerve thus complicating the interpretations.

Yet there are cases known in the literature (Lemcke, Reinhold) where with slight lesions in the sphere of the nucleus dorsalis vagi a serious bradycardia, further, disturbances of the respiration, as a result the Adam-Stokes' and Cheyne-Stokes' symptom-complex occurred, so that one can by the appearance of these symptoms infer an involvement of the gray substance of the ventricle in existing bulbar disease. But it seems unjustified to refer these disturbances, in particular Cheyne-Stokes' respiration, to the dorsal vagus-nucleus. For in the present state of knowledge that nucleus, as far as it concerns the lungs, is to be considered only as the center of the efferent fibers for the smooth musculature of the trachea and of the bronchi. Nevertheless if affections in the central gray substance around the fourth ventricle lead to serious troubles in respiration these disturbances may be referred rather to a deficiency in regulation of respiration depending upon the afferent vagus fibers.

The sensory fibers of the vagus nerve, that is, those fibers which provide for the "self regulation of respiration" (Breuer-Hering) end in small cells laterally from the nucleus dorsalis vagi. Lesions of the gray substance of the ventricle may destroy the ends of these fibers themselves or may injure the tracts going out from the sensory nuclei of the vagus which are the intermediaries for the reflex influence of the segmental centers of the respiratory musculature. They may lead therefore to disturbances of respiration without a mystical respiration center to bear the responsibility.

The prognostic value of these disturbances of respiration is quite obvious. The attempt has been made, therefore, to locate the "vital knot" of the physiologist in this region. Therefore, clinically it is not unimportant in affections of the medulla oblongata to make sure whether the affection approaches the feared "center of respiration" or not. In certain apoplectiform bulbar paralyses, hemorrhage in the lateral field of the medulla oblongata in the sphere of the arteria cerebelli inferior-posterior is to be taken into consideration.

General vasomotor disturbances, reduction of blood-pressure, may not serve to answer this question as the assumption of a general vasomotor center on the floor of the fourth ventricle (Rheinhold) could not withstand the penetrating criticism of Cassirer and on the whole the existence of a general vasomotor center in the medulla oblongata is still under discussion. Claude Bernard's celebrated sugar puncture is

no longer explicable on the basis of a sugar center. The truth lies nearer to the fact that when certain pathways are interrupted by the puncture the glycosuria develops. The pathway from the medulla passes in the cord to the upper cervical segment, then by means of the C_7, anterior root, passes to the inferior cervical ganglion, then to the next sympathetic ganglia to the splanchnics, passing to the adrenals by means of the celiac ganglion. The action on the liver is partly mediated by this adrenal output. Midbrain and cortical synapses exist. Thus certain paralysis agitans cases with glycosuria show lesions in the periventricular hypothalamic nuclei. Lewy shows that this group of cells is intercalated in the pathway.[1] The so-called Haubenbundel may carry these fibers. The changes in the islands of Langerhans in the pancreas can result from the interference of the neuron pathways from the cortex to the pancreas.

A glycosuria excluding other causes, points rather to the fact that perhaps the sphere of the nucleus dorsalis vagi is affected. The clinical observations in regard to that question are ambiguous. Brugsch, Dresel, Lewy[2] have shown that reciprocally destruction of the vegetative oblongata nuclei can produce changes in the organ functions, and that extirpations of the organs, liver, kidney, suprarenal, pancreas, can produce retrograde degenerations in the cells of the oblongata nuclei.

To revert, Marburg and Breuer have made it apparent that the sympathetic tract for the eye passes through the dorsal sphere of the substantia reticularis so that sympathetic ophthalmoplegia leads us to suppose that a lesion approaches the floor of the ventricle. Just as little is known concerning the more exact course of the sympathetic paths in the medulla oblongata as of the spinal cord. It is only known that in disturbances that concern the substantia reticularis, symptoms may occur on the part of the bloodvessels, of the sweat glands and of the smooth musculature of the eye, that the symptoms of the eye appears homolaterally while the sudoral and vasomotor disturbances of the bloodvessels are observed contralaterally in the extremities in lesions which are cerebral from the pyramidal decussation, and in the face homolaterally. For the vegetative fibers for the head cross directly behind the thalamus (cf. Marburg and Breuer), for the extremities at the height of the pyramidal crossing (Schlesinger, Karplus) or somewhat cerebral from it (Depisch). The independence of the vegetative pathways from the pyramidal pathways is at all events proved by the fact that unilateral symptoms of the vasomotors symptoms of irritation or paralyses, may occur as affections in the oblongata independently of disturbances of voluntary motility; the symptoms following Babinski, may be termed thermo-asymmetry and vaso-asymmetry.

[1] Dresel u. Lewy: Zeit. f. d. Exp. Med., 1922, **26**, 99.

[2] Beiträge zur Stoffwechselsneurologie. I. Ztschr. f. exp. Path. u. Therapie, 1920, **21**, 353. II. Ztschr. f. d. g. exper. Mediz., 1921, **25**, 262. Dresel, K.: Experimentelle Untersuchungen zur Anatomie und Physiologie der peripheren und zentralen vegetativen Neurvensystems Ztschr. f. d. g. exp. Med., 1923, **37**, 374. Lit. to 1923.

Pontine Synapses and Localizations.—For the localization in the region of the pons in the field of the vegetative nervous system, symptoms of the salivary glands are chiefly to be taken into consideration, the center of which is to be sought in scattered cells of the substantia reticularis. In particular cells of the formatio reticularis at the level of the seventh nucleus N. salivatorius superior of Kohnstamm, also of Yagita and Hayama, are considered as centers of the submaxillary gland, while cells between the inferior olive and the nucleus ambiguus (N. salvatorius inferior) (Kohnstamm) or groups of cells lying in the direct caudal continuation of the submaxillary center (Yagita and Hayama) are considered as the center of the parotid gland. Clinical observations (J. Fischl, Spiller) and recently also a more exact anatomically investigated case of Feiling show that apparently conditions are similar in man. In considering the anomalies of the salivary secretion in pontine, respectively bulbar affections, it is certainly necessary to exclude first the idea that an existing salivation is only a consequence of a paralysis of the mouth musculature existing at the same time before considering it as a sign of a lesion of the cells of origin or of the emerging fibers of the nerves of the gland.

Knowledge concerning the influence of the cerebellum on the vegetative nervous system is too little assured to be diagnostically of value.

Midbrain and Higher Synapses of Vegetative Pathways.[1]—These are being more definitely charted as time goes on. Grevings studies are here fundamental.[1] Edinger, Jacobsohn, Malone and others gave the earlier more specific histological evidence pointing to wide distributions of vegetative cells in the corpora striata, the hypothalamus, corpus Luysii, the thalamus and other interbrain structures. In the hypothalamic region are centrals for the general regulation of metabolic unctions of the body. Thus bodily temperature,[2] water and salt content of the body, osmotic pressure, blood-pressure, sleep and such general physiological functions have central prepresentation in these interbrain structures. Humoral regulation has central neural localization. In this sense a synthesis of the metabolic organs is effected through these interbrain structures (Greving). Here important connections are effected between the bodily organs and the psyche. Affective control is largely disturbed from lesions in these structures, as the study of epidemic encephalitis and of paresis show.

Exact localizations of important links in the chain of metabolic functioning are becoming more and more evident, though still imperfectly analyzed. Thus destruction of the corpora mammillaria causes polyuria. Controls for heat regulation are localizable in the globus pallidus and also in the tuber cinereum; of the lateral area of the gray matter on the floor of the midbrain, dilatation of the pupil

[1] Greving, R.: Zur Anatomie, Physiologie und Pathologie der vegetativen zentren im Zwischenhirn, Ztschr. f. d. g. Anatomie, 1922, **24**, 348. Lage und Tätigkeit der vegetativen zentren im Zwischenhirn, Ztschr. f. d. g. N. u. P., 1923, **83**, 22; Greving: Beiträge zur Anatomie d. Zwischenhirn und seiner Funktion 1-iv: Ztschr. f. d. g. Neur. u. Pschy., 1923, **90**, 231 (lit). Foix et Nicolesco, loc. cit.

[2] See Tonniessin: Veg. N. S. u. Warmerg. u. Stoffwechsels, Klin. Wchnschr. z. 11 and 12, 1923, pp. 477–525.

and widening of the lid slit. Stimulation of the ventricular floor in the region of the hypophysis brings about contraction of the bladder, the intestines and uterus, while stimulation of the tuber cinereum produces dyspnea and arrest of the heart's action, followed by bradycardia. Lesions of the floor of the midbrain induce marked metabolic and trophic changes in young and adult animals (genital dysplasia of the

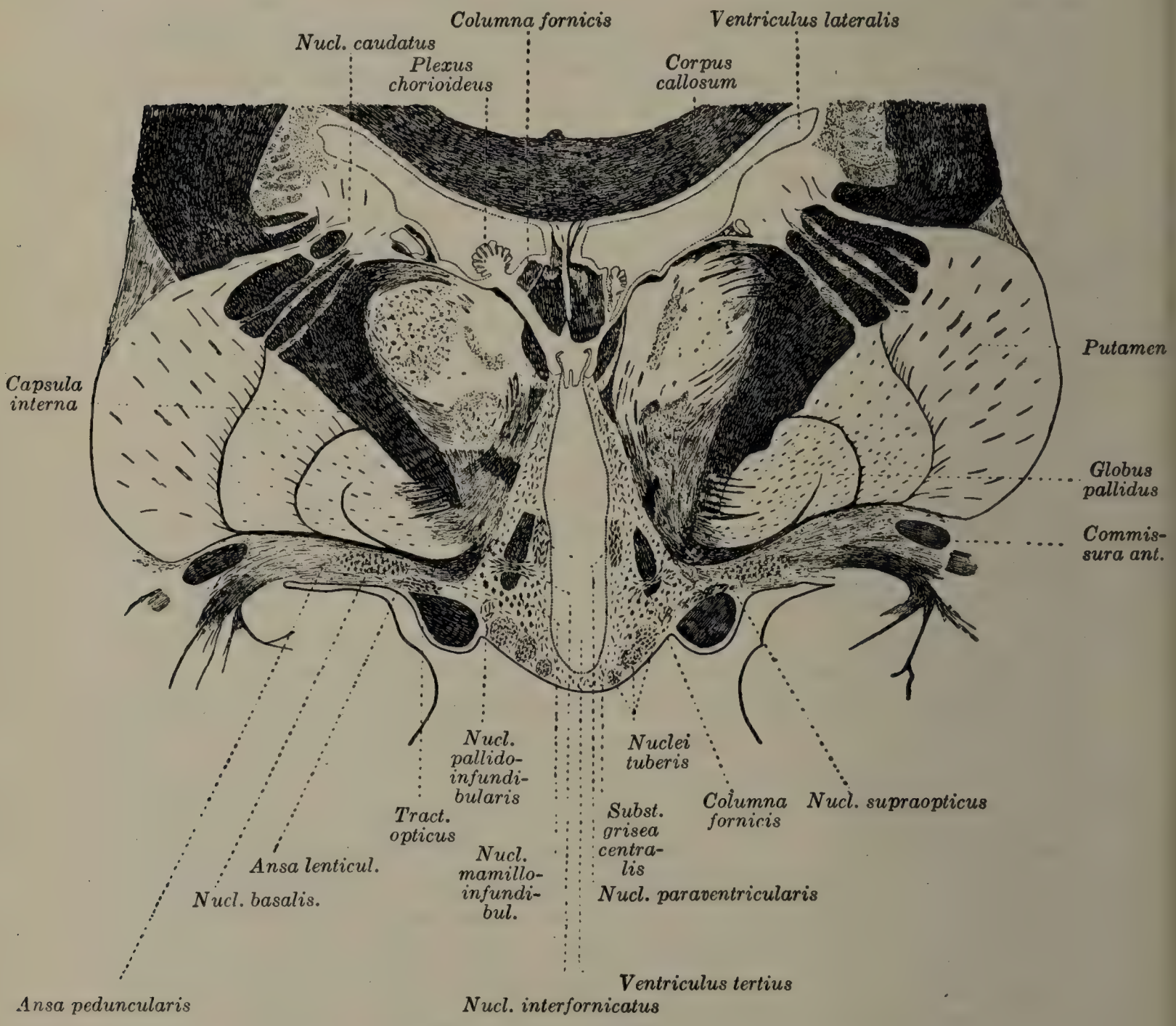


FIG. 47.—Frontal section through the tuber cinereum with schematically indicated cell groups in the hypothalamic region and the walls of the third ventricle (Greving). Compare pictures of Foix and Nicolesco.

ovaries and testes, retardation of growth) and puncture of the floor of the third ventricle causes intense glycosuria similar to the Claude Bernard puncture of the floor of the fourth ventricle. Simultaneous section of the distal ends of the sympathetic (NN splanchnii) prevents the occurrence of this glycosuria of hypothalamic origin as well as any glycosuria which may be caused by stimulation proximal to the place of section. Careful study of the vegetative disturbances in

paralysis agitans point in the same direction, while we are disposed to see in the extensive changes in the lenticular nucleus, so largely in evidence in Wilson's disease, and related syndromes direct evidence of impairment of vegetative pathways to and from the liver. What relationship, other than purely suggestive by analogy, the chromaffin-like substances of the substantia nigra have with other chromaffin structures is not known.

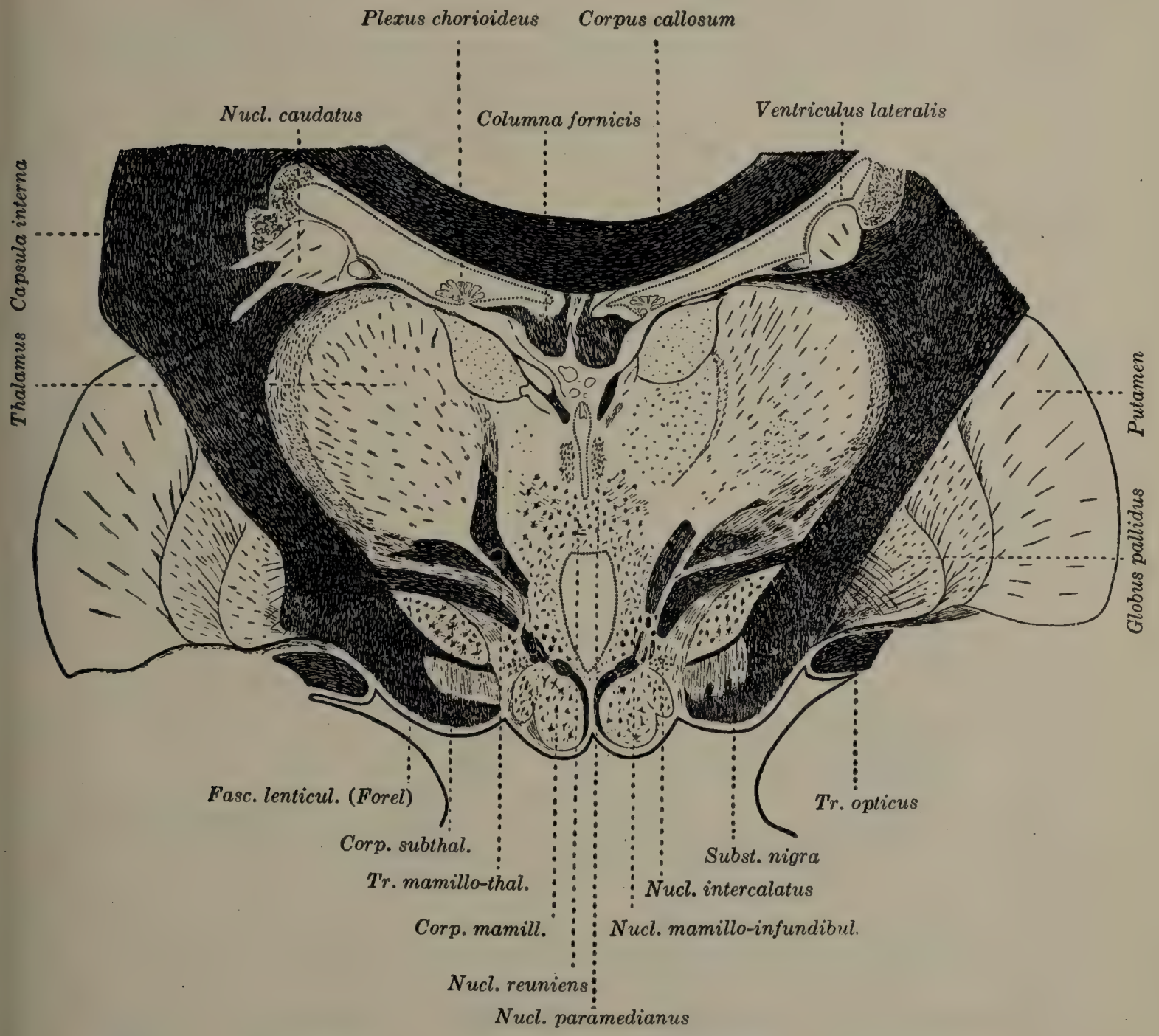


FIG. 48.—Frontal section through the corpora mammillaria with schematically indicated cell groups in the hypothalamus and in the walls of the third ventricle (Greving). Compare with Foix and Nicolesco.

Clinical Correlations.—As regards the midbrain, neurological science is interested in how far disturbances of the pupillary reflex are applicable for local diagnosis. Karplus and Kreidl have shown that the afferent fibers of the reflex arc pass over the brachia of the anterior corpora quadrigemina since section through the brachia leads to an isolated loss of the pupillary reflex to light. The further course of the reflex arc is not known.

Zweig and Spiegel have thought that the center of the fibers of the pupil is at least partly in the nucleus medius anterior but it is not impossible that also the anterior portions of the large-celled chief nucleus of the oculomotorius participate in the innervation of the pupil. It is in accord with this that it must be assumed the pupillary fibers have their course in the most anterior portions of the oculomotorius bundle as it passes out. For the local diagnosis in the midbrain loss of pupillary reflex in a process of the midbrain when other causes are excluded points to the fact that the most anterior portions of the midbrain are affected on their passage to the thalamus opticus.

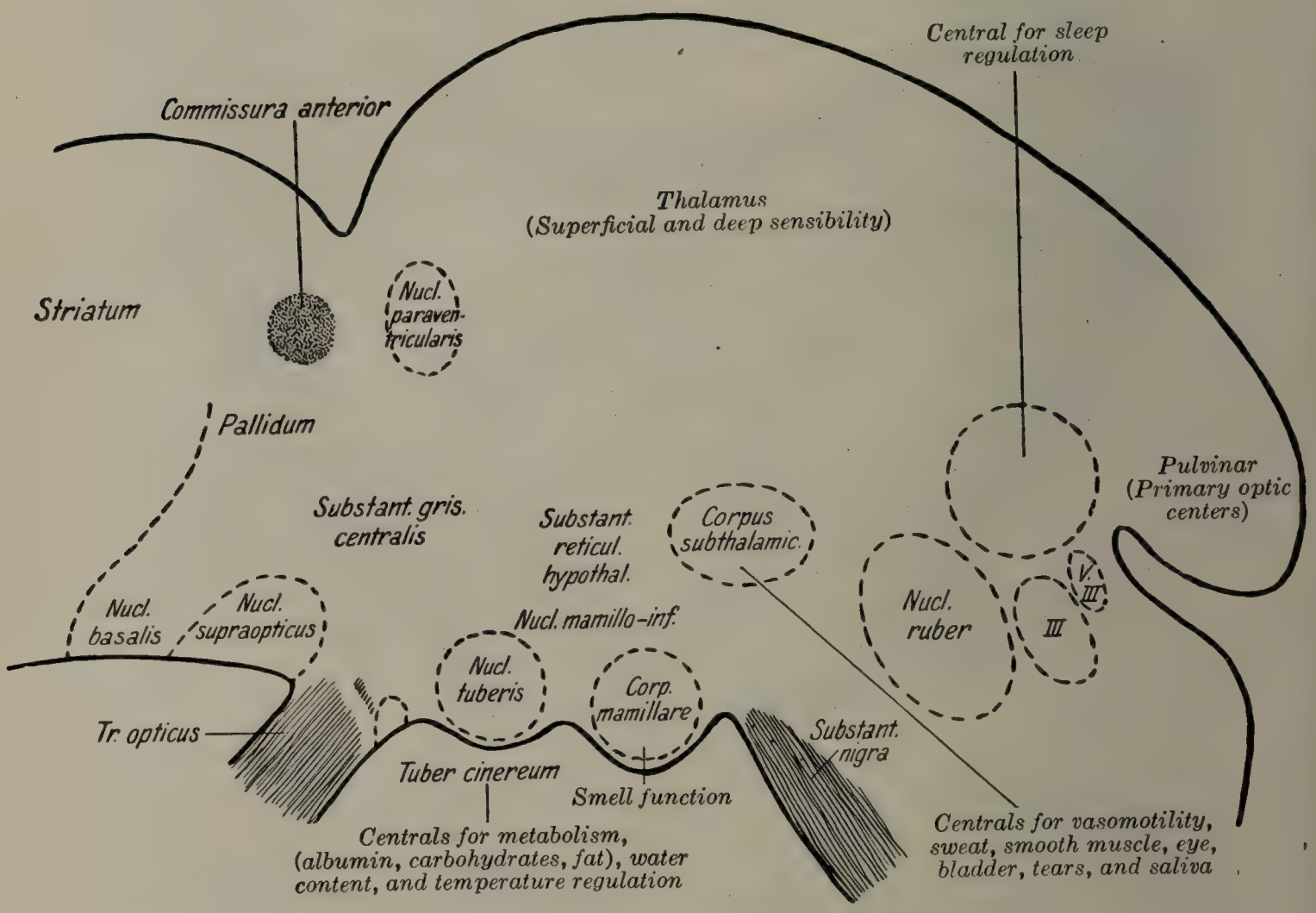


FIG. 49.—Schematic representation of the vegetative cell groups in the interbrain and their function (sagittal section (Greving)).

Concerning the interbrain attention was first directed to the hypothalamic region by experiments of Karplus and Kreidl. Both authors showed in different animals that an irritation behind the tractus opticus lateral from the infundibulum causes dilation of the pupil on both sides, and an enlargement of the palpebral fissure, an effect that persisted also after degeneration of the tract descending from the cortex, after section through the adjacent cranial nerves as after severance of all connections with the spot of irritation with the exception of that of the pedunculus. An irritation of the caudal half of a frontal section through the thalamus showed that this effect had been produced by irritation of the corpus subthalamicum. Secretion of perspiration and of tears as well as a

contraction of the bloodvessels could be proved on irritation of this center together with the symptoms of the eye already described. The corpus subthalamicum also seems to effect at least partly the sympathetic reflex of the eyes following pain stimulus, as after section through the brain caudal from the hypothalamus as a result of stimulus of the nervus ischiadicus dilation of the pupil was absent.

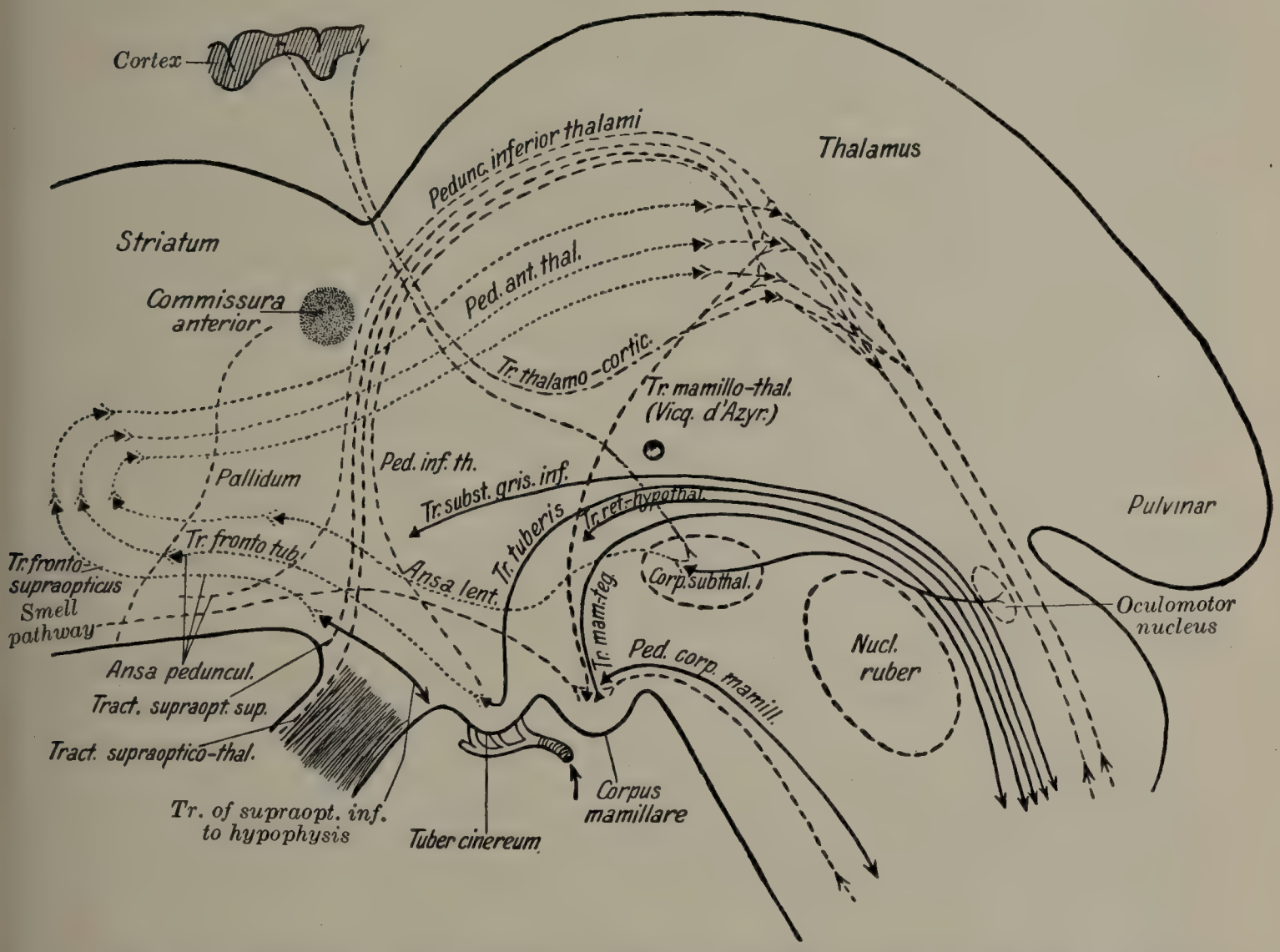


FIG. 50.—Regulatory mechanism of vegetative centers. A preliminary attempt at a schematic representation of the vegetative reflex pathways in the interbrain. (Greving.)

Clinical observations of symptoms of the center of Karplus-Kreidl are very rare. Gerstmann found with a gunshot wound of the right temple a left-sided increase of the tendon reflex, Babinski's sign, hypoesthesia and hyperalgesia combined with Horner's syndrome of the right side, which he referred to a lesion of the right corpus subthalamicum. Schrottenbach's case showed a left-sided motor and sensory paralysis, hemianopsia and conjugated paralysis of sight toward the same side, disturbance of secretion in the face and in the left eye, mimic palsy of the left half of the face, symptoms that we were able to explain by a lesion of the region of the right thalamus opticus, of the adjacent hemispheres and of the pyramidal tracts. Absence of the sympathetic pupillary reflex to pain could also be established similarly as in the experiments of Karplus and Kreidl, so that one may think of

participation on the part of the subthalamic center of the sympathicus. But as long as there are no cases investigated anatomically, the diagnosis of a lesion in the center of Karplus-Kreidl will be only a supposition. For the diagnosis may be based only on the combination of symptoms of the thalamus and the neighboring capsula interna with disturbance of the innervation of the smooth musculature of the eye, of the sweat glands and of the bloodvessels. These symptoms of failure of the autonomic system may be caused as well by injury to the cortical fibers which go down into the inner capsule to the center of Karplus-Kreidl as by a lesion of the corpus subthalamicum. It is known that with a hemiplegia, vasomotor disturbance (Oppenheim, Parhon and Goldstein, Ingelrans) may occur as anomalies of the sweat secretion (Pandi, Cassirer), sympathetic ophthalmoplegia (Seeligmüller, Prevost, Nothnagel, Klippel and Weil) symptoms that appear at affections cerebral from the thalamus contralaterally from the lesion (Marburg and Breuer). Even if it can be shown that an affection to be assumed in the region of the thalamus opticus annihilates the reflex dilation of the pupil to pain stimulus it cannot yet be stated with certainty that interruption of the reflex follows in the corpus subthalamicum itself. It could be that the fibers leading to that center or radiating from it may be injured as well.

One has to be very cautious as yet in referring bladder disturbances to thalamus lesions. The researches of Lichtenstern make it probable that the bladder contractions observed by the older authors (Nussbaum Bechterew and Mislawsky, Ott and Wood, Field) from irritation of the thalamus stand in connection with a hypothalamic sympathetic center. But there are no clear cases observed which would give one the right to refer with certainty the clinically observed bladder disturbances to the thalamus opticus. Bladder symptoms in cerebellar disturbances are often quite striking.[1]

There is more material on the other hand as to the connection of the midbrain with the kidney secretion.[2] Originally diabetes insipidus from lesion at the base of the skull was connected with the hypophysis, at first hyperfunction, later a hypofunction of the posterior lobe of this gland has been assumed (Frank, Goldzeiher, Simmonds). But cases occurred where a disappearance of polyuria was observed with enlargement of a tumor at the base of the skull. On the other hand even the total destruction of this gland occurred without polyuria. Therefore it has been thought that the activity of the preserved anterior lobe which seems to produce diuretic substances (Cushing) is necessary for the origin of the polyuria (Fleckseder, Hann). But it is was necessary to extend this explanation also as diabetes insipidus was observed with a hypophysis histologically intact. Fleckseder had already thought of a participation of the infundibulum and was able to show that indeed

[1] Bakker: Ztschr. f. d. N. P. 1924, vol. **89**, p. 213. Other vegetative cerebellar organizations are probable. Papilion: Jour. d. Path. Gen., 1926, No. 1.

[2] Stahl and Schute: Einfluss d. V. N. S. auf d. Nieren. Zeit. f. d. exp. Med., 1923, **35**, 312.

it did share in the changes in a series of cases. Recently Leschke especially has gone further trying to prove that destruction or atrophy of the entire hypophysis or of one of its lobes cannot lead to diabetes insipidus. An isolated lesion of the posterior lobe, according to him, proceeds without any symptoms in animals and in man, while a destruction of the anterior lobe leads to hypophysial cachexia. On the other hand polyuria arises after puncture of the base of the midbrain closely behind the infundibulum so that he is inclined to refer diabetes insipidus to functional disturbances of the midbrain. It seems questionable if one is permitted to go so far yet at the present time in the denial of the hypophysial genesis of diabetes insipidus. For in a part of the cases of the literature collected by Lechske secretory disturbances of the kidney are not noted. In most of them histological research of the hypophysis is lacking but a disturbance of the function of this gland may not be excluded summarily even when it is completely intact. Since the gland is innervated from the infundibulum lesion of this region leads probably to the symptoms of diabetes insipidus merely by a disturbance of the innervation of the hypophysis. Therefore one is merely able to say that the appearance of diabetes insipidus from a lesion supposed to be at the base of the skull is certainly of great importance as giving support to this diagnosis but that it is impossible to establish a more exact local diagnosis, whether this lesion concerns the hypophysis or the infundibulum, by the symptom of diabetes insipidus alone.

Lesions within the corpora striata can cause vegetative symptoms. The researches of Brugsch, Dresel and Lewy (loc. cit.) have shown this. Jelliffe suggested these early with reference to the relationships of Wilson's syndrome and the liver changes. Lewy has demonstrated this. The great constancy of the striatum (= nucleus caudatus + putamen) in the mammalian series, has led to the supposition that here is a ganglion which preëminently controls autonomic innervation (cf. Spiegel). In support of this view Spiegel mentions that the globus pallidus where the fibers end that arise in the nucleus caudatus and the putamen is in relation with the corpus hypothalamicum, thus with the sympathetic center of Karplus-Kreidl.

Thermal regulation is one of the most intricate of the body's neural mechanisms.[1]

Cortical Stations of the Vegetative Pathways.—There are many discordant observations concerning cortical stations in the vegetative pathways. Our conclusion from the many studies is that there are such, and it is here maintained, that every organ of the body has cortical representation. The evidence cannot be discussed here very fully as it transcends the limits of a text-book.[2] Much light on this question will

[1] Greving: Die pathogenese des Fiebers. u. s. w. Deut. med. Wchnschr., 1922, **50**, **51**, 1673. Isenschmid: Ueber das Wesen und Bedeutung des Fieber-Schw. med. Wchnschr., 1925, **55**, 1173.

[2] See studies already cited by L. R. Müller, Greving, Dresel, Laignel-Lavastine, Lewy, Glaser, v. Economo, Schilff, and others.

undoubtedly be thrown upon the problem when the cortical scleroses so abundantly demonstrated by Southard in schizophrenia, for example, are histologically correlated with the vegetative syndromy inclusive of the endocrinopathies, equally manifest in this heterogeneous group.

Higier in treating this question raises the following problems: (1) the location of the sympathetic centers, *i. e.*, the psycho-vegetative centers in the cortex analogous to psycho-motor centers, (2) the relationship of mental states upon the tone of the vegetative nervous system, (3) emotional states and vegetative activities. Kempf (loc. cit.) has discussed this latter aspect very exhaustively.

Have internal and external visceral organs cortical representation like voluntary muscles and the sensory functions of the skin? Higier calls attention to the relations of the vagus in this connection, since its anatomical relations, its viscero-bulbo-thalamo-cortical parts, have been very carefully studied. The vagus is a mixed nerve having at least three nuclei whose embryological development is different. The motor nucleus ambiguus corresponds to the anterior horn, the sensory nucleus solitarius to the posterior horn and the visceral nucleus dorsalis vagi to the vegetative nerve cell group which lies in the lateral gray matter of the spinal cord. The sensory fibers supply the mucous membranes of several cervical and thoracic organs—and they continue upward through the thalamus to the cerebral cortex. It is also known that there are cortical synapses for the fibers to the voluntary muscles of the pharynx and larynx. These exist whether such came at a period when these muscles became striated or whether they existed when they were *still involuntary, unstriated* muscles. The phylogenetic relations do not alter the facts. A cortical representation of the visceral part of the vagus, of the cardiac, bronchial, pulmonary and gastric fibers of the vagus has become clearly demonstrated. The same question of course arises in connection with the other autonomic and sympathetic fibers which have to do with the excretory and secretory organs of the skin and body cavities. There are now two quite opposite views upon this subject. One school is headed by Bechterew,[1] the other by Pavlof[2] and L. R. Müller.[3]

Bechterew's school tried to arrive at a conclusion by investigating the associated or conditioned reflexes. Associated reflexes according to this school consist in a reaction which is not determined by either the strength or the quality of the external stimulus, but by the relation of this stimulus to other reflexes in the past, *i. e.*, a relation that has been often repeated and whose repetition occurs in the same manner as the reactions produced by habit. The hissing of a snake produces an associative reflex which manifests itself in an intense motor reaction not because of the loudness or softness of the noise, not on account of the hissing itself, but because the hissing is associated with danger. The presence of a cat causes a dog to run because of past experiences

[1] Bechterew: Reflexologie, Berlin, 1926.
[2] Pavlof: Conditioned Reflexes, Tr. by Anrep, Oxford, 1927.
[3] Müller, L. R.: Die Lebensnerven, loc. cir.

with the cat's claws. The smell and sight of appetizing food starts gastric secretion. Since vegetative functions are concerned with the most vital of body processes, processes which are activated by simple reflexes, extirpation of the cortex does not interfere with their activity, does not cause any paralysis as extirpation of the motor cortex supplying the extremities. But there does result a great defect of nerve activity in so far as associative reflexes go.

If one removes in the dog what Bechterew regards as the cortex representing respiratory activities and remove both centers, respiration proceeds normally due to the presence of the spinal, the coordinating medullary and the subcortical midbrain centers. The respiratory reflex to artificial as well as normal sensory stimuli is the same as before. But the *psycho-reflexes*, the associative reflexes, are modified. The presence of a cat does not cause the intense motor effect upon respiration which occurred in the unoperated dog under like circumstances.

If synaptic junctions of sex-organ pathways, which Bechterew locates in the upper third of the gyrus postcruciatus be removed in a dog, the erection of the penis to mechanical stimulation obtains, but the psycho-reflexes related to the presence of a bitch in heat are absent. If the synapses of iris pathways in the frontal lobes be removed, certain symbols, as threatening with a stick or whip are not responded to by dilatation of the pupil. Thus Bechterew has shown that many symbolic conditioned reflexes are absent following cortical injury, and concluded that visceral tracts, motor and secretory, have cortical representation. Pavlov on the contrary reached conclusions which are diametrically opposed to those of Bechterew.

The enormous material now being accumulated from the war is tending to show more and more the validity of Bechterew's contentions and the results of histological examination of the brains in severe affect disturbances in the psychoses, notably dementia precox, paresis, and senile arteriosclerotic changes are also tending in the same direction. These cannot be entered into fully here. Only a few of the important findings can be mentioned. The student is referred to the rich literature which has been recorded since the war.[1]

Spiegel has called attention to the fact that in lesions of the central nervous system disturbances in function in the sphere of the vegetative nervous system in general will be the more inconstant the more neurons are interpolated between the affected ganglion and the peripheral organ. In respect to the cortex it may be recalled that muscles acting bilaterally symmetrically are generally represented bilaterally (Broadbent) so that with a lesion of one hemisphere the corresponding center of the other side becomes substituting and lasting disturbances are observed only at bilateral affections. Marburg and Czyhlarz showed the importance of Broadbent's law for the innervation of the bladder. Their observations, which led to the establishment of a center for the

[1] Goldstein, K.: München. med. Wchnschr., 1917, **68**, 1249; 1918, **69**, 104; also later studies in Ztschr. f. d. g. Neur. u. Pschy.; Spiegel, loc. cit.

bladder in the motor region between the center for the arm and that for the leg have since found frequent confirmation (Friedmann, Ed. Müller, Pfeifer). It was necessary, to be sure, to extend them after the experiences of the war in such a way that besides the center mentioned by them a second center is to be assumed in the region of the paracentral lobe in the neighborhood of the foot region (Bl_2). (Förster, Kleist, A. Alder). The importance of both these centers may be summarized very simply by saying that the region joining the center for the hip controls the discharge of urine, corresponding to the irritation of the Nn. pelvici, the field in the neighborhood of the center for the leg controls its retention, corresponding to the irritation of the Nn. hypogastrici. The failure of the first center is followed by retention, the destruction of the second by incontinence, while an irritation of these centers acts naturally but in the opposite direction.

In contrast to the transitory character of the functional disturbances in unilateral lesions, irritation symptoms of the cortex gain clinical importance. They are of diagnostic value chiefly in localized circumscribing processes in the cranial cavity in which a definite course of symptoms of irritation, a Jacksonian epilepsy, results from pressure upon the circumscribed portion of the cortex. As is well known, epileptic attack begins in that part of the body that corresponds to the directly affected part of the brain and spreads from here over the other regions of the body corresponding to their cortical localization. Therefore initial symptoms of the Jacksonian fit have a great value for local diagnosis. They point to that part of the brain that is directly exposed to the irritation. There are at hand observations of cases in which symptoms of irritation began in certain parts of the vegetative sphere and it has been possible, in fact, to establish circumscribed affections of the cortex. Oppenheim mentions vasomotor forms of Jacksonian epilepsy. Mayer observed a patient in whom a diminution of temperature was stated in the left upper extremity during convulsive attacks that were caused by a gumma of the right motor region. Emminghaus describes epileptic perspiration of the face. Senator convulsions and profuse perspiration on one arm localized in abscess in the opposite motor region. Through an observation of Korány of epileptic salivation it may be supposed that salivary secretion is influenced by a region situated near Broca's center.

It is still in discussion whether the vegetative organs possess also circumscribed cortical territories analogous to the voluntary innervated portions of the body, yet attack-like appearance of circumscribed vasomotor and secretory symptoms of irritation on the extremities, such as those observed in the Jacksonian fit, deserves the attention of the physician. It points to an accompanying irritation of the motor region.

The observations of Goldstein, loc. cit., would tend to show a large number of intricate and interesting correlations between cortical injury and vegetative disturbances. Among these attention can be called to alterations in carbohydrate metabolism, to vasomotor control, modifi-

PLATE IV

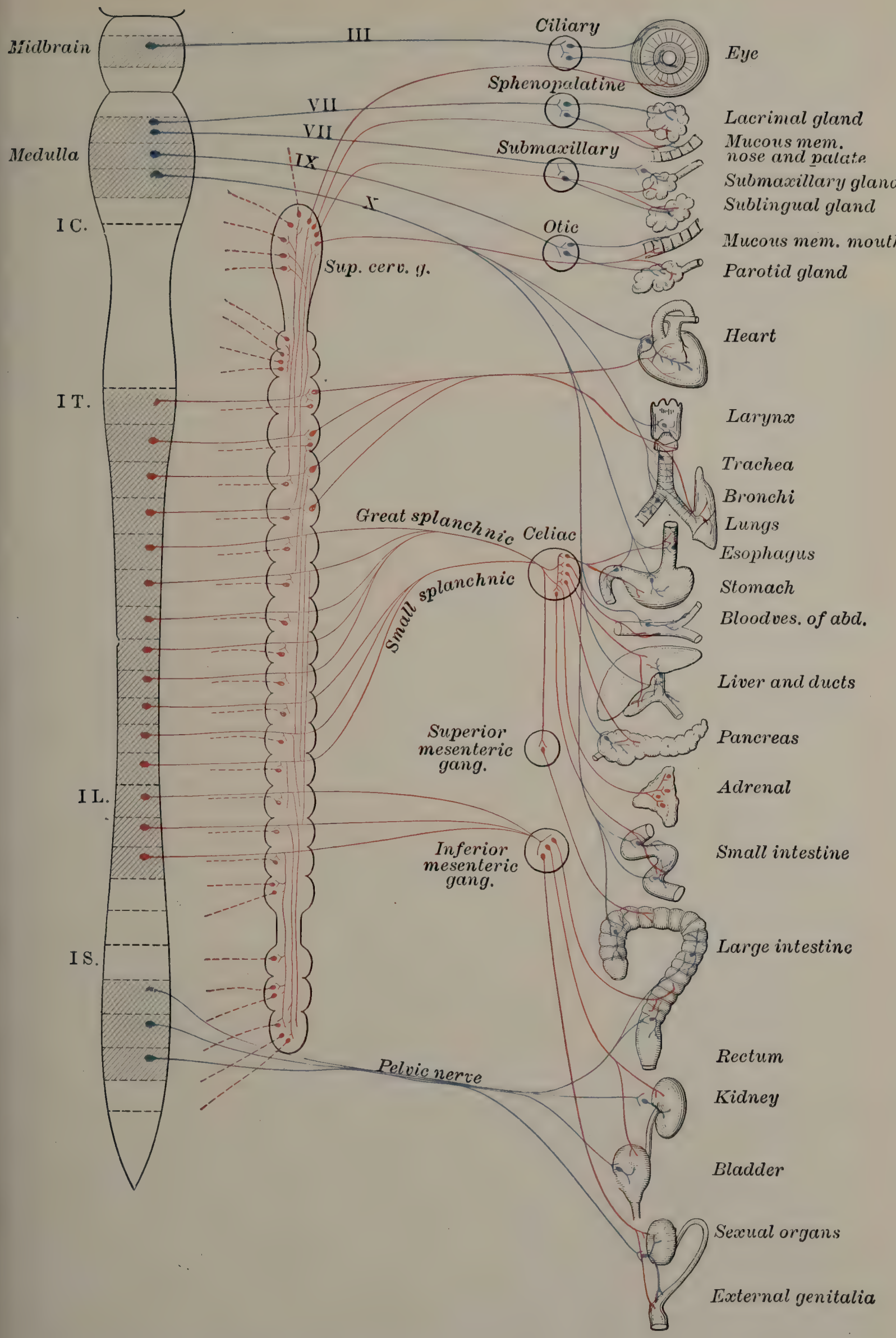


Diagram of Spinal Efferent Autonomic Nervous System. (Modified after Meyer and Gottlieb.)

Blue, cranial and sacral outflow. Red, thoraco-humeral outflow. ------, Postganglionic fibers to spinal and cranial nerves to supply vasomotors to head, trunk and limbs, motor fibers to smooth muscles of skin and fibers to sweat glands. (Local organ efferent cells are universal.)

cations of the blood picture, of blood-pressure, of temperature alterations, sweating anomalies, localized cyanosis, localized edema, etc.[1]

In fact a large number of bodily manifestations of psychic occurrences are so constant in the vegetative domain that they have been used as precise measures in psycho-physiological studies. Thus the following reactions have been used: (*a*) Measurement of the pupil (Bumke), (*b*) measurement of the activity of the salivary glands (Pavlov), (*c*) measurement of the involuntary activity of the bladder (Mosso), (*d*) measurement of the skin resistance during various degrees of sweating (Veraguth's psycho-galvanic reflex phenomenon), (*e*) measurement of the activity of the vasomotors and the heart (Mosso-Lehmann plethysmographic test of blood distribution). A large number are being added to these with the more intense study of the endocrinopathies associated with emotional disturbances.[2]

Provisionally it may be stated that it is the conviction of the authors, based upon an extensive series of dream analyses, that various organs of the body are symbolized in the dream work. It is believed that the dream plays an important role in endeavoring to bring under some form of conscious attention disease processes occurring in various organs of the body.[3]

Sympathetic and Parasympathetic Divisions.[4]—Anatomically, as well as pharmacologically, it appears that two opposing types of physiological activity are operative in the vegetative nervous system. These have been termed the sympathetic and the parasympathetic or autonomic. All those non-voluntarily influenced organs, smooth muscle structures, heart muscle, glands, etc., whose nerve fibers are related to the spinal cord from the first dorsal above to the fourth lumbar segment below, belong in the sympathetic system in the narrower sense. All other function for those activities which have been termed the parasympathetic or autonomic. The uppermost are integrated in the midbrain structures. They enter the ciliary ganglion, and are distributed to the smooth internal muscles of the eye. A second, or bulbar parasympathetic system, utilizes the facial and goes as the chorda tympani to the salivary glands. The glossopharyngeus and vagus belong to this bulbar parasympathetic system. A sacral parasympathetic system functions for the organs of the pelvis and genitalia. (See Plate IV.)

How this division will prove out in clinical work is yet to be tested, but it has become a practical working hypothesis to harmonize the results of pharmacological experimentation.[5] This is particularly to be seen in the reactions of the vegetative nervous system to certain

[1] Cerebellar localizations. See Bakker's paper, Ztschr. Neur. u. Psych., vol. **89**; and Papilion, loc. cit.

[2] See Higier: Loc. cit., p. 125. Laignel-Lavastine: Loc. cit. Kempf: Loc. cit.

[3] Jelliffe, S. E.: Paleopsychology. A Study of the Evolution of Symbolic Function, Psychoanalytic Review, January, 1923. Schilder: Das Körperschema, 1923.

[4] Frank, E.: Ueber die gegenwärtige Stand der Lehre von der Vagotonie u. Sympathicotonie, Deutsch. med. Wchnschr., February 10, 1921.

[5] Petren u. Thorling: Ztschr. f. innere Med., 173. Bauer, D.: Arch. f. klin. Med., 107.

products of the endocrinous glands, *i. e.*, hormones, and to certain toxic and anaphylactic substances, notably nicotin. Inasmuch as the vagus constitutes the chief representative of the parasympathetic system, the terminology is applied to it more particularly.

Thus atropin, its related alkaloids and the nitrates paralyze the vagus and its end-organs (vagoparalytic). The former paralyzes the positive or stimulating element of the parasympathetic system, causing mydriasis, lessened secretion; the latter a paralysis of the negative or depressing element, gives rise to vasodilatation. Vasospastic drugs, such as muscarin, pilocarpin, picrotoxin and physostigmin cause lowering of blood-pressure, weakening of the heart action, bradycardia, myosis, increased secretions, and increased peristalsis. Morphin gives similar reactions but its actions are extremely complicated.[1]

A number of the products of the endocrine glands act as vagotonics; among these is cholin, from the cortex of the suprarenals. The parasympathetic stimulation symptoms of cholin are myosis, diminished peristalsis, contraction of the uterus, bladder, bronchi, diminished cardiac force and rhythm, pallor of skin with increased, cold sweat and paresis of the abdominal bloodvessels. The sympathetic receptors seem to be unaffected by cholin. Iodothyrin, hypophysin, and adrenalin act partly as sympathicotonic, partly as autonomic or vagotonic. Iodothyrin, or the related hormone from the thyroid, acts upon both systems to produce some of the characteristic signs of exophthalmic goiter (see later).

The active principles of the hypophysis acts upon both systems to produce pictures of hypo- or hyperpituitarism or a mixture of both, dyspituitarism. Adrenalin, from the medullary portion of the adrenal, causes tachycardia, increase of blood-pressure from contraction of the bloodvessels, mydriasis and exophthalmos, paresis and anemia of the respiratory and stomach tracts, increase of sphincter tonus and of the secretion of certain glands, mobilization of glycogen, and increase in its oxidation. The amount of adrenalin set free by acute, conscious fear has been measured by Cannon; the effects of chronic, unconscious fear are undoubtedly more forceful. The antagonistic action of the sympathicotropic adrenalin and the vagotropic pilocarpin shows itself in that adrenalin can counteract a pilocarpin eosinophilia and pilocarpin an adrenal glycosuria. Other remarkable opposing reactions are known showing the striking antagonistic physiological possibilities of the vegetative system.

The chief contrasting activities of these two systems are here shown in tabular form as taken from the studies of Fröhlich, Eppinger, Hess, Löwi and others (see page 175), but it must be emphasized that increasing experience is showing that a cut and dried type of reaction is not in accordance with the facts. All that can be maintained is that an intense reaction to pharmacodynamic agents can be looked upon as a vegetative strain, and only when an intense and isolated reaction occurs in a definite organ or organ system with only a weak corre-

[1] Kraus: Jour. Nerv. and Ment. Dis., 1918.

COMPARISON OF ANTAGONISTIC ACTIONS OF SYMPATHETIC AND AUTONOMIC SYSTEMS.

Result of stimulation of the sympathetic system.	Action of		Organ.	Action of		Result of stimulation of autonomic system.
	Atropin.	Adrenalin.		Pilocarpin.	Ergotoxin.	
Stimulation Th. I–II	Paralysis	Stimulation	Sphincter iridis Dilator iridis	Stimulation		Stimulation N. III.
Stimulation Th. I–III	Paralysis	Stimulation	Ciliary muscle Orbital muscle	Stimulation Paralysis	Paralysis	Stimulation N. III.
Stimulation Th. II–IV	Paralysis	Stimulation ı ? Constriction	Salivary glands Cortical bloodvessels	Stimulation		Ch. tympani. secretion. Dilatation N. X.
Constriction Th. II–IV	Constriction?	Dilatation Constriction	Buccal bloodvessels Skin bloodvessels, head region			Constriction N. IX.
Constriction Th. II–IVL	Dilatation	Dilatation Constriction	Coronary bloodvessels Intestinal bloodvessels	Constriction	Dilatation	
Constriction L. I–IV		Constriction	Genital bloodvessels			Dilatation N. pelvic.
Stimulation Th. II–L. IV	Inhibition	Inhibition	Sweat glands	Stimulation		
Stimulation Th. IV–VII		Stimulation	Pilomotor muscles of the face		Paralysis	
Stimulation Th. I–V	Stimulation	Stimulation	Heart muscle	Inhibition	Inhibition	Inhibition N. X.
Relaxation Th. II–V	Relaxation	Relaxation	Esophagus	Stimulation		Stimulation N. X.
Paralysis Th. II–L. IV	Paralysis	Paralysis	Cardia	Stimulation		Stimulation N. X.
Diminished Th. II–L. IV	Diminished		Gastric tone	Increase		Increases N. X.
Paralysis Th. II–L. IV	Paralysis	Paralysis	Gastric peristalsis	Increase		Increases N. X.
Diminished ?	Diminished	Diminished ?	Gastric secretion	Increase		Increases N. X.
Inhibition Th. II–L. IV	Paralysis	Paralysis	Small intestine peristalsis	Stimulation		Stimulation N. X.
Relaxation L. I–IV	Relaxation	Relaxation	Colon	Stimulation		Stimulation N. pelvicus.
Relaxation L. I–IV	Relaxation	Relaxation	Sphincter ani (muscle)	Spasm		Spasm N. pelvicus.
Relaxation Th. II–L. IV	Relaxation	Relaxation	Gall-bladder	Contraction		Contraction N. X.
Inhibition ?	Inhibition	Inhibition	Pancreatic secretion	Stimulation		Stimulation N. X.
Contraction L. I–IV	Inhibition	Inhibition Contraction	Bronchial muscle Sphincter vesicæ	Stimulation		Stimulation N. X. Relaxation N. pelvicus.
Relaxation L. I–IV		Relaxation Contraction Relaxation	Detrusor vesicæ Uterus (pregnant) Uterus (gravid)	Relaxation Contraction		Contraction N.pelvicus.
Contraction L. I–IV		Contraction	M. retractor penis	...		Relaxation N. pelvicus.
Sugar puncture		Raised	Carbohydrate tonus	Diminished		
Heat puncture		Raised	Heat balance			
Contraction		Contraction	Pigment cells	Dilatation		

Th. = Dorsal, thoracic segments. L = Lumbar segments. N = Nerve.

sponding action, or none at all to the other points of contact with the same drug, then we are justified in assuming that it is due to a specially reactive disposition of this organ.[1]

Furthermore as this system is very markedly under psychical influences, its relations to what is known as affectivity and ambivalence in psychoanalytic literature is of far-reaching importance.[2] *It is important to bear in mind that these are reaction trends rather than clear-cut types.*

No psychic experience of any kind, joy, sorrow or any other can take place without there being reactions of a definite nature in the body. These are mediated chiefly through the vegetative system. Strange as it may seem, the James-Lange hypothesis that emotional states are peripherally induced seems to be clearly established by Kempf's recent studies. Man should be without shame did he not blush, and without rage if the muscles did not contract; the heart beat more rapidly and thump the breast, and if we had not all those other changes in our vegetative organs which accompany emotional activity. "That the mind acts upon the body and the body acts upon the mind in that important sphere of psychic activity, the emotional, is quite clear." It is significant that the psychoreflexes manifest themselves in many ways. The different emotional states have qualitatively different manifestations in various parts of the body. That which applies to sensory stimuli and emotions also applies to every mental act of the individual. Every psychic activity, every voluntary impulse, every fixation of attention, every stimulating idea brings with it a reaction, for all psychic activities are accompanied by emotional variations and feelings. Man is not only governed by pure sensory stimulation, but also by higher intellectual, ethical and esthetic feelings which are but compounded symbolizations of simpler affective states. He must handle the stimuli from without as well as the instinct impulses from within. These he must handle, *i. e.*, adapt either by alloplastic (modify the outside) or by autoplastic (modify self) means. Thus the beaver thickens his coat in winter (autoplastic); man builds a fire (alloplastic) to adapt to the stimulus. The well integrated individual in an automobile driving difficulty quickly calculates all of the dangerous factors and alloplastically steps on the gas or puts on his brakes, whereas the poorly integrated individual autoplastically shuts his eyes, and gets in a jam. The continual minute oscillations of the vegetative nervous system bear witness that the sum of stimuli going to the central nervous system is always varying; that the tone of the vegetative tracts is always changing, that the vegetative balance is never quite stationary.

Daniélopolu is among those who hold to the older conceptions of the

[1] Aschner, B.: On the Present Status of the Pharmacodynamic Functional Tests of the Vegetative Nervous System, Vienna Clinic, May, 1923.

[2] Kempf: The Autonomic Functions and the Personality, loc. cit. Pyschopathology, Mosby, St. Louis, 1921.

clear cut antagonism between these two systems. By complicated electropolygraphic studies[1] he arrives at the following scheme.

Vegetative hypertonia.	General.	Amphotonia.
		Vagotonia.
		Sympathicotonia.
	Local.	By lesions of nerve paths.
		By reflex areas.
		By organ lesions.
Vegetative hypotonia.	General.	Hypoamphotonia.
		Hypovagotonia.
		Hyposympathicotonia.
	Local.	By lesions of nerve paths.
		By reflex arcs.
		By lesions of organs.

He constructs a table of figures of the heart rhythm.

	Sympathetic tonus.	Parasympathetic (vagus) tonia.	Total dose of atropine to completely paralyze vagus, mg.
Normal	116–128	48–58	1.5
Sympathicotonia	136–156	48–58	1.5
Vagotonia	116–128	70–80+	1.75–2+
Amphotonia	140–150+	72–98	1.75–2+
Hyposympathicotonia	100–108	48–52	1.5
Hypovagotonia	116–128	0–33	1.25–1
Hypoamphotonia	78–104	30–36	1.5 –1

SPECIAL PATHOLOGY OF THE VEGETATIVE SYSTEM.

Eye Sympathetic.—The ciliary, pupillary sphincter and dilator muscles, Müller's orbital muscles, and the tear glands are all vegetative organs of the eye which are innervated in part by parasympathetic and part by sympathetic fibers. The pupillary innervation is of special moment. The synapse of the dilator sympathetic fibers is the ciliospinal center in D1-3. These fibers pass through to the superior cervical ganglion where a synapse is made. Here fibers pass to the Gasserian ganglion, join with the trigeminus and in the long ciliary nerves, pass to the vessels, dilator pupillæ and to Müller's muscle, which pushes the eyeball forward. A parasympathetic pathway (sphincter) passes by means of the oculomotorious and ciliary ganglion. Connections with the cerebrospinal axis are many. The central course of the precellular fibers of the smooth ciliary muscle of accommodation of the iris sphincter is not certainly fixed, although the evidence points to a midbrain synapse in the anterior median nucleus of the oculomotor. (See Oculomotor, also Grevings charts pp. 164, 165, 166, 167.)

Several important clinical conditions depend upon the complicated pupillary innervation, the chief of which are: (1) absolutely stiff pupils, (2) Argyll-Robertson pupil, (3) sympathetic paralysis, (4) variations and deformities in pupils.

1. In the first all automatic stimuli to the iris muscle are inoperative with the exception of the sympathetic, which of itself has a minimal

[1] La Bulletin Medicale, 1925, p. 977.

action. The pupils are dilated and distorted. Since the ciliary ganglion serves for parasympathetic activity, any disturbance of the ganglion gives rise to great dilatation, which is more marked than is produced by nuclear or peripheral lesions of the oculomotor. Absolute rigidity is seen particularly in fainting, high grades of anxiety or fear, frequently in hysteria, in most epileptic convulsive attacks, and in central cerebrospinal syphilis. Pupillary inequalities are frequent in the psychoneuroses and such anomalies have special significance in the psychoanalytic study of the repressions of unconscious material.

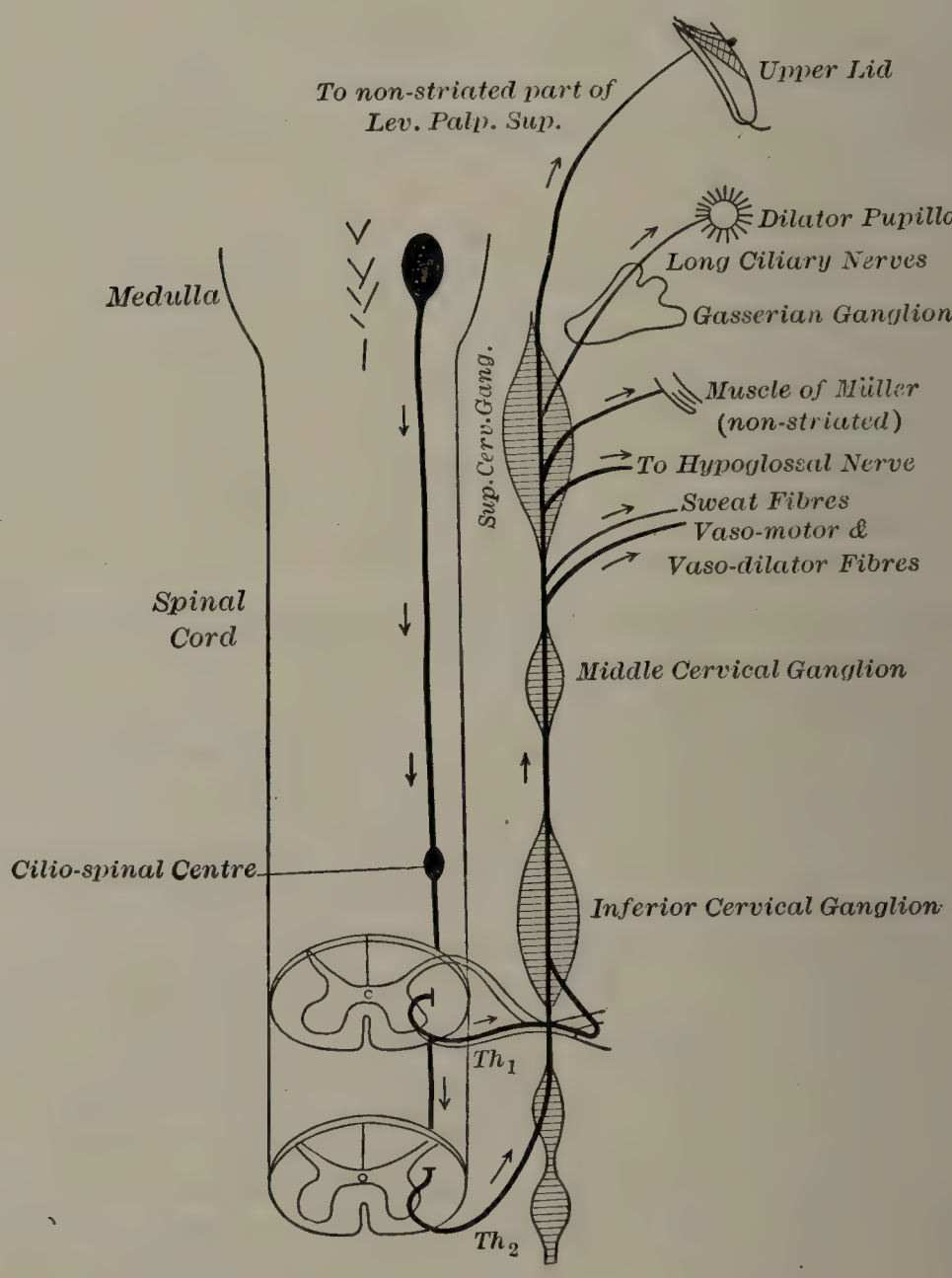


FIG. 51.—Diagram of course of oculopupillary fibers of cervical sympathetic. (Stewart.)

2. The Argyll-Robertson pupil is a complicated phenomenon. It has already been described. (See Examination.) It is an extremely common sign in neurosyphilis, but not in this alone. It is occasionally found in extreme alcoholism (Korsakov particularly) epidemic encephalitis, from other toxemias[1] and occurs from rare and isolated lesions of the corpora quadrigemina. In apes, Karplus and Kreidl have shown that a severance of commissural association fibers passing

[1] Bierman: Diabetes, Neur. Zentralblatt, 1914.

in the arm of the anterior corpora quadrigemina to the antero-lateral border of the anterior corpus will cause a bilateral reflex pupillary rigidity with retention of pupillary activity for accommodation, convergence and psychical stimuli. Chronic meningeal exudates in syphilis pressing upon these fibers may account for the frequency of this symptom in tabes and paresis. Explanations are numerous, however, and may be consulted in the literature. (Willbrand and Saenger, *Die Neurologie des Auges*, see citations in Examination section.)

3. Sympathetic paralysis, or Horner's syndrome, is characterized by retraction of the bulb, narrowing of the palpebral fissure, dropping of the upper and raising of the lower lid and myosis, with conservation of the psychical and light reflexes of the pupil.



FIG. 52.—Partial Bernard-Horner's syndrome of right eye in exophthalmic goiter.

Topographically the picture results from pressure on the sympathetic fibers (goiter), a lesion of the cervicodorsal cord (hematomyelia), gliosis (syringoencephalomyelia), myelitis, especially of the upper dorsal region (Dejerine-Klumpke, Budge's centers), thrombosis of the posterior inferior cerebellar artery, cervico-dorsal radiculitis, epidemic encephalitis[1] and in certain hysterical conversions, compulsive tics, and psychotic projections.

4. Combinations of the three just recorded.

Tests with cocaine and adrenalin are of value in determining the sympathicotropic activity, a 2 per cent. cocaine solution stimulating the dilator fibers. A failure to cause mydriasis is evidence of weakness

[1] Jelliffe: Journal of Nervous and Mental Diseases, June, 1925. Laignel-Lavastine and others.

of the sympathetic. Where such a paresis-producing lesion may lie, pre- or postganglionic, above or below the superior cervical ganglion, can be determined by the use of a 0.1 per cent. solution of the sympathicotonic hormone adrenalin. Six drops in five minutes normally causes no action. If after fifteen minutes, however, there is a marked dilatation the lesion is postganglionic. Adrenalin mydriasis is frequently present in anterior and middle fossa disturbances (orbital disease, fracture of base). This is through the activity of the sympathetic fibers of the carotid plexus which joins with the trigeminus at the Gasserian ganglion. Thus a combination of disturbances of the supra-orbital, with adrenalin mydriasis due to postganglionic sympathetic paralysis, may give important evidence as to the localization of a tumor, or fracture of the base of the skull. Double-sided adrenalin mydriasis (Löwi's reaction) is also seen in hyperthyroidism, pancreatic diabetes, and in increased irritability of the sympathetic nervous system in general. In psychoses, particularly in dementia precox, the reaction is very variable, and the test is complicated by a variety of emotional reactions.

Vagotonic reactions give rise to accommodation cramps with lacrimation which may be diminished by atropin. In youth, when vagotonia is more pronounced, atropin acts less protractedly than in older people, and pilocarpin in the eye may cause von Graefe's symptom as a sign of an increase in the tonus of the autonomic levator palpebræ.

There are a number of eye affections whose pathogeny is in part dependent upon vegetative nerve disturbance. A whole book could be written upon psychical correlates in eye pathology. These require too much time to be worked out by the orthodox ophthalmologist and hence are neglected. Psychogenic myopias and hypermetropias are not infrequent. Only a few of the more important vegetative disturbances can be referred to here, and briefly.[1]

Glaucoma.—This serious affection of the eye, speaking of the acute inflammatory or congestive type, is due to a disturbance in intraocular tension which is largely dependent upon sympathetic control. The precise mechanisms are still incompletely analyzed. The administration of atropin by its paralyzing action on parasympathetic fibers—paralysis of sphincter iridis, ciliary muscle—also produces marked increase in intraocular tension, and hence augments the difficulties. Pilocarpin and eserine (physostigmin), with their opposing actions on the sympathetic, decrease intraocular tension and hence alleviate (temporarily in the early stages) the syndrome. Excision of the cervical sympathetic ganglion diminishes the tension, and is resorted to in the treatment of glaucoma, thus showing the definite part played by the vegetative nerves in this affection.

Herpes cornæ, herpes ophthalmicus, keratitis neuroparalytica are

[1] Consult Archambault: The Symptomatology of Certain Infectious Processes Involving the Ciliary Ganglion, Jour. Nerv. and Ment. Dis., September, 1917, vol. **46**. Jelliffe: Myopia as a Castration Symbolism, Int. Ztschr. f. Psychoanalys, 1926. Inman: British Jour. Med. Psychol., 1926.

among the eye affections due to implication of vegetative fibers, located chiefly in the trigeminus sheaths, or in the Gasserian ganglion. Keratitis neuroparalytica occurs not infrequently as a complication in resection of the ganglion. Resection of the cervical sympathetic seems to cure this keratitis (Cimoroni).

Acute edemas of the conjunctiva, of the retina and iris, analogous to Quincke's edema are to be classed probably with vegetative nerve disorders. These have largely been disguised under pseudonyms as "rheumatic," or "lithemic," or "gouty." Vagotonic individuals frequently show a number of eye anomalies; floating particles in vitreous, etc.

Certain forms of accommodation disorders (hypermetropias, myopias, spasms, etc.), frequently called "eye-strains" are, properly speaking, vegetative disorders of the ciliary muscle. Many of them are largely psychogenic in origin.

Tear Glands.—These are parasympathetically innervated through the superior cervical ganglion and sympathetically through the sphenopalatine ganglion. Irritation of the neck sympathetics causes increase, paresis of the same, diminution in the secretions. The postcellular branches of the neck ganglia, secretory or vasomotor fibers, pass in the internal carotid plexus reaching the glands either by the way of the ophthalmic plexus or through the cavernous plexus and the lacrimal sensory branch of the trigeminus. The secretion is markedly under psychical influences as is universally recognized.

Mucous and Salivary Glands—The vegetative control of these is exercised through the sphenopalatine, otic, submaxillary, and sublingual glands. The sphenopalatine sends only parasympathetic vasodilator fibers through the posterior nasal nerves to the mucous membrane of the nose; sympathetic vasocontrictor fibers come from the cervical sympathetics.

The parotid gland has both a sympathetic and parasympathetic supply, the former from the cervical sympathetic, the latter through the otic ganglion. The small superficial petrosal is its viator or precellular root, the auriculo-temporalis of the trigeminus is its postcellular branch. The parasympathetic bulbar center is Kohnstamm's nucleus salivatorium inferior. Parasympathetic stimulation delivers a different type of secretion from sympathetic stimulation. Lesions of the tympanic in the mastoid operation give rise to parotid disturbance, and may be looked for in middle-ear disease.

The submaxillary and sublingual glands have a sympathetic and a parasympathetic supply. The latter causes vasodilator and secretory stimulating effects and has its parasympathetic bulbar synapse in Kohnstamm's superior salivatory nucleus, its rami communicans albi in the chorda tympani and through the lingual to the gland. The sympathetic vasoconstriction and secretory stimulating fibers are derived from the cervical sympathetic. Parasympathetic stimulation causes the full, thin, watery, salty secretion, cut off by atropin; the sympathetic, the scanty, viscous (organic constituent) secretion acted

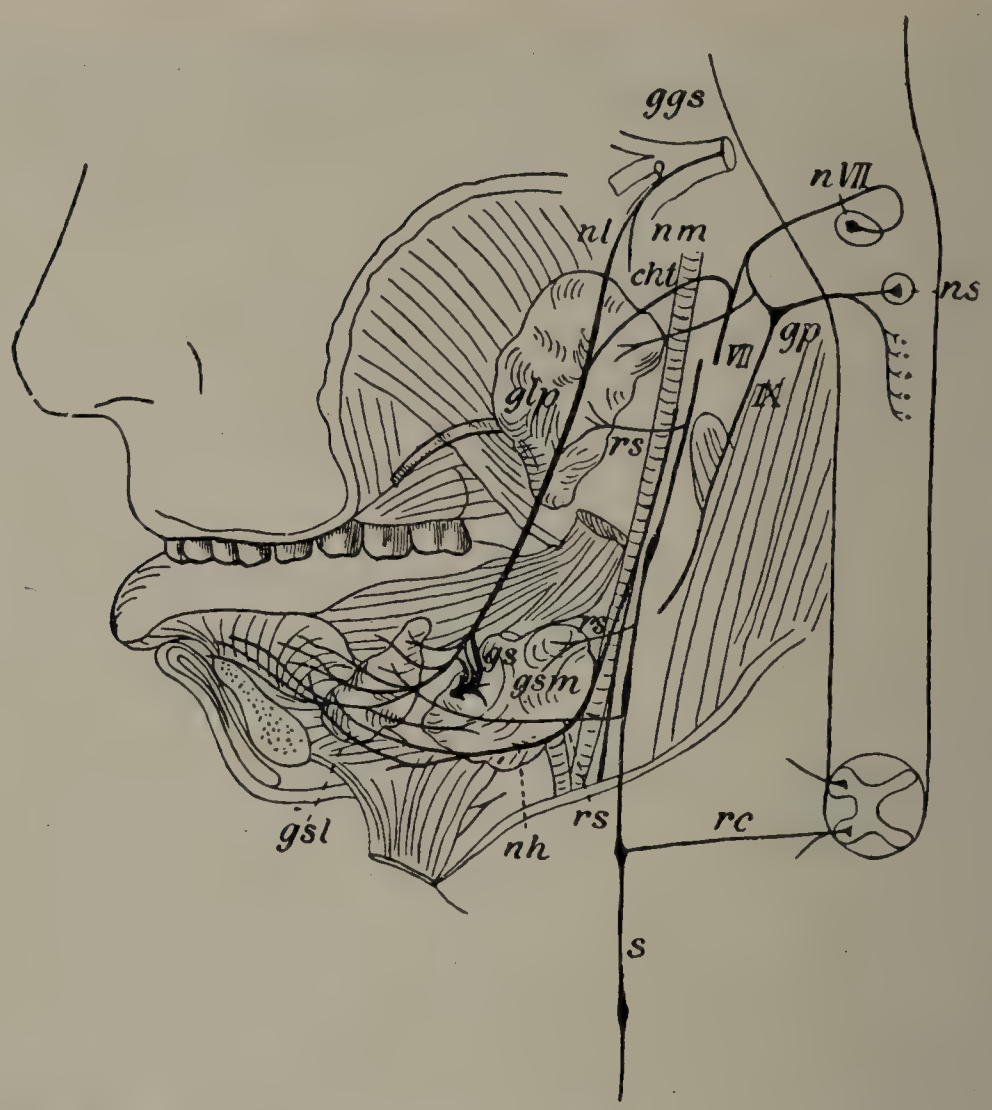


Fig. 53.—The innervation of the salivary glands: *glp*, parotid; *gsm*, submaxillary; *gsl*, sublingual; *ggs*, Gasserian ganglion; *nl*, lingual nerve; *nm*, mandibular nerve; *nVII* facial nerve nucleus; *cht*, chorda tympani; *VII*, facial nerve; *IX*, glossopharyngeal nerve; *ns*, nucleus salivatorius; *gp*, petrosal ganglion; *s*, sympathetic; *rs*, sympathetic branches; *gs*, submaxillary ganglion; *nh*, hypoglossal nerve; *rc*, ramus communicans. (Bechterew.)

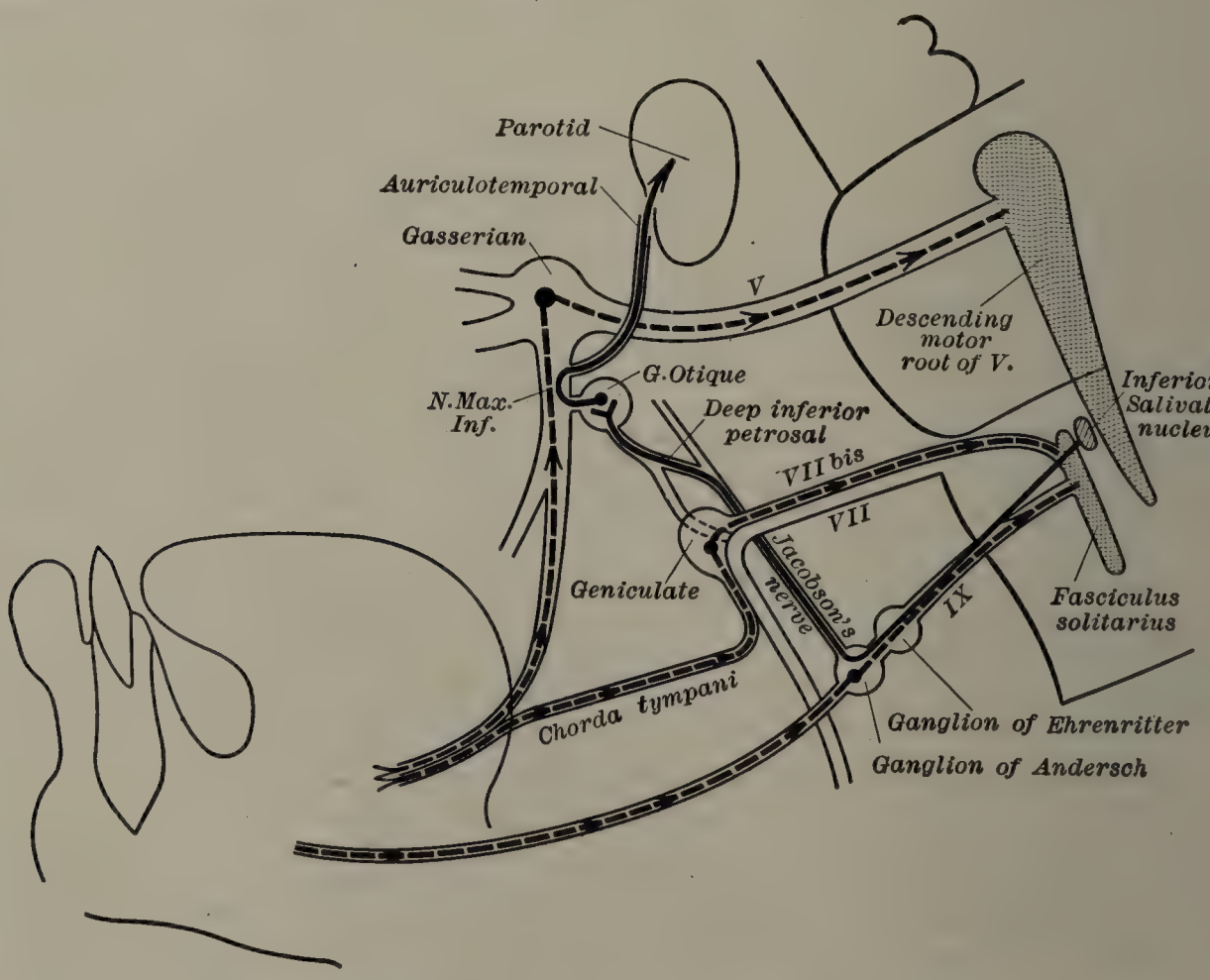


Fig. 54.—Organic apparatus of glossopharyngeal innervation, – – – –, afferent sensory-sensorial pathways; ——— efferent vegetative motor pathways. (Modified from Bechterew and Guillaume.)

upon by cocaine, cholin, adrenalin. Xerostomia (Hadden), xerostomia senilis, xerophobia, excessive production of frothy mucus, constant spitting of mucus in schizophrenia (often symbolic of semen), scanty secretions as seen in anxious states, in stage-fright and other types of fright, sometimes unconscious, as in marked depressions, etc., are among the disorders of the secretions of these glands of neurological and psychiatric interest. The symbolic significance of spitting is extremely complex and fertile in suggestions and its study, particularly in the psychoneuroses and psychoses, only just beginning. Cortical, glossopharyngeal, and trigeminal associations are the basis for reflex stimulation of the glands.

Neck Sympathetic.—The superior cervical sympathetic supplies, through the internal carotid nerve and the internal carotid plexus, the dilator of the pupils, Müller's muscle, tear, parotid, maxillary and lingual glands, the pilomotors, vasoconstrictors, and sweat glands of the face.

Cervical Sympathetic.—Partial syndromes due to implication of parts of the cervical sympathetic fibers have just been described under eye, tear gland and mucous and salivary gland disturbances. More extended syndromes both of stimulation or of paralysis of the cervical sympathetics are frequently seen and are of considerable importance. In military practice cervical sympathetic wounds are frequent. As has been pointed out fibers from many diverse sources converge within the cervical sympathetic. The action of these fibers, in response to stimulation or to paralysis, shows a great variety of combinations such as vasoconstriction, vasodilatation of the cerebral vessels giving rise to congestion, hyperemia, anemia, migraine, epileptic attacks, etc., of the thyroid vessels, causing varying states of dysthyroidism; of the base of the orbit, causing enophthalmos, exophthalmos; the fundus of the eye (glaucoma); of the salivary and lingual glands (variations in secretion, xerostoma, etc.); of the skin of the face and head (anidrosis, hyperidrosis, seborrhea, horripilation, skin eruptions, acne, eczema, anomalies in pigmentation, hair, beard, eyebrows) modifications of active unstriped muscle, partial ptosis, widening or narrowing of the palpebral fissures, dilatation or contraction of the pupils, acceleration or retardation of the heart action. These results, single symptoms or in various combinations, may be produced by lesions in the neck, in the cerebral cortex, medulla, and spinal cord (trauma, tumor, syphilis, multiple sclerosis, syringoencephalomyelia, etc.), in the brachial plexus, in the chest cavity (tuberculosis, tumors, pneumonia), depending upon the anatomical pathways or synapses implicated.

The typical complete picture of a cervical sympathetic stimulation will show dilatation of the pupils, exophthalmos, from retraction of the lids and protrusion of the bulbs, increased intraocular tension, myopia, submaxillary and parotid hypersecretion (sympathetic salivation, thick and comparatively sparse), cardiac acceleration, vasoconstriction of the skin of the head, neck; vasoconstriction of the meninges, the brain, the eyes, the mucous membrane of the mouth and tongue, and of the thyroid gland.

An analogous picture of sympathetic paralysis (Bernard-Horner syndrome), well contracted pupil, diminished intraocular tension, abolition of cilio-spinal reflex, loss of cocaine dilatation, retraction of the eyeballs (enophthalmos), hypermetropia, loss of luster of eye,

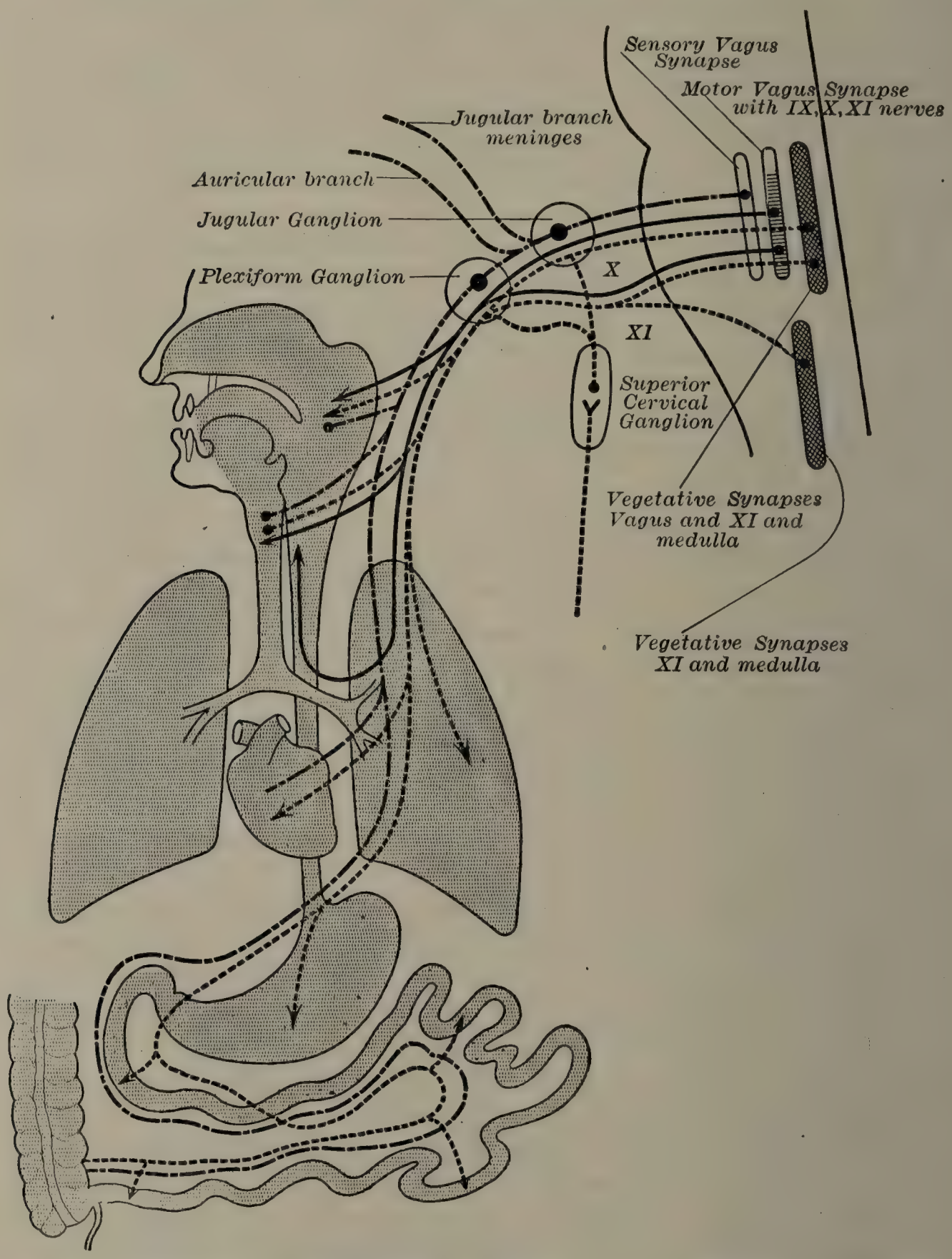


FIG. 55.—Innervation of digestive tract by vagus, – – – – –, sensory; ——— motor; vegetative motor. (Guillaume-modified.)

slight ptosis from palsy of Müller's muscle, easily overcome by voluntary action of III N, dilatation of the conjunctival vessels, homolateral rise in temperature of the side of the face, increase in the lacrimal secretions, sialorrhea (thin, watery secretion), slowing of the heart, anidrosis, and seborrhea sicca.

Gastro-intestinal Syndromes.—Vegetative disturbances in swallowing are commonplaces of every-day medical practice. The universal so-called "hysterical globus," "the lump in the throat," which Democritus described metaphorically as the wandering uterus, *i. e.*, the instinct for reproduction, and which Galen triumphantly, and stupidly disproved, since the "uterus could not move," is one of the most familiar. It is a very complex phenomenon, and its etiology is multiform (psychoanalytically speaking, much over-determined). In the psychoneuroses it is often a symbol of disgust, a surrogate for vomiting, an unconscious remnant of infantile food impregnation fantasies; again it is a symptom of fear (inferiority symbol) largely determined by the unconscious peeping and exhibitionistic infantile trends.

Local constrictions of the esophagus are frequently met with in neurotic individuals. The accompanying *x*-ray photograph shows such a variable stricture due to vagotonic disturbance in a case of anxiety hysteria in which rumination was a prominent feature and which could be induced by irritating the patient by even the simplest forms of contradiction (Fig. 56).

The *vagus* plays such a large role in these disturbances that a few general words may be said concerning its structure. A complete analysis of all of the vagus components would require a volume, hence only a mutilated thumb-nail sketch is here possible.[1] (Fig. 46).

The *vagus* like the oculomotor, glossopharyngeal and facial is a mixed nerve and contains motor, sensory and vegetative pathways. The somatomotor nucleus is the nucleus ambiguus; the nucleus solitarius is the sensory nucleus; the visceral nucleus for the heart, lungs, and digestive system is the nucleus dorsalis vagi lying in the walls surrounding the third ventricle. In the dorsal vagus nucleus there lie closely associated a number of most important visceral synaptic stations upon whose integrity depend breathing, the heart beat, the taking in of food and many metabolic organizations. Sympathetic synaptic connections have been demonstrated by retrograde degeneration methods (Lewy). These lie in close association with medullary synaptic cell groups, chiefly the nucleus periventricularis (Friedmann's nucleus infundibulo-mammillaris—Forel's field-nucleus). In certain cases of paralysis agitans, in epidemic encephalitis, muscular stiffness, etc., these nuclei are involved and determine marked vegetative symptoms. Cardiac, respiratory changes, glycosuria, etc. This nucleus is in connection with the striatum, the tuber cinereum, substantia nigra, corpus Luysii. These latter hypothalmic stations mediate for the proper functioning of coördinated vegetative, *i. e.*, metabolic functioning. Since the carbohydrate metabolic regulation pathways may here be interrupted muscle function may be disordered. From the study of the phyletic organization of these nuclear masses it can be

[1] Calhoun, H.: Résumé on the Circulatory System Literature of 1920. Am. Jour. Dis. Child., 1921, **21**, 586. Westphal, A: Studies on Gall Duct, Ztschr. f. klin. Med., 1923, vol. **96**. Gustav von Bergmann: Funktionelle Pathologie des vegetativen Nervensystems. Mohr and Staehelin: Handb. d. inn. Med., 2d ed., Berlin, 1926.

shown how the visceral, suprarenal, pancreatic and gonadal systems come to be associated here, and that such important functions as growth thermo regulation, recovery from fatigue, biochemical tonus, carbohydrate metabolism, and primary and secondary sexual developments

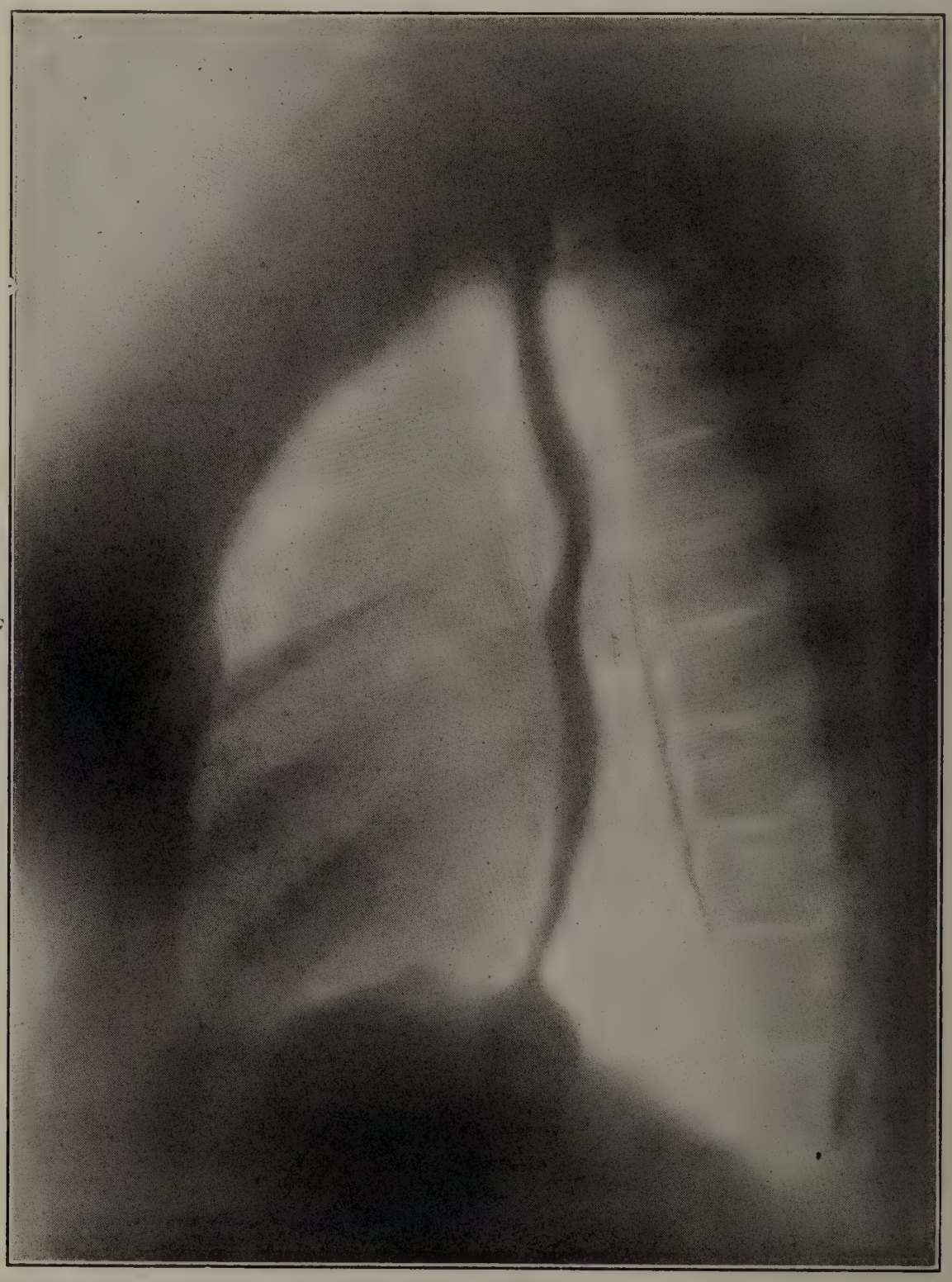


FIG. 56.—Vagotonic contraction of esophagus. From a patient with Manic-depressive make-up with Anxiety Hysteria symptoms.

may be variously modified when these structures are functionally interfered with either by toxins, inflammatory foci, or psychical stimuli.[1]

[1] For a more complete discussion of these most intricate problems which defy a didactic text-book summary see Müller's 1924 monograph, Die Lebensnerven, and particularly Spiegel's able summary of the localization of vegetative functions in the central nervous system, Ztschr. f. d. g. Neur. u. Psych. Referat., 1920, **22**, 142. Jour. Nerv. and Ment. Dis. June, 1922, vol. **56**. Also the important researches of von Monakow's students in his Arbeiten and the numerous studies of Dresel, Greving, Lewy, etc., already cited, also contribution of G. v. Bergmann, loc. cit.

All three sets of fibers travel through the jugular and nodosus ganglia to form the vagus; the jugular ganglion probably forming the synapse for sympathetic connections and anastomoses. The two ganglia phylogenetically seem to indicate two components: (*a*) The pure motor branches are the rami pharyngeus; (*b*) the pure sensory branches are the meningeal and superior laryngeal; (*c*) the mixed motor-sensory-visceral is the recurrent laryngeal sending motor fibers to the larynx, receiving sensory fibers from the trachea and the visceral fibers supplying the heart, aorta, and vessels of the larynx; (*d*) the purely visceral branches pass to the digestive tract, the heart, the liver and the lungs.

Only a partial analysis of the visceral branches will be taken up here; the motor and sensory are discussed later under the Cranial Nerves.

Esophagus.—The entire digestive tract is served by the sympathetic (narrow sense), whereas the vagus (parasympathetic) only is distributed to the lower two-thirds of the esophagus, the stomach, and the intestines to the descending colon. The combined action is stimulating (parasympathetic) and depressing (sympathetic), which actions are apparently reversed in the case of the heart muscle. Local ganglion cells seem to regulate the motor functions. Tactile and chemical stimuli are apparently unresponded to in the upper part of the esophagus. Thermal stimuli above 40° C., below 30° C., are felt (Boring). Deep pressure sensibility is present, but the pathways are not definitely located. The lower end of the esophagus responds to chemical, thermal, psychical (see Fig. 46), and possibly other types of stimuli (Heart-burn).

Stomach and Intestines.—The *receptors* in the stomach walls have not yet been sufficiently analyzed to permit physiological inferences. Local ganglion cells in the walls of the digestive tract are very frequent and may be regarded as the terminal motor neurons of the vegetative system. (See Fig. 46.) The stomach is under intimate associative relations with sight, hearing and smell and its affect (psychical) reactivity is extremely sensitive. Ordinary sensibility to tactile and thermal stimuli is lacking, but deep sensibility fibers are present and carry pressure stimuli (pain, colic, crises). Specific chemical stimuli are undoubtedly abundant. The pathways used for these are probably through the vagus (stimulating) and the splanchnics (opposing). Fig. 55 shows the distribution of the vagus fibers. Thus both parasympathetic and sympathetic systems are utilized. Eppinger and Hess have seemed to show that in the laryngeal, bronchial, esophageal, gastric, intestinal, genital and rectal crises, in tabes particularly, the parasympathetic system only is involved; the vagus bulbar central for the upper types, the pelvic parasympathetic muclei for the lower ones. Foerster describes vagus and splanchnic (?) crises. The former are without pain, but with nausea, hypersecretion, and vomiting, the latter with pain and hyperesthetic skin zones, and increased epigastric and abdominal reflexes.

Timme[1] conducted a series of experiments partially cutting off parasympathetic stimuli, thus producing increased sympathetic stimuli action upon the stomach with important and far-reaching implications bearing upon the problems of visceral disease, in general, and its relation to vegetative nervous system activity. Such imbalanced actions are constantly brought into operation, as has been stated, by mechanical factors, by toxins, by hormones and by mental stimuli. Visceral disease then should

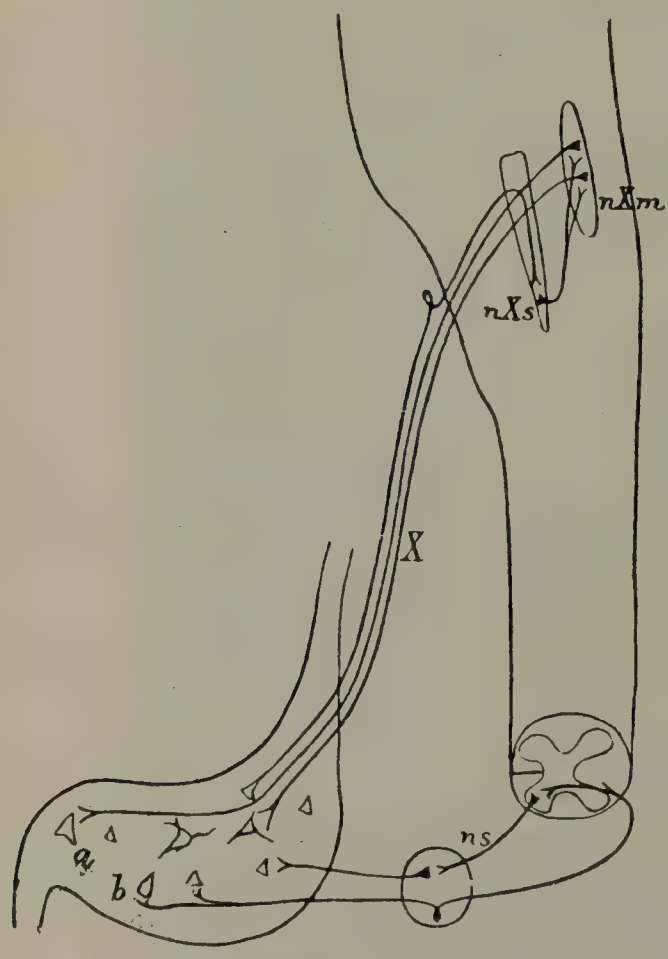


FIG. 57.—Scheme of stomach innervation: *a*, *b*, ganglia in walls of the stomach; *nXs*, sensory nucleus of the vagus; *nXm*, motor root of the vagus; *ns*, splanchnic. (Bechterew.)

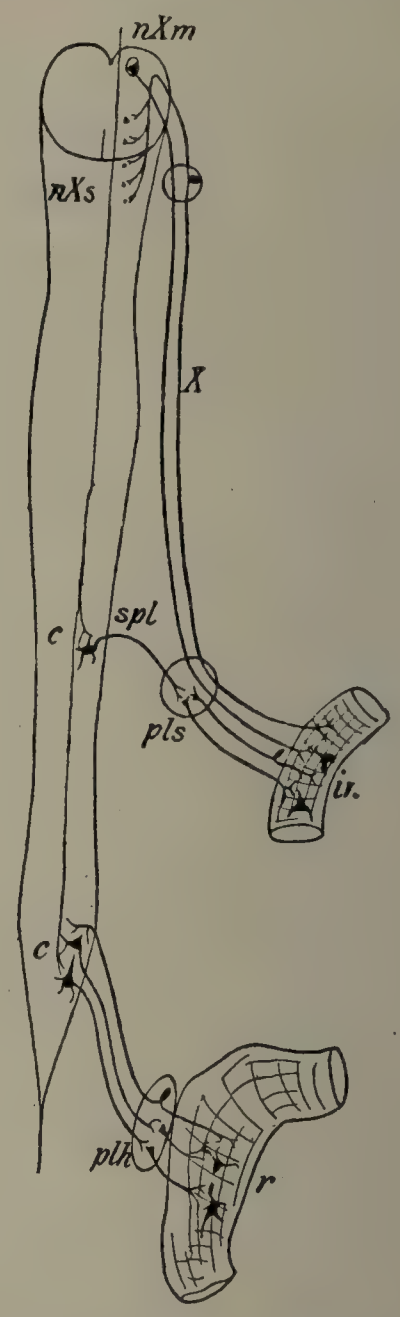


FIG. 58.—Scheme of intestinal innervation: *in*, small intestine; *r*, lower end of the large intestine; *pls*, celiac plexus; *plh*, hypogastric plexus; *spl*, splanchnic; *c,c*, spinal center of intestinal movements; *X*, vagus; *nXm*, motor nucleus of the vagus; *nXs*, sensory nucleus of the vagus. (Bechterew.)

be studied from the point of view of the balanced relations of these two antagonistic systems.[2]

The local topographical diagnosis and the physiological understanding of nervous dyspepsias, the motility and secretory anomalies (achylia, hypersecretion, hyperacidity, increase of gas, ptoses, pyloric

[1] Experimental Studies on the Nervous Mechanism in the Production of Hyperplasia, Jour. Nerv. and Ment. Dis., 1913, **40**, 311.

[2] Pottenger: 'Visceral Disease and the Vegetative Nervous System, 1925, vol. **3**. Eisler: Nervensystem und motorische Funktion des Magens, München. med. Wchnschr. 1914, 2048. Müller, L. R., loc. cit., Guillaume, loc. cit., G. v. Bergmann, loc. cit., and earlier studies of Head and Mackenzie.

spasm, hour-glass contractions), changes induced in Addison's disease[1] and in exophthalmic goiter and in all of which sympathetic (psychical) influences play a large role are as yet not thoroughly analyzed. Radiographic study[2] is aiding in a knowledge of these anomalies, especially the motor ones, but the alterations seen must be interpreted chiefly as *results* and not as *causes*. Emotions, toxins and their resultant endocrine stimuli are the *causes*, the radiographically seen anomalies the *results*. After years of maladjustment structural changes result and a vicious circle is established in which cause and result are inextricably interwoven in their general effects.

Individual and social adjustment at psychological levels seems to influence them much more effectually than measures addressed to modify the perverted chemisms and motility, especially at the beginning of these disorders. There is little doubt that long-continued psychical disturbances which cause very pronounced secretory and motor anomalies may ultimately induce definite structural changes. Many visceroptoses are of this type. The relaxation is due to irregularities in the reciprocal innervation of the sympathetic and autonomic pathways, induced in many instances through psychical maladjustments. Possibly toxic factors may ultimately play an important role.

Phrased in another manner, in certain patients emotional factors are undoubtedly the primary ones, in others it may be, although this must be proved, mere asseveration is not sufficient, toxic factors (intestinal absorption) may play the primary role. Speaking of the intestine as a "sewer" is a nonsensical metaphor, a left over of primitive medicine.

The vagus, by way of the solar ganglia, stimulating the terminal neurons, depresses the peristalsis and secretions of the intestines. The intestinal movements, however, may take place independently. The tactile-mechanism-reflexes are continuously active; chemical reflexes are operative during the passage of absorptive material. Each have their sympathetic and parasympathetic pathways—working independently one of the other. The chief psycho-reflex pathways seem to act through the vagus; thus at the upper end, such psycho-reflex activties show increase of secretion with appetite, loss of secretion from worry, fear, and, acting on the autonomic pelvic arc, the various constipations and diarrheas so frequently of psychic origin; the anal erotic and anxiety neurosis phenomena so well elaborated by Freud being among them. Purely sympathetic disturbances with increased peristalsis and serous fluid may result from loss of function of the splanchnics, either as a vital reaction to cutting, trauma, etc., or to psychical influences, as from shock, emotion, fear and desire, both conscious, foreconscious and unconscious; from toxins, hormones, anaphylactic substances,[3] also intestinal putrefactive products, etc.

[1] Hass, S. V.: New York State Med. Jour., October, 1919.

[2] Albu: Der nervöse Magen im Röntgenbild., Berl. klin. Wchnschr., January 5, 1920, and numerous recent radiological studies.

[3] See a Comprehensive and Valuable Study on the Relation of Anaphylaxis to the Vegetative Nervous System by J. H. Smith, Jour. Nerv. and Ment. Disease, January, 1917, vol. 45.

The great importance of the parasympathetic and sympathetic control factors on the vessels of the abdominal cavity and organs cannot be more than mentioned. Here the peripheral vascular regulations are in direct contrast with the abdominal ones, and hence the purely mechanical and vital process of adjustment of blood-pressure regulation takes place. The interpretation of the phenomena of shock must come about through a study of these factors, but such cannot be taken up here.[1]

Another feature of activity of the vegetative nervous system concerns itself with the gastro-intestinal ferments, and the specific secretions or hormones (gastrins, gastro-secretine, enterokinases of the various authors). Many hormones of the endocrinous glands undoubtedly influence the gastro-intestinal functions. The diarrhea of exophthalmic goiter (thyroxin) is a very classical example of this influence. Gastric ulcer, duodenal[2] ulcers resulting from increased adrenalin activities are other less comprehended reactions in this important field. Here the adrenalemia is directly under parasympathetic control and is largely a response to fear and hate—conscious and more temporary, unconscious and usually more persisting. Hence when it is said that these disorders appear in asthenic states, this means the asthenic states are usually unconscious fear states and are interpretable through psychoanalysis[3] (see Fig. 59). Pseudoappendicitis is a frequently observed situation, which with various forms of intestinal spasms, are conditioned by emotional factors.[4]

Westphal has shown further that in a large number of peptic ulcers he has observed the signs of vegetative disorder such as dilated pupils, exophthalmos, increased secretions, bradycardia, spastic constipation, increased vascularity of the thyroid, loss of abdominal reflexes, increased knee-jerks, strong reactions to adrenalin, atropin and pilocarpin. Accompanying symptoms of vegetative nature were gastrosuccorrhea, pylorospasm, hour-glass contraction. Pollak has found ulcers of the stomach in local encephalitis involving medulla and substantia reticularis. Similar vegetative pathway disturbances result from syphilis, thyroid toxemia, encephalitis from influenza, etc.[5]

The gastric mucous membrane secretes a hypothetical hormone which influences the activity of peristalsis through the sympathetic pathways.

Surgical procedures become necessary after a number of years of faulty vegetative functioning. The vicious cycle set up by faulty emotional discharges ultimately causes tissue changes. Premature

[1] Consult Crile: Study of the Emotions, Philadelphia. 1915.

[2] See Bircher, M. E.: Zur psychologischen Aetiologie eines Duodenal Geschwüres, Schw. m. Wchnschr., 1924-7 vol. **54**, 173.

[3] For suggestive studies on changes in vegetative ganglia, visceral disease and psychoses, see Morse, M. E., Jour. Nerv. and Ment. Dis., January, 1917, vol. **45.** Laignel Lavastine: Loc. cit.

[4] Liek: Ueber Pseudoappendicitis insbesonder über das Krankheitsbild des nervösen Darmspasmus, Mitt. a. d. Grenzgeb. Jour. Med. u. Chir., 1920, **32**, 153.

[5] Wien. med. Wchnschr., 1923, **73**, 335.

surgery is pernicious; too delayed a relief in long-standing cases equally foolhardy. Parasympathetic cases are apt to respond well to many surgical procedures. The atonic or sympathicotonic cases do badly as a rule.[1]



FIG. 59.—Duodenal ulcer as end-result of a vicious psychogenic maladjustment. Unconscious hate, unconscious flight, hyperadrenalemia with later exhaustion, duodenal spasm as compensating tension states, finally ulcer as links in chain. Curable only by surgery. Could have been prevented by psychoanalytic therapy in earlier years. A, filling defects; B, incisure.

Rectum.—The chief innervation is through the hemorrhoidal plexus and the inferior mesenteric. Both parasympathetic and cerebrospinal influences are active. Voluntary muscle activities play a large role in defecation, the grade of tension in the rectum, however, is registered by the splanchnic system, which is responsible for the original impulses, after which voluntary and involuntary activities are operative. The spinal splanchnic synapses are located in the lumbo-sacral spinal segments. The cortical association connections are thought by Bechterew to be in the sigmoid gyrus. Frontal association pathways are also present, interference with which causes involuntary defecation, as in frontal tumor, general paresis, epileptiform convulsion, profound stupor, emotional loss of control, etc.

Interruption of spinal pathways may cause obstipation or diarrhea (tabes, poliomyelitis, multiple sclerosis, tumor, syphilis of cord, hematomyelia, syringomyelia, etc.). Here deep sensibility conducting fibers—splanchnic and cerebrospinal—are interfered with and the splanchnic reflexes fail to establish the psychical connections either for

[1] Hutchinson, R.: Disappointments after Gastro-enterostomy, British Med. Jour., May 3, 1919. Also see L'ulcère chronique de l'éstomac et du duodenum. Verhandl. d. Schw. Gesellschaft f. Chir. Correspblt. f. Schw. Aerzte, 1919, **49**, 708 and 709.

compulsion (discharge) or control of sphincter (retention). The anal reflex here is of great localizing value, its positive appearance ruling out somatic disease of the lower sacral and coccygeal segments. Lesions of the cervical or dorsal cord interfere with the voluntary activities of the abdominal muscles in defecation, while lesions of the lower lumbar cord cause changes in the voluntary sphincters. In sacral lesions, with involvement of the external sphincter nucleus, the anus remains wider open, not so sharply corrugated, not as vigorous in

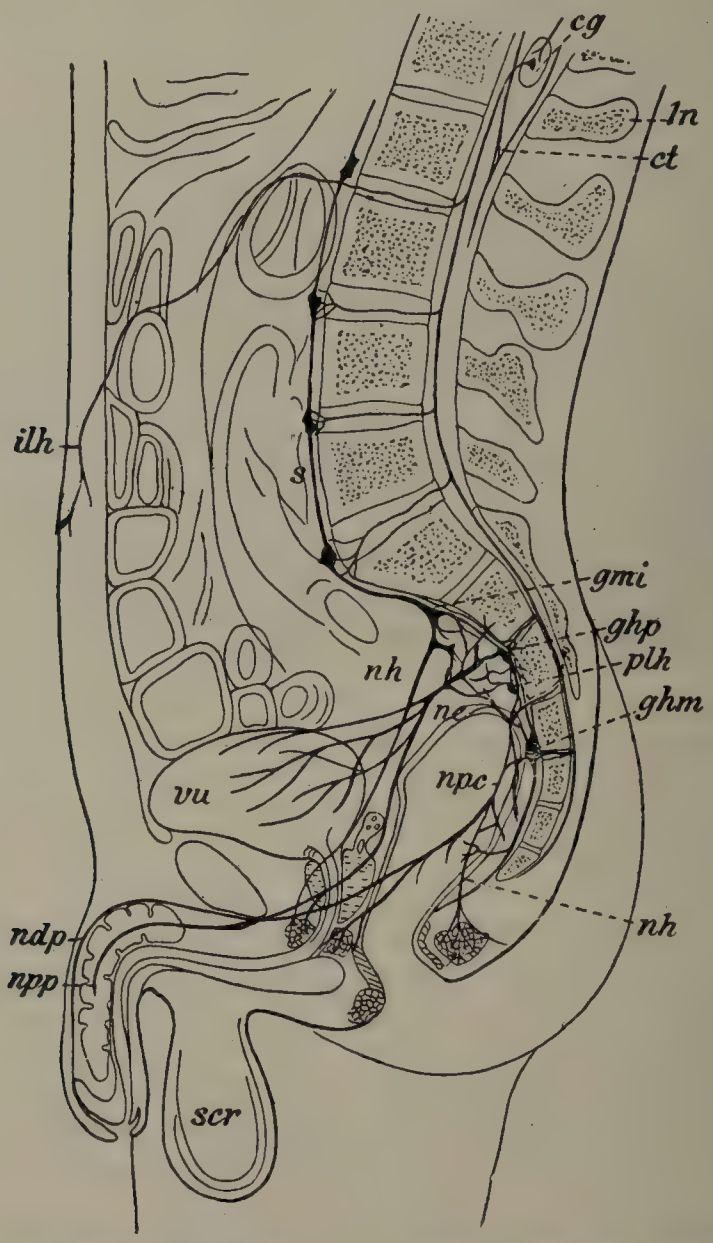


FIG. 60.—Scheme of pelvic innervation; *cg*, spinal genital center; *1n*, first lumbar; *s*, sympathetic; *ct*, conus; *ilh*, ileohypogastric; *gmi*, inferior mesenteric ganglion; *ghp*, hypogastric ganglion; *plh*, hypogastric plexus; *ghm*, hemorrhoidal ganglion; *nh* (above), hypogastric nerve; *ne*, sacral nerve; *s*, erigens; *npc*, common pudendal; *nh* (below), hemorrhoidal nerve; *ndp*, dorsalis penis; *npp*, deep perineal; *vu*, bladder; *scr*, scrotum. (Bechterew.)

closing and there is loss of the anal reflex. Notwithstanding the loss of the voluntary sphincter, splanchnic closure is possible. There is therefore no single defecation center in the lower cord.

Severe constipation may be of purely splanchnic origin. This constipation reaction may occur at physico-chemical levels, such as those due to excessive absorption of water; at vital levels, *i. e.*, resulting from pain from kidneys, gall-bladder, peritonitis, chronic appendicitis, hemorrhoids; or at purely psychical levels, where infantile pleasure

fantasies may play a large role, *i. e.*, anal erotic in displacement of affects, birth fantasies, etc. Birth and death fantasies which deal with feces, and which are concealed behind constipations and diarrheas are very frequent among psychoneurotics and psychotics, particularly in schizophrenics, (*q. v.*) In schizophrenics frequent fecal discharges, fecal smearings, fecal eating are symbolic activities for which the psychoanalytic technic often reveals the psychical equivalents. The anatomical pathways which make such relationships comprehensible exist in the autonomic fibers. Constipation as a correlate of miserliness is an instance in point, and is more fully discussed in the chapters on the Psychoneuroses. Most constipation is purely psychogenic and the habitual use of cathartics, mineral oils, enemas, rectal dilators, etc., tends to increase it since it provides a recurring anal-erotic (unconscious sodomistic) satisfaction through frequent or large bowel movements.

Spastic Constipation states of varying degrees are enormously frequent. The universal use of cathartics is evidence that the human being does not manage himself well. The trouble usually begins on a psychogenic basis. Study of the unconscious reveals that the Biblical myth of Sodom and Gomorrah still is operative in less obvious degrees in human colon and rectal pathology. The early stages of psychogenic constipations are usually handled up to about the twenty-eighth to thirty-third year of life without striking difficulties. Then there commence to radiate out a host of pathological derivatives which commence to crystallize out as definite syndromes, copiously described in proctological literature. One frequent syndrome develops as follows: Following the spastic constipated states—pouching of the lower bowel commences. Local minute or larger diverticulæ arise—at first largely functional and registered as cramp states, with diarrheas, mucus discharges, colitis of minor grades: Peridiverticular inflammatory complications then begin. In severer forms infectious foci produce vesical reactions, such as accompanying cystitis (colon bacilli cystitis, etc.). Pyelitis may result. Severe sympathicotonic dilatations may arise (Hirschsprung's syndrome in some of its forms). These infectious foci may later determine widespread shock reactions, or chronic septic states. In a large number of introverted individuals, (chronic depressed states, hypochondriacal states, dementia precox cases, etc.), a veritable honey-comb of colonic foci may develop and contribute largely to continue the psychotic reaction. Lane's hypotheses concern resultants of these complicating focal infections, and the drastic surgical procedures (colon resection, etc.), the apparent reliefs necessary. In beginning stages a competent psychotherapy, if the patients are capable of coöperating, can avert the later severe destructive lesions. Between the general ages of forty to fifty, surgery can be avoided. After fifty the vicious cycle has usually progressed to such a point that surgical interference may be necessary to save life. The point to be emphasized is that careful analysis early in the disorder is essential, in which analysis neglect of psychological factors is bound to result in disastrous later developments.

Vagotonic manifestations within the gastro-intestinal tract are of considerable importance, although as yet far from being definitely analyzed. Pilocarpin and physostigmin increase them, whereas adrenalin and atropin diminish them. In vagotonic individuals there are increased esophageal cardiac spasms, tendency to increased salivation and to increased secretions from the nose and eyes. There is spasticity in the peristalsis, as shown by radioscopic examination, due to increased muscular tonus. This latter causes the stomach form of hyperkinetic motility gastroneurosis. It may arise from disorder at the physico-chemical or psychical levels. Hypersecretion and hyperacidity are accompaniments with pylorospasm. Certain cerebrospinal levels may also be involved, as shown by the Head hypersensitive skin areas. Membranous enteritis or colitis with mucus and many eosinophile cells in the blood and mucous secretions is associated with this condition summarized as vagotonia. Here psychical influences are of great moment. The constipation just spoken of may be arranged in this vagotonic group. Renal and biliary colics, spasmodic jaundice, reflex anuria, eosinophilia, and increased glucose tolerance are to be found in this vagotonic group. This type of vagotonics or of regression tends to develop the depressed phases of a manic depressive psychosis if their (usually paranoid) compensations break down.

Diarrheal states in hyperthyroidism, in anxiety neurosis and in various systemic toxemias (acidosis in children) are mediated through vegetative mechanisms. Involvement of the sympathetic nuclei in the cord by poliomyelitis may cause severe diarrhea and constipation symptoms.

Respiratory Apparatus.—The dynamic pathology of the respiratory apparatus can only be incidentally touched upon here. Neurological concepts are imperative, however, if such pathology is to be adequate.[1] The respiratory apparatus is not only a purely vegetative organ serving for oxygen intake and water and carbon dioxide out go. In mammalian evolution it has more and more become an essential part of the bodies energy delivery system, chiefly through symbolic speech activities. The speech mechanisms and the acoustic receiving apparatus, culminating in the organ of Corti, have evolved *pari passu* to control spatial and temporal displacements of the individual, and of the environment, material or social. Unless this important psychological function of the respiratory apparatus is kept in mind no adequate dynamic pathology of its various parts is possible. The laryngeal and pharyngeal aspects, as well as the hypoglossal, trigeminal, and facial nerve component activities are discussed in other chapters. Here only certain features of the lung structure itself are under partial inspection.

The tone of the bronchial musculature, the secretion of mucus, the permeability of the vessel walls of the bloodvessels are all held in physiological balance through the reciprocal action of parasympathetic

[1] Guth, E.: Lungentuberculose u. vegetatives Nervensystem, I, II, III, IV; Beitr. z. Klinik. d. Tuberkulose, **53**, **54**, **55**, Pottenger: Tubercolosis; Pottenger: Symptoms of Visceral Disease, 3d ed., 1925.

and sympathetic fibers. Increased spasm, diminished mucus, contracted bloodvessels, stand in more or less opposing relations to relaxation, increased secretion, or tendency to exudative phenomena. Whether the stimuli are bacterial toxins, toxic gases, emotional states, hormone stimuli, these fundamental components are operative. Thus localized respiratory sympathicotonic and vagotonic trends are recognizable, the more detailed analysis of which is necessary in pulmonary pathology.

The parasympathetic fibers to the pulmonary structures make their synaptic connections in the dorsal vagus (visceral) nucleus. The effective cells lie within the bronchi. The sympathetic arcs utilize the upper (5,6) thoracic cord segments, the stellate ganglion and the plexus pulmonalis. Both plexi course along the chief vascular channels. The pulmonary cells themselves have vegetative nerve terminals.

Pulmonary Visceromotor and Viscerosensory Reflexes.—Among the signs of acute and chiefly chronic pulmonary disease (chronic bronchitis, tuberculosis) certain hypertonic muscular states, as well as painful areas, may be present. These muscles of the anterior or posterior walls of the chest are found on careful palpation to be slightly hypertonic—corresponding to the reflex arc area of the involved chest region. This reflex is mediated through the vegetative system, and therapeutically can be made use of by means of external stimulation (mustard plaster, etc.) to influence internal, vascular, glandular and muscle fiber states. The viscerosensory reflex shows itself as sensations of cold, heat, pain, etc., on the skin corresponding to the affected area within. Trophic reflex arcs also follow closely related localizations.

Pottenger[1] has carefully studied these vegetative reflex pathways and has been one of the first to approach the pathology of tuberculosis by an essentially dynamic pathway. No complete understanding of this important disease will be reached, however, until the patients are also studied from the standpoint of the respiratory libido. That is, How are the lungs being utilized in their symbolic function through speech? This psychical component is an important measure of the delivery capacity of the human machine.[2] The symbols utilized as expressive of the individuals creative strivings find their formulation through the speech mechanism. Even when written outlets are chiefly used, as in literature, the cortical speech zones incorporate lung activities—thought being but modified action from one viewpoint. The study of tuberculosis as of many other pulmonary affections from this angle promises much.

The pulmonary structures are frequently utilized for a number of symbolic activities. Conversion hysterical syndromes are frequent.

[1] See Pottenger: Clinical Tuberculosis, 1917, Mosby, St. Louis.

[2] Jelliffe: The Symbol as an Energy Container, Jour. Nerv. and Ment. Dis., December, 1919, vol. **50**. Psychoanalysis and the Drama, Nervous and Mental Disease Monograph Series, New York and Washington, 1922, vol. **34**. Also Psychotherapy and Tuberculosis, Am. Rev. Tuberculosis, 1919, vol. **3**. See Jelliffe, Postencephalitic Respiratory Syndromes, Nerv. and Ment. Dis., Monograph No. 45, also Jour. Nerv. and Ment. Dis., 1926; Turner and Critchley: Postencephalitic Respiratory Disorders, Brain, 1926.

These show as mutisms in the laryngeal structures; various types of cough, clearing the throat, hoarsenesses, voice timbers, stutterings, stammerings, barkings, etc. Certain quite severe peribronchitic scleroses, with emphysema may be traced to severe and almost unconquerable compulsive neurotic compensatory reactions. Some may even lead to death. After many years of vicious maladjustment the lung structures become so altered (sympathetic fibrosis) as to be unalterable. The so-called hyperthyroidism of tuberculosis receives much illumination when seen from the standpoint of unconscious symbolizations.

In certain severe toxic states, such as those resulting from influenza, a marked sympathetic paralysis with parasympathetic stimulus leads to an excessive vagotonic reaction—pulmonary flooding—and death. Not infrequently this type of individual shows other signs of vagotonia. Eczemas, hypomanic trends, etc.[1]

Asthmatic attacks, with spasm of the bronchi, difficulty in breathing, slowing of respiratory phases, emphysema and eosinophilic sputum, are types of increased vagotonia, relieved in part by adrenalin or ephedrin. Here the exciting causes may also lie at any of three levels. Physico-chemical (parathyroid with tetany, calcium metabolism); sensorimotor (from pressure phenomena on laryngeal and bronchial nerves, reflexes from nose), or psychical (emotions, erotic frustration, repressed sexuality). The problem in treating asthma is therefore to find which nervous system level is chiefly implicated. Psychoanalysis would be folly for those asthmatic attacks which are due, for instance, to cheesy, tuberculous deposits pressing upon nerve structures in the posterior mediastinum, while it alone would remedy those asthmas that are of psychical origin solely. Combined therapy—interrelational—is of greatest value.

Cheyne-Stokes respiration is found in a great variety of pathological states such as high cervical myelitis, medullary and basal, epidemic encephalitis[2] hemorrhage, midbrain tumors and occasionally in certain cortical atrophies or hemorrhages. It is present rarely, in certain hysterics.

Vascular Apparatus.—Only the briefest outline is possible, although the study of the cardiac activities lies mostly in the vegetative neurological field.

Heart.—Sympathetic, parasympathetic and intraganglionic mechanisms are present. The sympathetic pathways arise from D 1-5. Wrisberg's ganglion is the first synapse, the postganglionic fibers passing to the heart musculature. The vagus is active through three main branches one arising below the superior laryngeal, a second from the recurrens, a third from the thoracic part of the vagus. The deeper layers of the heart are supplied through the right vagus, the superficial cardiac

[1] Jelliffe: Nervous Complications of Influenza, Crookshank, London, 1922. Renaud. La mort dans la Grippe, et son mechanisme, Annales de Méd., 1919, **6**, No. 4. Asher: Die Physiologie der Atmung, Schw. med. Wchnschr., January 5, 1922.

[2] See rich literature on Epidemic Encephalitis particularly Stern, Wimmer, Hall, Goldflam, von Economo, Achard, etc.

plexus supplying through the left. The sympathetic fibers which accelerate the heart's action are in relation with the extracardiac ganglia, the end branches of the vagus in connection with the intracardial ganglion, *i. e.*, cells. The intracardial cells are here assumed

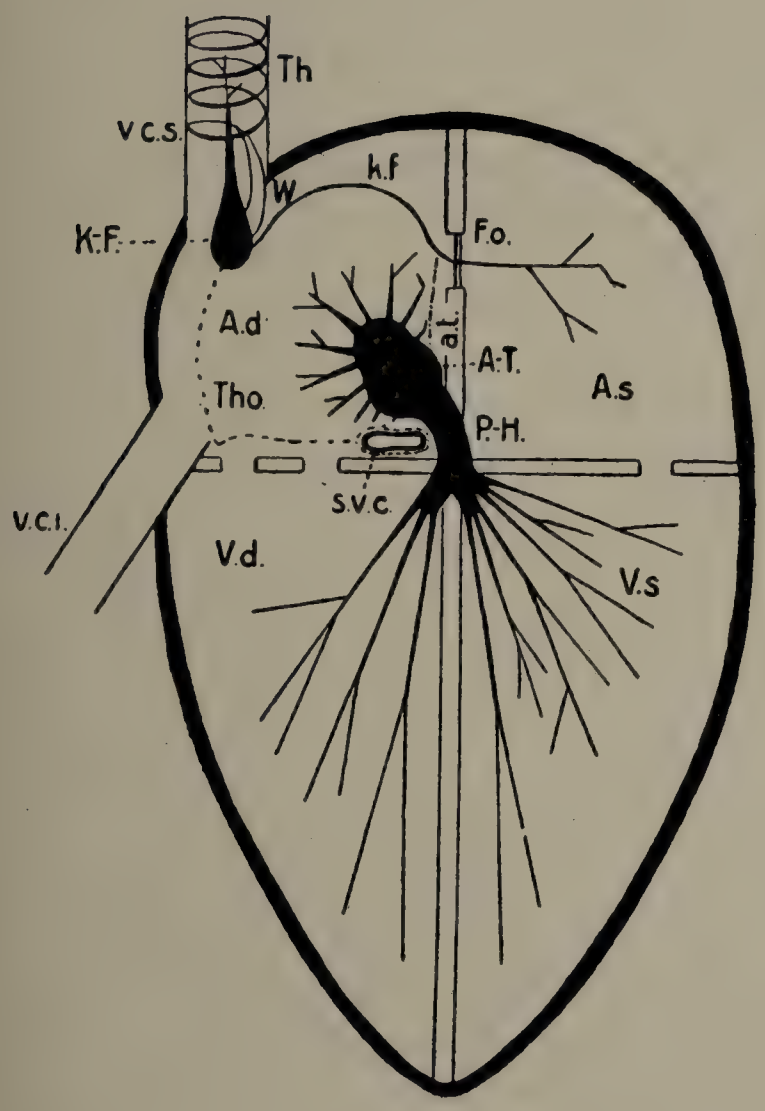


FIG. 61.—Scheme of cardiac innervation: *A.d*, right auricle; *A.s*, left auricle; *V.d*, right ventricle; *V.s*, left ventricle; *v.c.s*, and *v.c.i*, superior and inferior vena cava; *s.v.c*, sinus venosus; *F.o*, foramen ovale; *K.F.*, Keith-Flack sinus node; *A.T*, Aschoff-Tawara auriculoventricular node; *Th*, Thorel's superior vena cava bundle; *Tho*, Thorel's intermediary node bundle; *W*. Wenckenbach's auriculovenous bundle; *k.f.*, Keith-Flack fibers between *K.F.* and *F.o.*, *a. t.*, Aschoff-Tawara bundle between *A.t.* and *F.o.*, *P.H.* Paladino-His bundle. (Janowski.)

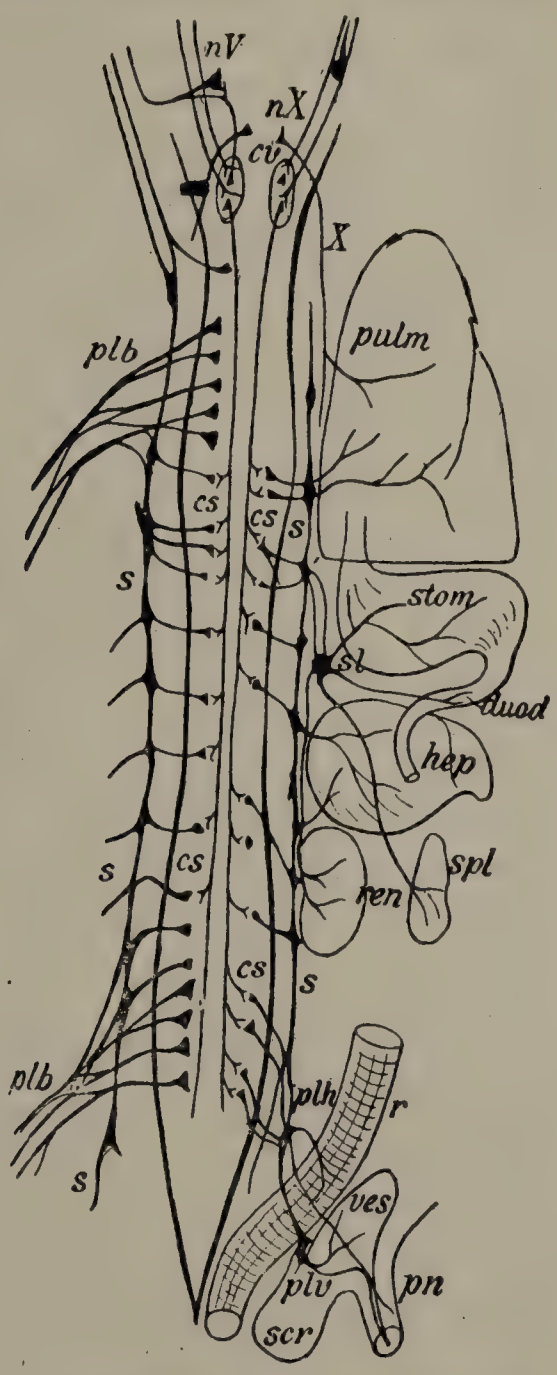


FIG. 62.—Scheme of innervation of the vasomotors: *cv*, main center of vasomotors in the medulla; *cs*, spinal vasomotor centers; *s*, sympathetic; *nX*, vagus nucleus; *X*, vagus; *nV*, trigeminus nucleus; *plb*, brachial plexus; *plv*, vesical plexus; *pulm*, lungs; *stom*, stomach; *duod*, duodenum; *hep*, liver; *spl*, spleen; *ren*, kidney; *r*, rectum; *ves*, bladder; *scr*, scrotum; *pn*, penis. (After Langley.)

to be visceromotor, and are thickest at the origin of His's bundle, Tawara's nodes and at the origin of the Keith-Flack bundle. The activity of the vagus upon the muscles seems to manifest itself chiefly through the ganglionic net work. "Reizleitungs system."[1]

[1] Consult Nomura: Function of Reizleitungssystem in the Heart, Heart, 1923, **10**, No. 4.

The gray matter of the midbrain in the neighborhood of the floor of the third ventricle is thought to be a higher coördinating switchboard—the nucleus dorsalis vagi, an end station. Through this portion of the mechanism, psychical influences are switched in, modifying the tonus through emotions, pain and local stimuli.

Bradycardia appears through a number of influences, chiefly following acute infectious intoxications, by increased intracranial pressure, in hypothyroidism, digitalis and allied glucosidal actions. Trigeminal reflexes through the nose, eyes (pressure) may also cause bradycardia. The various arrhythmias, dislocations, and blocks cannot be discussed here.

The relation of changes in or due to His's bundle cannot be entered into here, although they may properly be discussed in a text-book on neurology.

Angina pectoris, in some of its forms at least, is due to parasympathetic overestimulation whereby vascular cramp states are brought about; vagus paralyzing and vasodilator drugs therefore aid in overcoming the condition, particularly in the vasomotor types of angina.[1] Cardiac discomfort so frequent in visceral heart disease, as well as in psychical disorders is carried to consciousness chiefly through communicating sympathetic branches through the spinal ganglia, or directly to the spinal systems. Head's hyperalgesias are explained in this manner. The vagus (parasympathetic) fibers are not implicated. They are involved in the cardiac crises of tabes.

In the anxiety neurosis, cardiac disturbances are extremely frequent. Pseudoangina pectoris is frequently a symptom of this state, the further elucidation of which will be found in later chapters. Neurotic cardiac disturbances are frequent. In the great World War many prospective soldiers developed cardiac irregularities. Many showed extrasystoles which graphic tracings separated from heart-block. A systolic murmur at the apex growing louder on exertion with a positive Wassermann will likely prove a precursor of angina pectoris. The use of adrenalin and a study of the blood will aid in the clinical diagnosis of organic cardiac difficulties. Thus increased adrenalin mydriasis and positive lymphocytosis with cardiac irregularity is usually organic from a dysthyroid state. In determining military capacity the test is valuable.

Bloodvessels: Vasomotor Neuroses.—The anatomy, physiology and clinical disturbances of the bloodvessels make a large chapter in contemporary neurology. Cassirer has devoted a monograph of 1000 pages alone to their consideration. Only the briefest sketch is offered here.

The bloodvessels of the face are innervated from the upper cervical sympathetic fibers passing over the internal carotid plexus to the Gasserian ganglion, and with the pathways to the sweat glands pass with the sensory fibers of the face. Those of the upper extremities are supplied chiefly from C5 to D7, mostly leaving by way of the D3 to D7 root segments. Those of the lower extremities arise from D1–2

[1] Consult Spiegel on the surgical treatment of angina pectoris, Wien. kl. Wchnschr., 1927, p. 853.

to L3. In the spinal axis are located only the local segmental functions. Bulbar centers are present in the nucleus dorsalis vagi, which is a parasympathetic synapse zone for peripheral vessels as well as those of the intestines. Stimuli in the bulbar centers tend to cause contraction of the peripheral vessels and dilatation of the visceral ones. Intracranial bloodvessels have vasodilator and vasoconstrictor fibers conveyed through the cervical sympathetics.

Cortical centers have been placed in the frontal areas (Lewandowsky, Weber; denied by Müller and Glaser, who claim the midbrain as the highest center), from which the pathways pass through the internal capsule, caudate nucleus, thalamus, hypothalamus, pons, central gray of third ventricle, oblongata, Helweg's triangular bundle, anterolateral bundle to lateral horns—the fibers crossing in the posterior commissure (Helweg). The parasympathetic and sympathetic fibers apparently follow different pathways in the cord, the vasodilator parasympathetic pathways following the course of the sensory roots, the vasoconstrictor sympathetic by way of the anterior roots, the motor nerves and the sympathetic ganglion. Thus irritation of the posterior roots causes hyperemia (vasodilatation) with pain, paralysis of the same causes anemia with anesthesia.

Within the bloodvessels themselves ganglion cells are found, save perhaps in those whose vasomotors run in the spinal nerves (Müller and Glaser), and reflexes occur here exactly as in all of the skin and tendon reflexes from terminal stimuli. Hence an analysis of vascular disturbances must include a study of the sensory, motor and central portions of the reflex arc—the last including both medullary and corticospinal reflex pathways.

Disturbances of the peripheral mechanisms of the vasomotor pathways have been more completely analyzed than those resulting from lesions in the spinal, bulbar, thalamic, or cortical portions of the same. Of these, more detailed mention may be made of the: (1) tonic hyperemias (erythromelalgia), (2) spastic anemias (pseudosclerosis, Raynaud's disease, migraine, intermittent claudication), and (3) vasomotor irritability, as in acute angioneurotic edema, multiple gangrene of the skin, etc.

Arteriosclerosis as a disease could be discussed here but space does not permit.[1]

Physiological alterations in the tonus of the peripheral vessels are seen in sleep, emotional states, active digestion, overexercise, overheating, in collapse and in toxic and fatigue states.

1. Tonic Hyperemias.

These consist in prolonged irritability of the peripheral vasomotors. In certain individuals (sympathicotonic) a diminished alkalinity of the

[1] Staemmler, M.: Zur Pathologie d. Sympathischen Nervensystem, Ueber seine Bedeutung f. d. Entstehung d. Arteriosklerose, Beit. z. path. Anat. u. z. allg. Path. 1923, **71**, 388.

blood is thought to bring about such a stimulation of the sympathetics. These tonic hyperemias are seen more particularly in neuralgias, neuritides, in infections, or toxic erythemas, and reach a pronounced grade in the syndrome known as erythromelalgia. Thymic persistence should always be inquired into.

Erythromelalgia.—Two main trends may be distinguished—those with pain, as described by Weir Mitchell, and those without pain but with hyperidrosis and hyperalgesia (Hess).

Graves as early as 1843 gave a recognizable description and offered useful interpretative generalizations. Paget, 1871, W. Mitchell, 1872, Grenier, Sigerson, 1873, Vulpian, 1874, described certain details, but it was Weir Mitchell who in 1878, made a definite synthesis of this paroxysmal disorder of the extremities which was marked by a painful redness and swelling of the feet. Lannois, in 1880, wrote an important monograph on the subject, and Cassirer, in the second edition of his *Vasomotorischtrophischen Neurosen,* 1912, has given a complete description of the general group to which the name erythromelalgias may be given. He was able to gather reports of about 130 cases. One may conclude it to be rare. Only 2 in Oppenheim's 25,000 dispensary patients are reported, while in Jelliffe's statistics of Starr's dispensary service of 18,000 patients 21 were observed, 15 in males and 6 in females. It is more often observed in the later years of life, although six- to ten-year-old (Baginsky) patients are recorded.

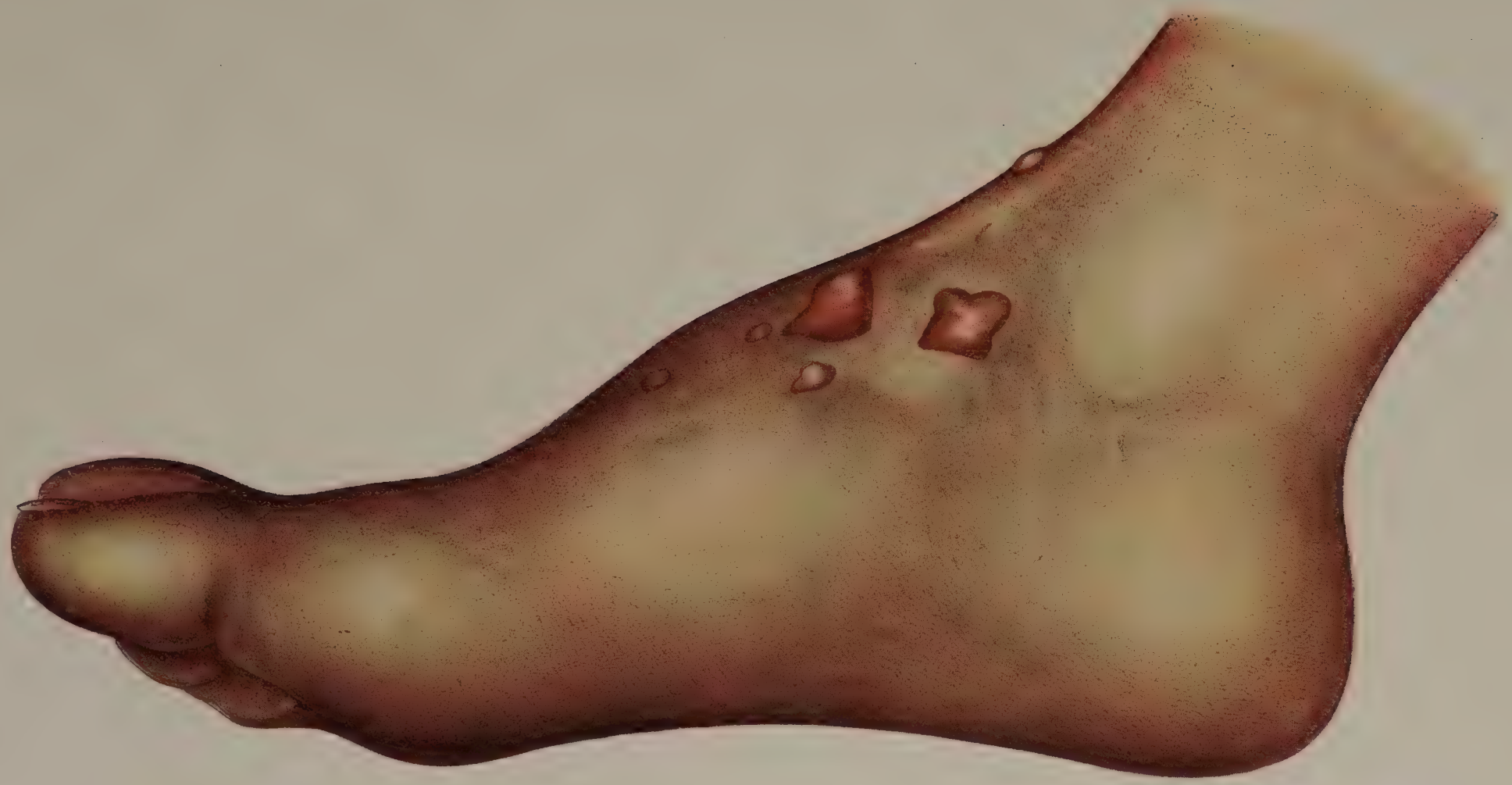
Causes are difficult to run down. Thermic influences apparently play some role as exciting agents at most. Psychical factors may determine an attack.

Hypothetically erythromelalgia is a pure sympathetic affection, an angioneurosis, due to prolonged sympathetic stimulation.[1] Practically it shows itself in combined forms, being an accompaniment of spinal disorder (involving the sympathetic cell groups) in multiple sclerosis, and in tabes; it may occasionally be seen in cerebral disorders, hemiplegia, thalamic involvement, or may be a part of a peripheral nerve disorder, accompanying a neuritis, or it may be a cause of or a part of a chronic vascular disease of an obliterating or spasmodic type. In each of these the chief action is directed upon the sympathetics. Thus a number of gradations and variants exist which are discussed in the works already cited, particularly in Cassirer and Oppenheim.

Symptoms.—The chief symptoms are heat, redness and pain in the extremities, either localized along a definite nerve distribution, often following a root area, or peripheral. It is usually intermittent, worse at night, and the patient suffers tortures. Heat usually makes it worse, so also does movement, especially walking, whereas any position reducing passive congestion, thus overcoming the tonic hyperemia, affords relief. Severe grades of the disorder show a purple cyanotic skin, with erythema, usually due to transudation following stasis from slowed circulation in dilatated area. Hyperidrosis may be present.

[1] May et Hillemand: L'Erythromelalgie, Ann. Med., 1924, **16**, 51 (with literature).

PLATE V



Erythromelalgia. Foot after hanging down for twenty minutes. (Mitchell.)

(Case of Dr. J. M. Taylor.)

Accessory symptoms, such as headache, palpitation, and fainting are reactions to the pain, and in part to the fear, or may be another facet of a psychoneuroses in which the erythromelalgia is also a symptom. Trophic changes in the skin, hair and nails may take place which are either a part of the sympathetic disturbance itself or are results common to the angioneurosis, and a producing or accompanying lesion—tabes, multiple sclerosis, paresis, etc.

Course and Therapy.—The outcome depends much upon the causation. An erythromelalgia due to spinal changes may get better if these do (syphilis) or not (tumor, multiple sclerosis). The therapy will be determined by the cause. Palliatives, such as the use of high frequency current, violet rays, cold, antipyrin, are valuable as well.

A neuritic erythromelalgia will improve or not as the neuritis does; similarly an arteriosclerotic one; but it usually gets worse. The therapy is for the more fundamental condition. Leriche has had some good results with sympathectomy operations, but the evidence of later years is against it. Opotherapy (ovary and thyroid) has helped some cases (Laignel-Lavastine). A psychically determined erythromelalgia, possibly a conversion hysteria, needs psychoanalysis.

2. Spastic Anemic Group.

Here the chief results are due to persistent or intermittent vasoconstriction. The syndromes are numerous and confusing, but among them a few are sufficiently distinct or constant to be given diagnostic titles such as Raynaud's disease, intermittent claudication, acroparesthesia, migraine, pseudosclerosis, asphygmia alternans, etc. Only the chief types can be taken up; the purely tentative nature of the classification must be emphasized.

Certain of these cases are unquestionably related to underlying endocrinopathies (thyroid, adrenal), others are primary and secondary neuritic syndromes, or are related to anatomical changes involving the sympathetic synapses in the lateral horns of the cord, syringomyelia, poliomyelitis, often overlooked if slight attack, multiple sclerosis, spinal syphilis, etc., others are exclusively psychogenic, chiefly hysterical conversions, or schizophrenic defense substitutions.

Raynaud's Disease.—Raynaud's disease is also known as symmetrical gangrene, local asphyxia. This syndrome, like the preceding, one, may be of many origins. It may be psychical (shock, hysteria, schizophrenia), cerebral, or spinal organic (capsular (thalamic) hemorrhage, trauma, paresis, multiple sclerosis, tabes, syringomyelia, tumors) or peripheral in nerve or bloodvessel, neuritis of all various etiologies, mild endocrinopathies, arteriosclerosis either peripheral or of the large vessels (aorta).

It may readily be seen that from such a polyetiological viewpoint, there is no single Raynaud's disorder. Hence Cassirer's attempt to make true sympathetic types and those due to complicating disorders, such as the local gangrenes due to diabetic neuritis, or to arterio-

sclerosis, etc. Even this is difficult to accomplish. Thus a spinal gliosis (syringomyelobulbia) may invade the sympathetic cells in a given segment and bring about a local paroxysmal gangrene. This is truly a vegetative affection, but it is to be considered as the very beginning symptom of the syringomyelia. Some patients after a mild poliomyelitis, *i. e.*, mild so far as motor involvement is concerned, show marked vascular instability and develop Raynaud's syndrome very readily from a variety of disturbing factors—cold, alcohol, fright, etc.



FIG. 63.



FIG. 64.

FIGS. 63 and 64.—Raynaud's disease, showing superficial gangrene. (Osler.)

Symptoms.—The attacks are paroxysmal. The fingers or toes begin to get cold, and have the feeling of prickling and of "going asleep." They become pale and waxy from the vasoconstriction. Pain is frequently felt and local coldness is present. An attack of this kind may come and go in a few hours.

More persistent attacks lead to more marked grades of local asphyxia, with cyanosis, or bluish-red discoloration of the extremities. Pain is extreme. Vesicles may form—the fingers may even get bluish-black—and gradually disappear after a few days, or leave slowly healing, broken vesicles, or more deeply lying trophic ulcers (protopathic nerve fiber injury). Other types of sensibility also suffer. Epicritic touch and thermal as well as protopathic pain, thermal and deep sensibility, may also be involved. Gangrene is a severe grade with loss of fingers or finger-tips.

Accessory symptoms (such as trophic changes in the nails, in hair, in the bones, etc.), which are due to the different etiological factors, syringomyelia, neuritis, arteriosclerosis, etc., need not be entered into. Attacks, with recovery, may persist as long as three or four months.

Treatment.—The therapy is often without avail, as the underlying condition is unmodifiable (syringomyelia, multiple sclerosis, etc.). As a rule, however, the attack subsides, although to appear again. Then attention should be addressed to the general health of the patient, especially to emotional features which produce vascular instability. Mild massage, local warmth and Bier's hyperemic treatment are of value during the attack, strong analgesics being necessary for the pain at the time. Endocrinopathic cases do well with thyroid. In psychogenic cases psychotherapy is alone available.

Intermittent Claudication.—This is an angiospastic syndrome and rests upon a number of foundations. Clinically it consists of a spastic vascular state with weakness, pain, and coldness in the affected region. In the majority of cases it appears in the leg or legs. After the patient has walked, perhaps rapidly, the leg or legs begin to be fatigued, and commence to feel numb and painful until it is impossible to keep up the pace or walk at all. After a rest the patient may resume his walk for a time free from distress, but the state of pain and fatigue recurs to be again relieved following rest. There is a later tendency for the state to recur when the limbs are at rest. Cyanosis, coldness, paleness are accompanying phenomena. There is mild hyperesthesia of the affected part but no other sensory signs. The chief vessels may be pulseless. These should be tested by touch and the eye aided by the sphygmograph.

The chief sites are the vessels of the legs but the arms may be involved. Any muscular group may show the symptoms. Lumbago-like forms occur in the back muscles. The vessels of the intestines, internal organs, brain, and spinal cord may be involved.

The chief lesion is arteriosclerosis, but others are operative. The arteriosclerosis itself may be secondary to syphilis, alcoholism, to chronic nicotine poisoning.[1]

Oppenheim has called attention to the frequency with which these arterial changes are found in Russian Jews. This disorder is mostly confined to the men of this people. Here flat-foot probably plays a

[1] Frankl-Hochwart, Deutsch. Zeit. f. Nervenheilk., 1913, vols. xlvii and xlviii.

role—excess in walking (peddlers) may aid. Psychoneurotic factors also may play a part in the causation of these arterial cramps independent of any definite arteriosclerosis. The complicated question of altered chemism within the vessel walls cannot be entered into. Hereditarily inferior vascular systems are factors.

Therapy.—Rest, warm applications to the parts, and high-frequency current application are of value in treating the attack in its acute stage. Treatment of the condition rests upon the proper conception of the individual provocative disorder. Arteriosclerotic cases need treatment for this; psychoneurotics require psychotherapy. Of the more fundamental therapy of the vegetative system which permits the spasticities as well as modifies the calcium metabolism in the vascular walls nothing as yet can be laid down.

Ophthalmic Migraine.—This is also known as sick headache; megrims; hemicrania; bilious headache.

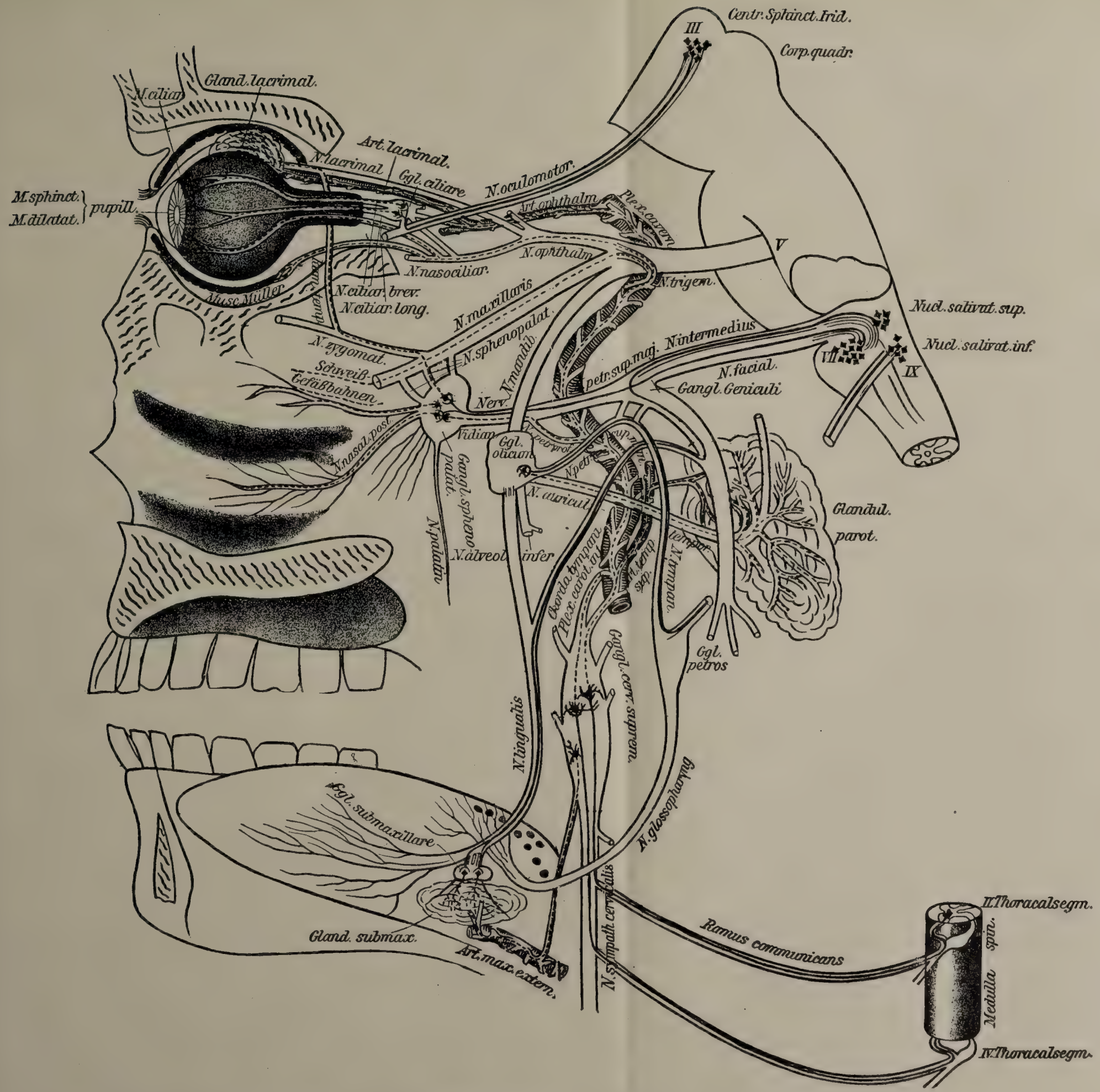
This protean affection is difficult to define. It may be a simple or an extremely complex condition. Migraine may, however, be defined as a periodical disordered state in which the patient suffers from a peculiar oppressive pain in the head, unilateral or bilateral, localized or general, which develops very gradually from heaviness to dulness, to pain that is splitting, and is accompanied or more often preceded by characteristic visual signs, such as scotomata, flying specks, or partial blindness. Chilliness, depression, and sensory disturbances, particularly in the stomach, and which may lead to nausea or vomiting, are also usually present. An attack may be terminated, after a few minutes, by vomiting, or it may persist hours or even days. After a variable length of time, usually following a heavy sleep, the patient regains his previous condition of well-being. Nearly every one has had an attack or attacks of migraine during his life-time, hence its extended description here.

History.—A heritage of the rich and the poor, the great and the small alike, it has numbered among its sufferers many of the master minds of all times, and no disorder can vie with it in richness of description from medical writers who have been themselves subject to its vagaries.[1] Aretaeus is credited with having given the first description of migraine. Celsus gave a description which, while not corresponding in many details with what is now understood to be migraine, is nevertheless very suggestive. Caelius Aurelianus noted for the first time that the Greeks called it hemicrania. Lepois, in the seventeenth century, gave his personal experiences through fourteen years, and called attention to the fact that the usual after-effects of vomiting and sopor might come on without the presence of the headache. Wepfer in the same century seems to have more clearly appreciated the eye symptoms.

Tissot's description, 1784, remained authoritative up to the appearance of Liveing's monograph, *On Megrim, Sick Headache and Some Allied Disorders* (1873), although in the interim the symptomatology

[1] See article by Jelliffe: Osler and McCrae, Modern Medicine, 1928, vol. 6.

PLATE VI



Schematic Representation of the Vegetative Nerve Innervation of the Head. (Müller & Dahl.)

was becoming richer and the case analyses more exhaustive. Thus, Vater, Hennicke, and Heberden made observations upon the scotomata. Plenck, Parry, Wollaston drew from personal experiences the picture of half-sided blindness. Schönlein and Romberg introduced the neuralgic theories, while Dubois-Reymond, influenced by the newer work of Claude Bernard, developed the hypothesis of arterial spasm which Möllendorf controverted, and postulated a sympathetic paralysis, both of which views were conciliated by Jaccoud and by Eulenberg (1867), who described angiotonic and angioparalytic conditions.

Etiology.—Migraine is here considered as a vasomotor disturbance due to a variety of stimuli acting upon the vegetative nervous system. These stimuli may be physical, as seen in attacks following severe blows, falls, fast movements, sudden alteration in temperature, of pressure—high mountains, caisson, deep diving, lumbar puncture, etc. They may be of chemical origin, nicotine, alcohol, endocrinological (adrenalin, thyroid), morphine, protein sensitization, toxic substances from various sources. They are infrequently of somatic reflex character, fatigue, neuritic, tumor formations, meningitis, etc. They may be emotional, great anger, fear (which may act by producing metabolism disorders—acidosis), disappointment, chagrin, which psychical stimuli may be conscious or unconscious. One or more exciting factors may coöperate. Those mediating in the cerebral sympathetic nervous system cause vasomotor spasms and paralyses with hyperemia and pressure in the brain substance and cerebral ventricles usually of a temporary and transient nature. At times the pressure produces persistent or more or less persistent sequelæ, such as ophthalmoplegia, hemianopsia, hemiplegia, aphasia, optic nerve lesions, etc.

Milder Attacks.—Incomplete or abortive attacks may be said to be the rule rather than the exception and attempts to classify the disorder according to the number of symptoms present offer no help in the understanding of the complete picture.

Möbius suggested that the parents of patients suffering from migraine with scotomata often have suffered from migraine without scotomata, but he also speaks of the reverse as happening. The extreme prevalence of migraine makes many of the conceptions regarding its necessary hereditary nature very dubious, and the extreme variability of the individual attacks in the same patient makes general hereditary features extremely improbable. It is by no means infrequent to find patients that show at one time or another almost every symptom mentioned in the voluminous literature of migraine. Thus one patient under personal observation had about two attacks weekly for a year. He then went two years without a single attack, and he then had several severe ones with aphasia and psychical symptoms, interspersed with abortive attacks, with hardly any two alike. He was a veritable museum of migraine attacks in the fifteen years that he was under observation. He consulted scores of physicians and had hundreds of prescriptions. He had but few attacks after a happy marriage.

Many families are known in which both parents have been sufferers from chronic migraine for years, and yet none of the children, now in some instances over forty years of age, have ever had more than one or two attacks. The high percentage of incidence makes it almost impossible to calculate a hereditary factor. Again, it may be borne in mind that as there are many kinds of epilepsies, so also there are undoubtedly many migraines. Some may be constitutional while others have nothing to do with anything of a hereditary character. Thus, one can speak of migraines that are possibly hereditary but this conveys no information whatever.

The commonest milder attacks are those that begin in the classical manner, with chilliness, perhaps with pinched face, and cold extremities. The patient then has the scotomata and wretchedness, depression and apprehension, and then while waiting for the headache he notices that it does not come, and, although he may still have heaviness and a sense of discomfort, the feeling of relief is sufficient to make him feel well.

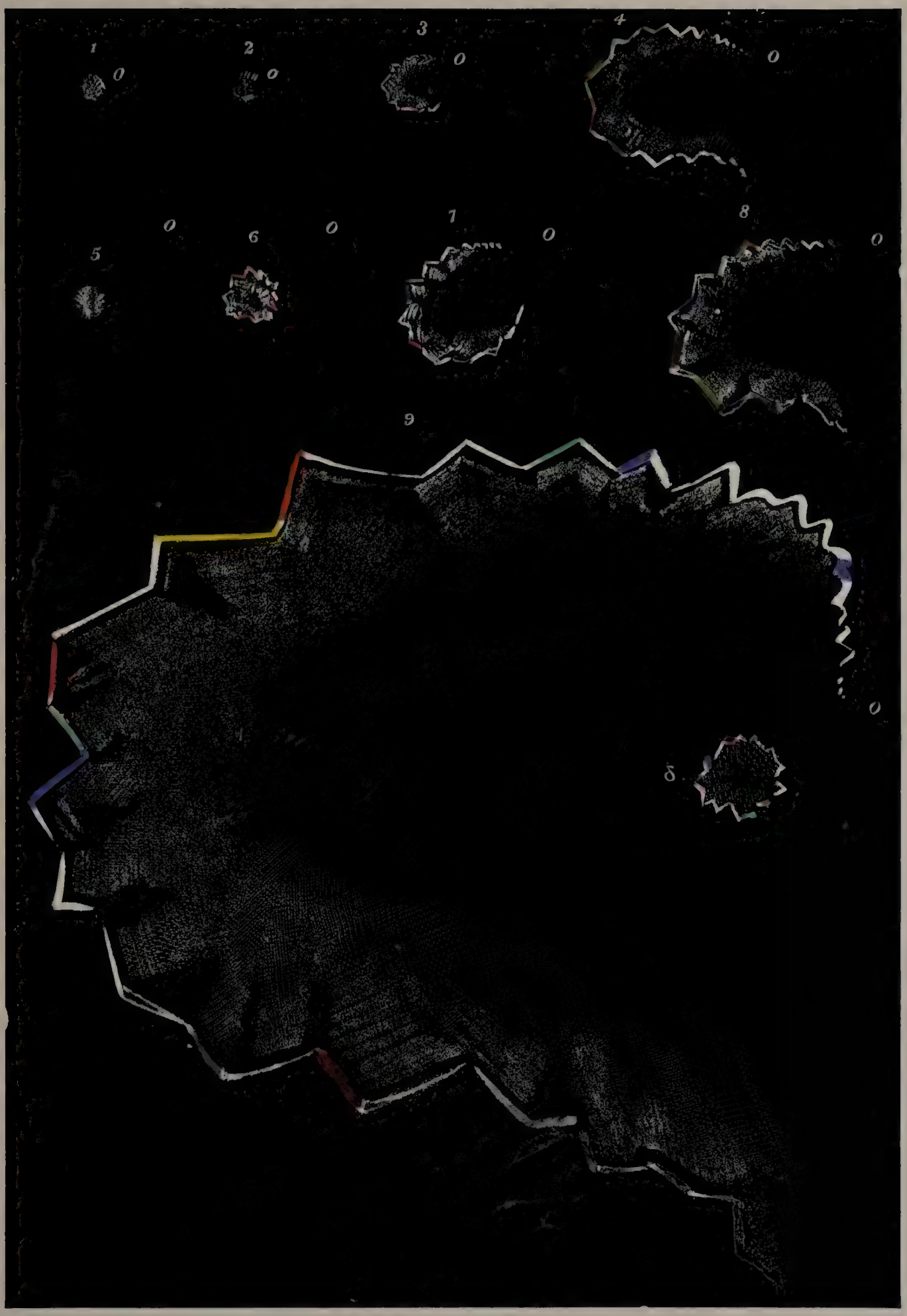
Others have added the sensation of prickling in the fingers, numbness in the hand or arm, or other sensory disturbances without the headache. In some the entire attack will consist of a disturbed painful sense of discomfort, without sensory symptoms, scotomata, or headache, but they feel sick at the stomach, and have an attack of what old fashioned medicine called "biliousness," which clears up after vomiting. This feeling will recur with sufficient frequency, and at times be combined with such other symptoms of a migraine attack, in its varying aspects, as to stamp the whole process as a variant of a severe attack. Isolated attacks of vomiting as the sole expression of a migraine are known.

Attacks of scotomata occur alone, without antecedent distress, and no after-attacks are noted. These are not uncommon. Historically it may be noted that Parry and Airy had such attacks. It is highly probable that the majority of patients who have had many migraine attacks will have had some of this nature. Attacks of scotomata and vomiting occur without headache. In many on the contrary headache is the only symptom.

Some patients have attacks of hemiparesthesia with no other symptoms of migraine. These generally occur at night, and usually follow severe mental exertion; in one patient under observation a severe ordeal in playing a difficult piece of music would bring on such an attack without other signs. This patient's severe attacks were very extreme, being associated with hemiedema, hemiparesis, hemianesthesia and marked hysteromaniacal outbursts.

Under the heading of equivalents, Liveing speaks of stomach attacks associated with some of the vascular phenomena of migraine; glossal spasms are also mentioned by him. Attacks of giddiness, vertigo, intestinal colic, mental anxiety and depression which occur periodically in partial association with migraine symptoms, also have been noted as equivalents. There is need of further study of these isolated phenomena associated with vasomotor disturbances.

PLATE VII



From Dr. Hubert Airy's Paper on a Distinct Form of Transient Hemiopsia. Philosophical Transactions, 1870, p. 247.

Figs. 1 to 4.—Early stages of Fortification Spectra as seen in dark. O = sight point.
Figs. 5 to 8—Similar series, beginning lower.
Fig. 9.—Fully developed. ϑ = secondary attack within.

Attempts have been made to determine the relative frequency of migraine attacks with and without the visual signs. These are not overreliable, because of the vast preponderance of abortive attacks over those of the complete classical type.

Möbius expresses the opinion that the percentage of visual accompaniments of the attacks is usually overstated. His statistics show 130 cases, with 14 visual aura. In Liveing's 60 patients, 37 suffered from scotomata. Gowers says that the cases are about half and half, with and without eye signs. Galezowski maintains that the visual aura migraines appear later in life, thirty to fifty years, than ordinary migraines.

It is difficult to state an individual position, the results of personal inquiries having been so diverse. Close questioning has revealed the fact that at some time or other in the course of the disease the majority of patients have had visual symptoms, and it is not improbable that the usual statistics are largely derived from studies of too few attacks, *i. e.*, largely from the severer attacks only. Some notes on individual histories are of interest. Several patients have kept fairly accurate records of their migraine attacks for several years. One shows 168 attacks in a period of about ten years; of these, about 100 were milder attacks, the vast majority of which, 60 per cent., consisted of scotomata alone. Of the 68 remaining attacks, about 50 per cent. were ordinary hemicrania, lateral or bilateral, without scotomata, the others ophthalmic migraine, usually unilateral and with scotomata. Not one of the attacks was ever accompanied by vomiting. Two were associated with aphasia, 15 with sensory tactile associations; there were 5 or 6 attacks of hemiparesthesia, 1 in the daytime, the rest at night. Spasms of the orbicularis were a common accompaniment. Every attack sufficiently severe to require an analgesic was promptly relieved by from 5 to 10 grains of either antipyrin, acetanilid, or phenacetin.

Severe Migraine.—*Early Symptoms.*—These may be termed precursors of a full attack of migraine, or they may constitute the symptoms of an abortive attack. The most striking are a sense of heaviness, with yawning, chilliness, dizziness, or depression, motor twitching, even sharp spasmodic closure of the eyelids, sensory phenomena, chiefly paresthesiæ, occasionally anesthesia, and affections of the eyes or other sensory organs, ringing in the ears, blowing, whistling, modifications of taste, of smell, of touch, etc. There may be failure of appetite, constipation, diarrhea, vascular instability, hot flashes chasing here and there over the body, throbbing in the carotids, etc. The temporal arteries are often smaller, the saliva diminished, and the pupils narrowed.

The premonitory signs which show a great deal of variability in different individuals, and also in different attacks in the same individual, may be felt several minutes before the attack, in some rare instances even days. This is frequently the case in women in whom the onset of the menstrual function seems to bear some relation to the

attack. The ordinary depression felt at this time is a thing apart from this special type of depression that pervades them. At times such attacks of depression and anxiety, combined with a sense of chilliness and dizziness, will constitute the entire picture of the abortive attack. Many attacks come apparently without the slightest warning.

Many patients having attacks at night find themselves heavy and tired, with sore spots on the scalp in the morning. Möbius relates a case in which the patient dreamed of having swallowed a rabbit, which ate its way out through the stomach wall. After this unpleasant dream the patient had a severe migraine on awakening.

In all probability premonitory symptoms of some type are invariably present; when thought to be absent it is because the patient has overlooked them, either by reason of their mild character, because the symptoms appeared in a dream, as in Möbius's patient, or because of naturally poor powers of observation. Many patients, who have had headaches for years, have never noticed their one-sided localization or the well-known fortification spectra, until their attention has been directed specifically to them. Many patients will deny ever having had zig-zags of light, etc., until shown Airy's pictures, when they remember having seen such phenomena. It is because of such poor observation that many cases of true migraine are overlooked, which fact lends further support to the belief that this disorder is very much more prevalent than is usually supposed.

Sensory Symptoms.—In the more classical attacks the patient has preliminary sensory symptoms. These are spoken of by Möbius in the sense of an aura. If the term aura be used as, for instance, the term "fever" is used, there can be no objection, but if by an aura is meant a restricted phenomenon essentially related to an epileptic aura, the term should be eliminated.

A sense of coldness and chilliness is one of the commonest sensations. This is usually general, and is associated with a pale countenance, gooseflesh, perhaps clammy hands, and a sense of misery. Cases are known, and are by no means uncommon, in which the chilliness has been one-sided, and is accompanied by other phenomena involving one-half of the body, including the face, of the same side. Yawning is a common early sign.

Unilateral paresthesia is not an uncommon early sign. Many patients note a tingling or numbness in the fingers of one hand; this may spread up the arm, and in rare instances general unilateral paresthesia of a very uncomfortable nature may be present. In some instances such unilateral paresthesiæ have constituted the sole symptom of an attack, save for the heaviness and usual discomfort. Occurring at night, such attacks are often extremely wearing, keeping the patient awake. Photophobia, flow of tears, strange sounds, tinnitus, peculiar odors, queer peppery or flat tastes, may be noted.

Anesthesia is less often observed, largely because of the negative character of the symptom. When involving the face or mouth it is

complained of. Anesthesia frequently follows the tingling of the early paresthetic disturbances. Franz[1] has shown that there is a very evident decrease in the pain threshold, especially after the headache has set in.

The *visual phenomena* are the most striking, and hence held to be of the most frequent occurrence. The ease of observation in part accounts for the usually accepted opinion that they are the commonest of the early symptoms. Very few individuals have been subjected to a careful sensory examination. If more were investigated, it is probable that other slight sensory signs would be found to be equally prevalent and as evanescent. The visual signs have been described by many writers, and many illustrations have been made showing their chief characteristics. The extreme uniformity of their general character is striking, as well as the variations of the same pattern.

As a rule the patient notices a slight blurring of his vision if reading or a slight flicker of light located in one eye, to one side of the center. Closer observation reveals either a slight cloudy spot, which seems to follow the eye in reading, cutting out the after-images, or a letter or so from the center of clear vision. The slight subjective sense of difficulty in reading may precede the discovery of a scintillating spot which becomes visible on closing the eyes. Little by little this spot spreads out, usually in a crescent-like fashion. General statistics are thus far unavailable, but a special study has shown that the majority of these scotomata have begun in the left eye, are situated to the left of the middle line, with the convexity of the crescentic border to the left. As the crescent gradually grows larger, the difficulty in seeing clearly becomes more marked, especially on the periphery of the visual field. For most, the scotomata are in constant motion, flashing in its spectral zig-zag fashion, thus causing the classical name "fortification spectrum" from the play of colors, and the fortress-like "ins and outs" of the outline.

After a variable time, from five to twenty minutes, the scotomata gradually subside, or suddenly disappear, to be followed by the headache. Not infrequently the headache never comes, and the preliminary sensory phenomena of chilliness, heaviness, and scotomata constitute an abortive attack. A description of the scotomata of migraine might fill a volume. The classic of Liveing reproduces the excellent illustration of Airy's, which is here reproduced.

Occasionally the right half of the field is involved. Sometimes it is the upper half, one of Möbius's patients saying that everybody seemed headless; occasionally, it is the lower. In rare instances the patients complain of total blindness, *i. e.*, central scotomata. Berbez reports an interesting case of a ring-like scotoma—the patient, on looking at his watch, could see only the central pin where the hands were united; the figures on the dial were all obscured by the scintillating scotoma. These scotomata are usually bilateral phenomena. They may begin in one eye before appearing in the other, and be some-

[1] Amer. Jour. Physiol., 1905.

what different in the two eyes, and may disappear in one eye sooner than the other. Scotomata limited to one eye are probably rare.

The explanation of the scotomata is still a mystery.[1]

THE DEVELOPMENT OF SCOTOMATA IN MIGRAINE WHILE READING.

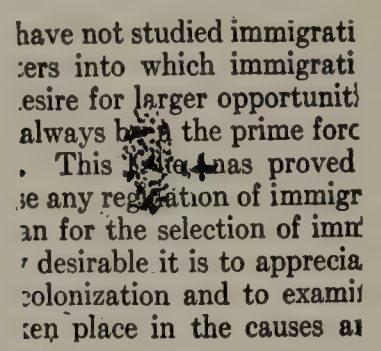


FIG. 65.—Stage of blurring three minutes.

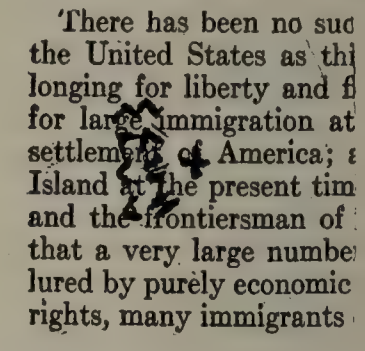


FIG. 66.—First outlines of scintillations.

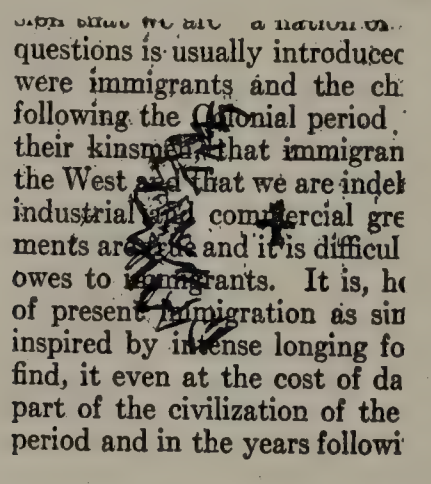


FIG. 67.—Five to ten minutes growing scotoma.

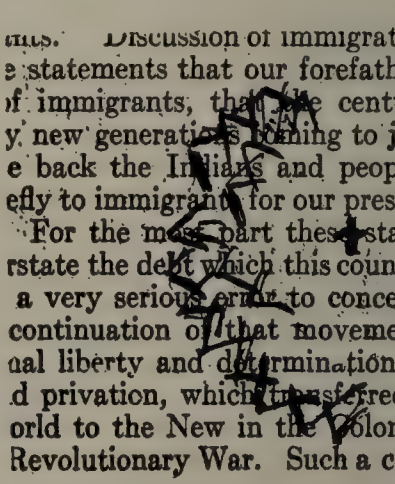


FIG. 68.—Ten to fifteen minutes.

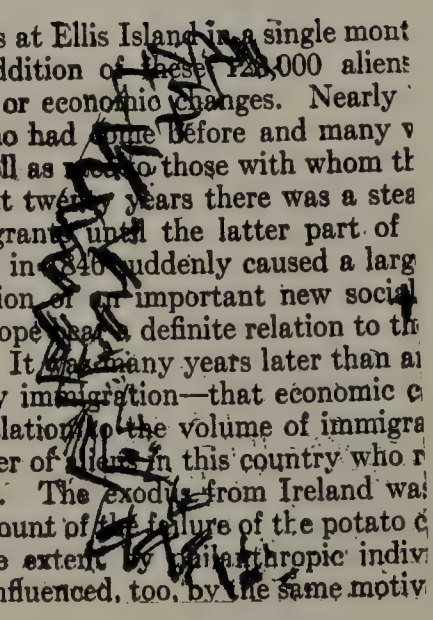


FIG. 69.—Fifteen to twenty minutes.

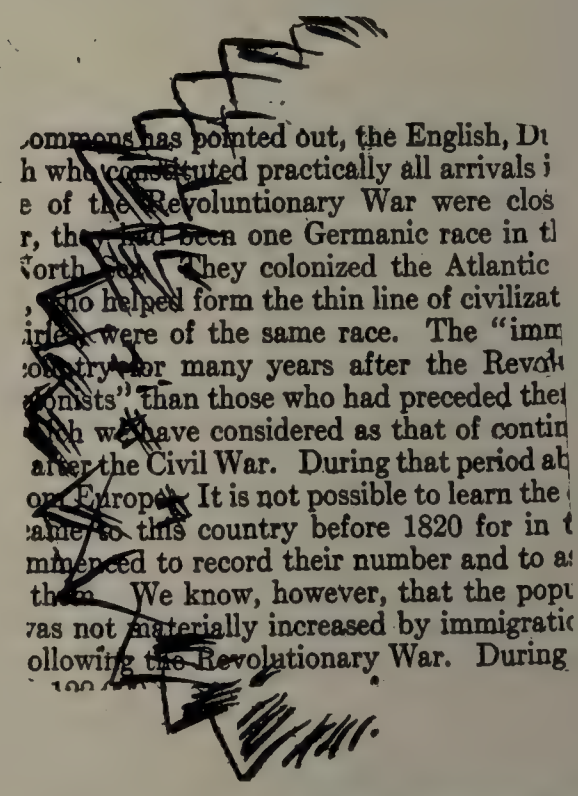


FIG. 70.—Just before disappearing, twenty to thirty minutes, and beginning of headache. (Jelliffe; personal observation.)

[1] The student interested in the why and wherefore of the scotomata is referred to a study by Klein, Zeit. f. d. g. N. u. P., 1917, **36**, 322, where he can commence his researches.

The retinal appearances during the time of these scotomata are uncertain. Blanching of the papillæ has been observed by some (Galezowski); pulsation of the retinal arteries, with dilatation by others. Personal experience has shown similar dilatation in a few cases, but, as a rule, a normal fundus is found. The picture seen will depend upon the stage of the attack and its severity.

Pupillary dilatation occurs late. Slight irregularity of the pupils during a severe attack of an ophthalmic migraine, dilatation being usual on the affected side, is not unusual. Bilateral pupillary contraction is the rule in the headache stage.

During the onset of the fortification spectra it not infrequently happens that mild motor phenomena occur in the eyelid of the side to be affected. The eyelids droop a little, and Gowers and others report double vision, interpretable as a sign of paresis in an ocular muscle.

Motor Disturbances.—Speech.—This may be considered as both a motor and sensory phenomenon, for the most frequent type of change is a transitory sensory aphasia. Anarthrias are known, especially in the ophthalmoplegic variety, but for ophthalmic migraine the type of aphasia found is very characteristic. As described by Charcot, it is an intermittent halting aphasia. At one moment the patient can get the right word, at the next he cannot. He stumbles on a word; uses madame for monsieur, etc. In Liveing's cases 15 out of 20 had speech disturbances; 1 on hearing clock bells was unable to inquire what they were. Féré cites the case of a coachman who forgot where he was going to drive his passengers; Berbez a like case in which a pedestrian lost his way, as he could not read the street signs understandingly. Gowers speaks of a case of word-deafness. Cases of agraphia are also known. Möbius reports a case with typical scintillating scotomata at one time on the right side, at another on the left. When the patient suffered from a right-sided scotoma he had sensory aphasic signs, but they were not present when the scotoma was on the left side. Schob has reported a striking case of migraine with sensory aphasia, apraxia, disturbances of touch, paresthesiæ and hemianopsia. Illusions, hallucinations, changing delusional interpretations and fierce attacks rage with clouded consciousness were also observed.[1]

Other observers have noted the same phenomena, while contradictory observations are also recorded. The speech disturbance sometimes resembles a paraphasia, the patient using a jumble of words. In a personal case the patient could not sing a well-known tune correctly, his sense of musical values having been interfered with.

The onset of the aphasic disturbance may vary greatly. It is usually temporary, persisting at times for only a few minutes, again persisting a few hours. It frequently antedates the headache, or is coincident with it. In a case reported by Meige the aphasia persisted

[1] Zeit. f. d. g. N. u. P., 1916, **35**, 151.

as long as the headache, and disappeared, as a rule, when that disappeared. The patient showed a loss of ability to say certain words and a tendency to the employment of incorrect words. There was no anarthria.

Cerebellar Symptoms.—Oppenheim has called attention to a cerebellar hemicrania in a patient in whom every attack of migraine was accompanied by typical cerebellar symptoms. The patient was uncertain in his gait, walked like a drunken man, was dizzy, and had the sensation that his body, or individual parts of it were doubled. The sense of equilibrium was disturbed in each attack. Dizziness and loss of the sense of equilibrium are not infrequent but such a complete syndrome has been described only by Oppenheim.

Paralytic Phenomena.—Attention has already been called to the rare occurrence of hemiparesis, which may even involve the facial muscles. Up to the present time no instances of crossed hemiplegic types have been found in the literature. This is of interest in connection with the hypothesis of the bulbar origin of migraine, especially of the ophthalmoplegic variety. Other palsies are known, monoplegias of the extremities, ophthalmoplegias, etc. Topical and minute brain swellings may produce a great variety of paralytic phenomena usually of a transitory nature but at times persisting.

Headache.—This is the most common feature and exhibits a great amount of variability as to location, quality, intensity, and duration. In the more classical attacks the headache begins on the average about fifteen to thirty minutes after the appearance of the scotomata or other sensory phenomena. It frequently begins on one side, and may remain so or become bilateral. As a rule it is frontal, or occupies the vertex, but may involve the temporal regions, the occiput, sometimes as low down as the neck. Gower's experience points to the parietal region as being oftenest affected, and usually over a small area. Henschen, in 123 patients, shows the pain to have been located 110 times in the forehead, 100 times in the parietal region, and 54 times in the occiput. There is usually pain over the eyes, and the eyeballs are usually painful to pressure. In a few instances pressure over the malar bones is painful, and occasionally there is a well-marked jawache.

Statistics of the percentage of different locations are uncertain since one individual will have all the different varieties. Thus, in a case already cited, in which the abortive attacks were so frequent, the headaches comparatively rare, the strictly unilateral headaches were only 5 per cent. of the entire number. In others the hemicranic type runs much higher. In Henschen's records of 123 cases, 56 had one-sided attacks, in 67 both sides were involved. In Liveing's 61 patients, 17 had one-sided attacks, in 7 the attacks were variable, while in 34 both sides were involved. Möbius and others note that the headache often appears on the side opposite to that affected by the sensory aura. Personal studies do not confirm Möbius's statement. It does seem, however, as first noted by Liveing, that one-sided sensory symptoms are oftener accompanied by one-sided than by bilateral

headaches. With bilateral sensory phenomena, scotomata, etc., bilateral pains are the commonest.

In many attacks the pains are limited to the eyes, the feeling of soreness of the eyeballs being so very marked that it is painful to move them. Pain in the neck may also cause the desire to hold the neck rigid.

The relationship of these pains to psychical symbolizations has not yet been sufficiently investigated. In a few cases thus far personally analyzed, left-sided symptoms have symbolized the unconscious sex conflicts in a masochistic setting, right-handed ones, the nutritive in a sadistic setting. Pains in the back of the head have been also frequently associated with repressed hate complexes, as have been severe pains in the jaws.

The character of the pain defies analysis, since descriptive phrases are used in such various ways by different observers. In some attacks, the head simply feels slightly sore, or heavy, or dull, or thick; "like a block of wood," is a frequent expression. "Filled with sawdust," one patient says. Again, the pain is agonizing, impossible to describe. Some patients shriek with the pain, become excited and roll about the floor, grasping the head between the hands, wishing to beat their brains out. Between these extremes numberless variants are found among different individuals, and in different attacks in the same individual. Nearly all patients have said that the severe pains have been throbbing or thumping, usually indicating great pressure from within or without; as Möbius has said, "some patients think the head will burst, others that it is being squeezed in a vise." Descriptions of bursting are more common. The pain is an all-pervading one, gradually mounting to a maximum, then running along continuously without any let-up, with, at all times, sudden accessions, especially on movement, if one leans over, or is forced to sudden exertion. In but the rarest instances is it described as lancinating in quality. It is the type of pain apparently seen in cerebral tumor, in acute hydrocephalus, in cerebrospinal meningitis, and is allied to the pain of opium poisoning, or of sea-sickness; all pointing in the direction of a modification of intracerebral pressure, at times an increase, or it may be a decrease, either of which may cause severe pain. Occasionally the phenomenon of a bilateral headache with marked predominance of one-sided pain will be observed.

The severity of the pain may be conditioned by a number of factors. Movement uniformly increases it. Bending over becomes impossible. The first movement on lying down is usually accompanied by a sudden rise in severity, but this gradually subsides. The taking of alcohol usually increases the severity of the pain, as does also the use of tobacco. Eating, if possible, may help somewhat, but usually augments the pain, and is avoided. Strong sensory impressions invariably increase the pain. Noises of various kinds often aggravate the pain tremendously and cause certain patients marked distress. The "Fourth of July" invariably drives many migrainous patients to some quiet spot in the country, free from crackers and bombs. Möbius notes that

the rage of migrainous parents directed toward their noisy children often resembles a pathological hatred. Strong light is invariably avoided, because of its tendency to increase the pain. The movements of the eyeball and attempts at visual accommodation cause an increase in the pain.

Intellectual application is often impossible; in milder attacks the awakening of a strong mental stimulus may make one forget the pain. Möbius says that his attacks, usually light ones, are frequently forgotten during an interesting visit to the Polyclinik, to be once more prominent afterward. One of us had frequently begun a lecture with a severe migraine to find it almost forgotten until the close, when it reappears often with renewed vigor.

The movements of straining at stool, and vomiting, coughing, etc., invariably cause a rapid and sharp rise in the severity of the pain. Sensory stimuli may have an unpleasant effect on the psyche. Thus, certain odors cause distress; the smell of cooking acts much as it does on shipboard; it accelerates vomiting. Certain skin phenomena, such as sore spots on the scalp, are frequent after the headaches.

In certain personal experiments with drugs the following have invariably increased the headache within a few minutes: A few whiffs of chloroform or of ether, adrenalin by mouth, digitalis, strophantin, and ergot. Drugs that raise the blood-pressure, in general, increase the pain when taken, especially at the beginning of the headache. The headache may clear away very suddenly after an attack of vomiting, or it may pass without vomiting; in some it fades away gradually. It may last a few minutes, a few hours, or a few days. Some cases of what Möbius calls status hemicranicus are recorded.

Vasomotor Disturbances.—Practically all attacks of migraine are accompanied by visible vasomotor disturbances. In most cases vasoconstrictor phenomena (coldness, paleness, gooseflesh, etc.) precede, to be followed later by vasodilator changes. Thomas and Cornu both point this out as a result of their experiences. Thomas contributes a statistical study of 107 cases in support of the early pallor, small pulse and coldness, which pass over to the phenomena of warm, red, flushed face and skin, and full pulse. The period of initial constriction may be unnoticed by reason of its transitory character. In some instances this initial vasoconstriction may be very marked and give rise to the phenomena of localized cyanosis, even advancing to the picture of the constriction phase of the Raynaud disease type. (Vagotonic.)

In the same manner the secondary vasomotor dilatation may pass the bounds ordinarily observed and lead to localized edema, to the erythromelalgic type, or, exceptionally, to hemorrhagic phenomena in the conjunctiva, ocular tissues, or even in the walls, of the stomach. (Sympathicotonic.)

Secretions.—Alteration in secretory functions are frequently observed early or late in the attacks. Reference has been made to the excess of secretion of tears as a frequent precursor. Vomiting of frothy

mucus, serous diarrhea, increase of sweat, coryza (Calmeil), or incessant salivation (Liveing, Tissot), are common phenomena.

The changes in urinary secretion have attracted careful attention. The early vasoconstriction of the periphery, coldness, lack of secretion of perspiration, etc., seem to afford a mechanical explanation for the increase of urinary secretion in the early stages. Metabolic studies show no fundamental disturbances (Bioglio). Although it is not possible to exclude metabolic disturbance as causing changes in vegetative control, probably it is more true that emotional influences cause such metabolic disturbances when present.

Trophic Disturbances.—These have been reported by several observers. Cornu says that nearly all of his cases of migraine show facial asymmetry, and facial atrophy is recorded. These instances are nearly always coincidences and are not necessarily attributes of the migraine. A facial atrophy which can be interpreted only on the basis of a migrainous disturbance of the vasomotor apparatus is very problematic, and certainly Cornu's results are not confirmed by others. Loss of weight in the severe rapidly recurrent cases is probably due to disturbance in general nutrition from stomach action. Herpes labialis is a not infrequent accompaniment of the attack in some patients.

Psychical Disturbances.—These have been noted by many observers, Liveing being one of the first to point out the relationship of disturbed psychical states to the attacks of migraine.

In the majority of migraine attacks there are few conscious mental changes before, during, or after the attacks. Mild depression, hopelessness, despondency with clear consciousness, are frequent moods. Möbius describes clouding of consciousness, and even severe stuporous states due to pain. Mingazzini believes there is justification for erecting a hemicranic dysphrenia group in which he distinguishes transitory and permanent forms. Recent observers are practically in accord, in showing that severe mental disturbances varying in character and intensity may be part of a migraine attack. Guidi has amplified these observations by reporting the history of a number of cases in which the patients suffered during the day before the onset, in a much more decided manner than by feelings of anxiety or depression as described by Liveing. He calls attention to grave alterations in the psychical state of a number of his patients. In one the entire character of the personality would change preceding the attack. A patient who had always been calm, reserved, quiet and modest, suddenly became much agitated, was forward, noisy, and loquacious, and told salacious stories, which was far from his usual behavior. While in health a spare eater, preceding an attack he suddenly became hungry and hankered especially for starchy foods. During the attack the patient had glycosuria, which disappeared later.

With the onset of pain the picture is less clear, yet there is little doubt that many patients suffer from profound psychical disturbances, which arise independently of the pain. One such case, under

personal observation, would be interpreted by Möbius, and rightly so, as one in which the pain is the first link in a hysterical reaction. But there are other cases which do not belong to this group. Mingazzini's hemicranic dysphrenias may be cited as examples, in part, at least. In others severe disturbances have occurred, such as states of anxiety, rising to actual anguish (Charcot); phobias of inability to perform acts (Cornu-Charcot); terror (Liveing, Féré, Kraft-Ebing); hallucinations of sight (phosphenes, colored lights, animals) and hearing with mental confusion (Forli, Mingazzini); maniacal excitement (Mingazzini, Jelliffe, Ranzou[1]) and stupor; unconsciousness (many authors).

Liveing reports that 25 per cent. of his cases showed psychical symptoms. The Italian observers record fewer, but it appears that at least from 10 to 15 per cent. of the cases of grave hemicrania show some distinct mental disturbance in some one or more of their attacks which is more significant than the usual depression which is so universal. These severe types only emphasize the fact that unconscious conflict is an important etiological factor in the migraines.

Symptomatic Headaches.—The occurrence of migraine-like attacks accompanying, or due to, definite somatic involvements, notably organic disease of the brain, is well known. The association of migraine with gout and malarial affections has been noted. It is very well known that attacks of migraine may be very frequent during the continuance of a malarial infection. Such may disappear for months after quinine therapy, and then reappear at the time of a later malarial infection.

Migraine-like attacks are not infrequent in cerebral tumor; they may appear periodically, as in cases fully reported by Abercrombie and Möbius, or they may be continuous and distinguishable with great difficulty from the pain of tumor, as in cases reported by Wernicke, who has said that such attacks may be quite readily confused with those more typical of tumors. In tumors, however, vomiting brings little or no relief; quiet gives less relief, and the fluctuation in intensity of the pain is less prominent. A primary onset of migraine-like attacks in adult life should always awaken the suspicion of an organic brain lesion.

The headache of pituitary disease, either as a pure pituitary syndrome, or arising from functional hyperplasia of the pituitary as a compensatory relation in thymic disease, or from other types of imbalanced endocrine function—adrenalin—pituitary balancing, etc., is frequent. Roentgen-ray study of the sella turcica shows certain anomalies here which inferentially show such possibilities.

Oppenheim has called particular attention to the occurrence of migraine-like attacks at the onset of tabes; Möbius is inclined to think it a rare combination, and regards it either as a pure coincidence or a migraine-like neuralgia. In general paresis, migraine-like attacks may be an initial symptom. Migraine attacks are not infrequent

[1] Ranzou: Monatschr. f. Psych. u. Neur., 1919, **47**, 98, has collected more of the cases to 1919.

throughout the early stages of the disease, but the structural correlations are still uncertain.

Diagnosis.—The difficulties appear in the consideration of ordinary headaches and in neurasthenic headaches; in distinguishing between the scotomata of migraine and other scotomata; the paresthesia of migraine and other paresthesias; the aphasia, the vomiting, etc., as seen in migraine, and the same as due to other causes. In most individuals abortive and incomplete attacks are the rule, and it is often extremely difficult to determine their precise significance.

Möbius has suggested that the problem is not only whether the case is one of migraine or not, but whether it is migraine alone, and not something additional. This author's contention that migraine is hereditary and begins in youth, would seem to make it a simple matter, but clinical experience shows that real migraines do appear in later years, apart from other affections, and as for the hereditary factor, the extreme prevalence of the affection makes this bogey generalization mean nothing. The periodic recurrence is a difficult criterion. There is usually no difficulty in diagnosing the classical attacks from simple headache, but at times such differentiation is impossible. Many chronic sufferers from migraine know well their real attacks, are able to distinguish abortive attacks, and also have headaches of an entirely different nature. The simplest test in separating abortive migraines from simple headaches is the occurrence of sensory phenomena, other than pain, which have their main origin in vasomotor disturbances. It is on this account that the severe headaches following the use of alcohol, ether, chloroform, opium, or analogous drugs should be allied to the migraines rather than to simple headaches. The headaches of neurasthenia, anemia, syphilis, lead poisoning, nasal sinus involvement, supraorbital neuralgia, nephritis, eye-strain, glaucoma, etc., may be differentiated if a careful history be taken.

Treatment.—The treatment of the migraine *attack* for the most part, is fairly satisfactory. There are few patients for whom some relief cannot be obtained, both with reference to the diminution of the number of attacks, and to the mitigation of the severity of the attack itself. The migraine habit, constitution, or liability—call it what one will—exists in very varying degrees; in some a very slight disturbance is sufficient to set free those forces which culminate in an attack; for others it requires a very much greater maladjustment. If the general reflex vascular hypothesis be taken as a tentative explanation, it may be readily understood why the elimination of various forms of peripheral irritation may result in eradication of one or more, and in certain instances of all, of the causes which start the migraine reaction.

Gastro-intestinal factors always occupy the front row in orthodox medicine. In the minds of most clinicians, and certainly, as generalized in the feelings of the patients, it is in the stomach, liver, or intestines that the main seat of the trouble is to be sought. The gastro-intestinal factor is undoubted in many cases; it may be exclusively gastric or colonic; perverted chemism, perverted bacterial action (primary or

secondary factors, no one can yet say). The significance of chemical features, resulting from altered gastric secretions or from toxic bacterial products, is not known positively. It is certain that none of the products which have been held responsible as autointoxicants are universal causes. At any rate, general gastro-intestinal hygiene should be carried out. Extreme constipation is to be avoided. The anal-erotic factors in many migraine attacks is indubitable even if *unconscious*.

In some excessive carbohydrate intake acts disastrously; in others wine, whisky, or gin. The history of inability to eat fatty food, particularly sausages, is not infrequent. In rarer instances, one notes that certain auditory stimuli may bring on a migraine. Some patients invariably have migrainous attacks after witnessing operatic or dramatic performances. In all of these psychological factors are operative.

If the multitudinous stimuli here indicated have any real relation, it is evident why such a variety of measures will be of help to a few, and why so many more will be worthless for many but useful for some. Medication between attacks is largely useless, save naturally in the symptomatic migraines. General medication, for no definite purpose but just in the hope that it may do good, as iodides, bromides, strychnine, etc., is senseless. If definite factors are found that need correction, and can be so modified by drugs in the desired direction, then they will prove useful. Thus iodides will undoubtedly help many presenile arteriosclerotic migraines; bromides are useful for sleepless and irritable conditions which provide a good foundation for the nervous instability that permits an attack; laxatives are called for if persistent constipation bears any causal relationship, and thyroid is invaluable in certain hypothyroid states which are very liable to affect the tonus of the vegetative nervous system, particularly in that type known as sympathicotonic. Very minute doses are useful in certain mild hyperthyroid migraines, as is also belladonna, or violet-ray treatment of the thyroid. In the migraines related to menstrual disorders in women a careful analysis of psychical and gonadal factors is essential.

Complicated systems of diet have been devised by many clinicians. Such are usually more prolific in engendering semi-invalidism than useful for the migrainous patient. Here and there a patient may derive benefit from a strict dietary régime, but unless there are real reasons why a patient should not eat red meat, or tomatoes, or sundry other articles, as determined by actual experience and under repeated experimental trials, in order to eliminate faddist's errors, the patient is better off without a diet card. The reasons sought for are not those contained in many treatises on dietetics, in which primitive notions concerning differences in red meat and white meat, vegetables growing under the ground and those above the ground, are idiotically perpetuated. The only satisfactory manner to attack the metabolic problem is to carry out a complete metabolism analysis. Haphazard attacks here and there lead only to premature and insecure judgments.

Complete formulas for attacking excessive bacterial putrefaction

are applicable only when it is proved that such excessive bacterial action exists and has a relation to the migraine. The hypothesis cannot be excluded *ex cathedra,* but it remains unproved for most cases, and of doubtful applicability in a few. The belief that the presence of indicanuria is an infallible index of harmful putrefactive products is not well founded.

The avoidance of alcohol and tobacco is only a relative problem. The individual's reaction to all influences should be rigidly estimated before purely personal prejudices are imposed "in the name of health."

In certain individuals a change of occupation may be indicated, but here again one must be conservative, and not consign all migraine patients to any definite regimen. Outdoor workers are by no means exempt from migraine; such, perhaps, should be clerks. The character of the work is to be borne in mind. The elements, haste, of pressure, and of lack of leisure are to be weighed.

Psychoanalysis should be advised for severe recurrent migraines in neurotic individuals. Chronic headaches are most frequently psychogenic in origin and need psychoanalysis. *Migraine is one of the most universal scapegoats for meliorizing psychological conflicts.*

For the *treatment of the attack* itself, one finds that a like fitting of remedies to the individual is called for. In the initial phase of vasoconstriction a number of vasodilators are of service, although their action is extremely unequal. The nitrites and nitrates have been employed for years, and usually with a fair degree of success if the dosage and individual member of the group be correctly chosen with reference to the severity of the attack. A mixture is of greatest value; nitroglycerin and erythrol tetranitrate give the best combination, for following the very evanescent and powerful action of the former, the more prolonged and steady action of the latter maintains the effect. The slower acting nitrates are practically useless. Nature's readjustment, vasodilatation by vomiting, etc., has already reduced the cerebral pressure, and the stage has passed when the dilating remedies might be useful. It is practically only in the vasoconstriction stage that the nitrites are worth much; and in many they are inefficient, the reason for this being as yet unappreciated. If given too late, they overdo the dilatation, and tend to increase the difficulties rather than minimize them.

The analgesic vasodilators have come to occupy the front rank. The precise pharmacology of each must be appreciated in order to obtain the best results. Solubility, time of absorption, slight differences in the chemical formula and n action, continuance of effect with minimum by-effects, are all to be studied. The list is a long one and is constantly on the increase. Antipyrin, acetanilid, phenacetin, and the related salicylic acid (aspirin, etc.) compounds are the chief members. It is to be remembered that while their general action is closely related, there are specific differences in the working of each, and the measure of success that one has in mastering the majority of migraines depends upon a knowledge of these factors. Antipyrin, by reason of its rapid

solubility and quick action, occupies an important place, but is not always applicable. Acetanilid, alone or in combination with other analgesics of related type (salicylic acid derivatives), bromides, and caffeine, is also valuable. The dosage should be graded according to the usual severity of the attacks. Tolerance is established in the quickly recurring attacks, and changes must be made. It is not yet certain what part is played by the respective analgesic and vasodilatation actions of this group. They have robbed migraine of most of its terrors, and tended to diminish the use of morphine and its derivatives very markedly.

Caffeine is a much overrated drug. In the abortive attacks and in the morning aftermaths of a migraine it is useful; but for a full-fledged attack it is not efficient. Similarly, bromides alone, chloral, and other widely used drugs are valuable only in mild attacks. They should be used in preference to other more potent remedies, which should be reserved for the severer attacks, in order that one's therapeutic measures may more correctly approximate the needs of each individual occasion.

The use of aconite and Cannabis indica is more restricted now that really efficient analgesics are known. Aconite is rarely called for, while Cannabis indica or Cannabis americana has a limited, though no less definite, place. In attacks associated with much mental depression the addition of cannabis is often useful. The often experienced inefficacy of this latter remedy is largely due to its extreme variability. Great care is therefore to be exercised in the selection of a proper preparation. Tablet preparations are usually worthless. This is equally true of the volatile nitrite preparations. Opium, or its main derivative, morphine, should be used only as a last resort. It is rarely really needed.

Lying down in a quiet, darkened room—a brisk saline laxative taken as early as possible, the patient being undressed and well covered—these are essential in the severe exhausting attacks. A very hot bath often aids very materially in restoring the patient to comparative freshness. Cold is to be avoided.

The greatest of all follies is to treat all patients and every attack alike.

Periodic Palsies: Ophthalmoplegic, Facioplegic, Hemiplegic Migraine.—It is known that in the ordinary attack of ophthalmic migraine there may occur various sensory or motor phenomena, among which anesthesias or paralyses are the most marked. These sensory and motor changes are extremely diverse when the entire range of the migraine symptomatology is brought into review, but there is one symptom grouping which, by reason of its comparative frequency and close similarity, was set apart from others occurring in this affection and named by Charcot *ophthalmoplegic migraine* in order to distinguish it from its more classical relation. It consists in a paresis or a paralysis of one or more muscles of the eye, innervated chiefly by the oculomotorius, which comes on either following or during a migraine attack.

Inasmuch as oculomotor pareses or paralyses may occur from a great diversity of causes, apart from a migraine, and may appear periodically, it has been held by many that the term ophthalmoplegic migraine has no particular right to exist, but the evidence is too great to eliminate migraine as a competent producing cause for these periodic oculomotor paralyses.

Furthermore, evidence is accumulating that indicates that paralyses of the oculomotorius are not the only paralytic syndromes, and it is recognized that a number of other muscles suffer from similar affections, which are more or less transitory in their character and which are in all probability due to vegetative nerve disorders chiefly of a vascular character. To these changeable and flitting palsies the name *periodic palsies* is given. Some are marked in certain families and hence have been termed familial. Among the rarer of these migraine equivalents, or periodic palsies are the facioplegic and hemiplegic types. Monoplegic syndromes are still rarer.

Etiology.—Whether constitutional factors play any greater part here than in migraine in general is difficult to decide. Certain periodic palsies not usually classed with migraines show marked heredity characteristics.

Symptoms.—Leaving aside for the moment the atypical and symptomatic periodic oculomotor paralyses due to other causes than migraine, one finds in these patients, usually during or after a severe attack of unilateral migraine, with headache, nausea, vomiting, etc., a ptosis of the eyelid on the same side, and a loss, partial or complete, of the upward, downward, and inward movements of the eye of the same side. This eye is usually directed outward and downward, and the patient sees double. This may or may not be accompanied by sensory disturbances in the superior branch of the trigeminus, just as may be observed in ordinary ophthalmic migraine.

After a variable length of time, a few days, a week or more, the paralysis disappears, usually gradually, and the patient suffers no inconvenience from the ocular palsies or the ptosis. In some individuals such palsies accompanying a migraine have come on comparatively early in life, almost with the beginning of the migraine attacks; for the majority, however, they follow several years after the establishment of a migraine, in some instances as late as sixty years. In some only a very severe attack will be accompanied by the oculomotor signs, or only slight palsies; transitory ptosis may occur frequently. But in others the palsies develop with each attack of migraine and often in increasing severity. The effects may persist longer and longer between the attacks, until in a few they become permanent palsies. This type, however, often permits of other interpretations.

A double lesion can be understood, although it rarely occurs. Isolated abducens palsy has been described, also isolated trochlearis; and complete ophthalmoplegia has been reported but in view of the many possible contributory factors it perhaps is preferable to view such cases from another standpoint.

Ophthalmoplegic Migraine.—There has been much speculation whether this form of third-nerve palsy is of nuclear or of peripheral origin. The view here held regarding migraine in general, that it is due to a disturbance in cerebral pressure secondary to vascular modifications would account for the oculomotor palsies as peripheral, in view of the location of the branches of the third nerve in relation to the cerebral vascular plexuses. In fact, the occurrence of the ophthalmoplegic type is one of the strong arguments for the general pressure hypothesis, as Spitzer has well argued. If, as has been shown by several autopsies, to these considerations additional local causes be added, which increase or permanently maintain such pressure effects, this interpretation is made more certain. Thus, exudates, fibrous processes, swelling in the cavernous sinuses, swelling of the hypophysis, tumor formation, gummata, etc., have been found in patients suffering from periodic oculomotor paralyses associated with migraine.

It is true that some of these are to be interpreted as symptomatic migraines, in which the foreign body acts primarily as an irritant to cause the vascular disturbance which sets free the migraine reaction, and secondarily serves as an additional cause of pressure to bring about the palsy. In a personally observed case, with basal gummata, the periodic oculomotor palsy and migraine attacks had occurred for a period extending over four or five years, usually with every menstrual period. Here were three interplaying factors, and the exact part played by each can only be inferred. The slight disturbance of menstruation, usually adjusted, in this case was not by reason of the exudate. A migraine was set up, the acute pressure of which, added to that of the exudate, caused the ophthalmoplegia. This ophthalmoplegia became fairly persistent in the intermigrainous periods in recent years. A few similar cases are found in the literature.

Facioplegic and Hemiplegic Types.—These are much rarer complications. Some observers would rule out the facial type, but there is no good reason for this. It does occur, and certain recurrent facial palsies which Bernhardt in his voluminous study has shown to occur in 7 or 8 per cent. of the cases are of this type. Hemiplegic attacks accompanying migraine are also rare.[1] Hemianopsia and optic neuritis (probably edematous or hemorrhagic) are still rarer complications.

Some *periodic palsies* belong in this group. Vasomotor palsies occur in the spinal cord as well and are accompanied or occasioned by edematous infiltrations usually of a mild grade. They are caused by much the same combination of stimuli as are the migraines but they are apparently much rarer. The resulting palsies are known as *periodic palsies* and are also discussed in the next group.

Diagnosis.—Every patient should be regarded as one suffering from something more than the migraine, until all accessory causes are excluded. What these may be have been mentioned already.

[1] Jelliffe: New York Med. Jour., January 6, 1906.

Treatment.—Little needs to be added to the therapy outlined under migraine. Syphilis as a cause for both a migraine and an exudate should be treated, and the Wassermann reaction utilized to clear up the diagnosis and therapeutic indications.

3. **Vasomotor Irritability Group.**

Angioneurotic Edema.—This condition is better described as acute circumscribed edema (Quincke[1]), since such a name does not commit one to its being a vascular allergic reaction, although this is possible.

These most striking skin edemas were described as early as 1778 by Salpertus. Crichton, in 1801, also observed them, and Graves, who also gave such an excellent outline of exophthalmic goiter, in 1848 described a patient whom he spoke of as "gouty" with localized swelling of the face, forehead, and eyes, in whom the edema persisted only a few hours. Since then various aberrant localizations have been described often under different names. Naturally hysteria bulked large in the diagnosis in the earlier days. Other synonyms indicate under which different diagnostic groups these cases may be found: urticaria, urticaria edematosa, epidermolysis bullosa, urticaria tuberosa, urticaria gangrenosa, and giant urticaria (Milton 1872). Further, one finds rheumatic edema, arthritic edema, repeating rheumatic edema, hydrops articulorum, intermittent rheumatic edema, neuroarthritic edema, in the period when the cases were grouped among the "rheumatisms." Gastrosuccorrhea periodica is a stomach localization. Probably there are others of obscure nature. Acute brain swelling, meningitis serosa, spinal swelling, local transient edema, intermittent edema, constitutional asthma, parotid swelling, and periodic paralysis are among them.

Quincke, in 1882, described it as acute circumscribed skin edema, while in a Kiel dissertation, one of his students, Dinkelacker, brought together many of the older descriptions, and showed the unity of several apparently dissimilar processes. He termed it acute edema. Quincke and Gross gave a more detailed description in 1904, but with no clear etiological insight into the syndrome. Recent students define these phenomena as specific allergic reactions.[2]

Occurrence.—The disorder is not frequent, yet it is not rare. Men and women appear about equally involved. It may be present in young children—one and a half months (Crozer Griffith); three months (Dinkelacker). After forty it appears very rarely, as an initial development, although in affected individuals it may persist until late in life. Cassirer reported histories of patients of seventy-nine and sixty-nine, in which the disease appeared comparatively late in life. Raven reports a case in a woman of eighty-six.

Occupation apparently plays no role. Heredity, on the other hand, is conspicuous. Many authors have mentioned this feature. Osler's family tree with 22 affected members has been freely cited, and is

[1] Monat. f. for Derm., July, 1882.

[2] Kammerer, H.: Münch. med. Wchnschr., 1924, **71**, 459, literature to 1924.

here reproduced in slightly changed form. Ensor[1] reports a family of 80 members, with 33 affected individuals, 12 of whom died of edema of the glottis. Whitings[2] family includes a study of at least 205 members, 30 of whom died of edema of the glottis. Similar hereditary features are reported by several observers, Bolton's[3] family of 12 with occasional tetany complications being one worthy of closer scrutiny. Skin edema, sneezing attacks, urticaria, periodic vomiting, were among the equivalents. The question of its transmission has not been completely cleared up. In Apert and Delille's families only the males were affected, but this does not seem to be an invariable rule.

In many families similar types of localized edema prevail in the members, while in others, apparently more often, all of the possible variants appear (Meige-Desnos). Other nervous system involvements appear associated in many of the families; how much of this is largely coincidence, how much general neuropathic causal relationship, is difficult to determine from the studies at hand.

The syndrome is associated infrequently with tabes, myasthenia gravis, spinal cord tumor, exophthalmic goiter, myxedema, epileptic attacks, periodic palsies, while it seems very frequently associated with many so-called psychogenic states—hysteria,[4] compulsion neuroses, migraine, etc.—and in certain psychotic individuals with schizophrenia, manic-depressive psychosis, feeble-mindedness.

Local traumata play a role at times, particularly in determining the location of the swelling. Emotional shock seems to be a direct etiological factor, as does also the action of thermal influences. Cold is very frequently an exciting factor in the reaction. The menstrual cycle factor seems to enter into the causative chain in certain cases.

A moment's reflection, therefore, will show that under the term acute circumscribed edema, one is dealing with a syndrome of great variability and multiform genetic pathogeny. In discussing the pathology this many-sided etiology will be referred to.

Symptoms.—The original conception of Quincke has been much employed, and Cassirer in his large monograph shows the present-day trend to include a large number of acute edematous swellings within a single nosological group. This group includes localized edema of the skin, edemas of the mucous membranes, of the eyelids, mouth, glottis, esophagus, stomach, intestines, respiratory tract, edemas of the joints, the meninges, the tendinous aponeuroses, the muscles, of the spinal cord, of the brain, of the kidneys, with polyuria, albuminuria, hemoglobinuria, diminished secretions, and edema of other structures.

The *onset* is usually acute, with some initial prodromal signs of malaise, fatigue, chilliness, anorexia, nausea, and slight rise in tem-

[1] Guy's Hosp. Rep., 1904.

[2] Lancet, 1908, ii, 1356.

[3] See Bolton, G. C. D.: Ztschr. f. Neurol., 1919, **63**, 360, Monats, f. Psy. u. Nerv., 1919, **45**, 201, for literature to 1919.

[4] Bolton: D. Z. f. N., 1922, **73**, 349.

perature (toxic cases of Bolton). The symptoms that develop will depend upon the localization of the process.

In the *skin* there are isolated swellings. These are localized, variable in size, at times small, resembling urticarial blotches (intermediary forms) but usually as distinct swellings, with an elastic feel, and due to local accumulations of clear serum within the skin. The color of the swelling is usually that of the skin, or paler, rarely red or reddish. The swelling comes on with great rapidity, in a few moments, and remains a few hours, mostly a few days, and then disappears without leaving any trace. They are, as a rule, non-irritating, painless, and only cause discomfort as a result of the tension. Certain patients experience burning, itching, and intense pain.

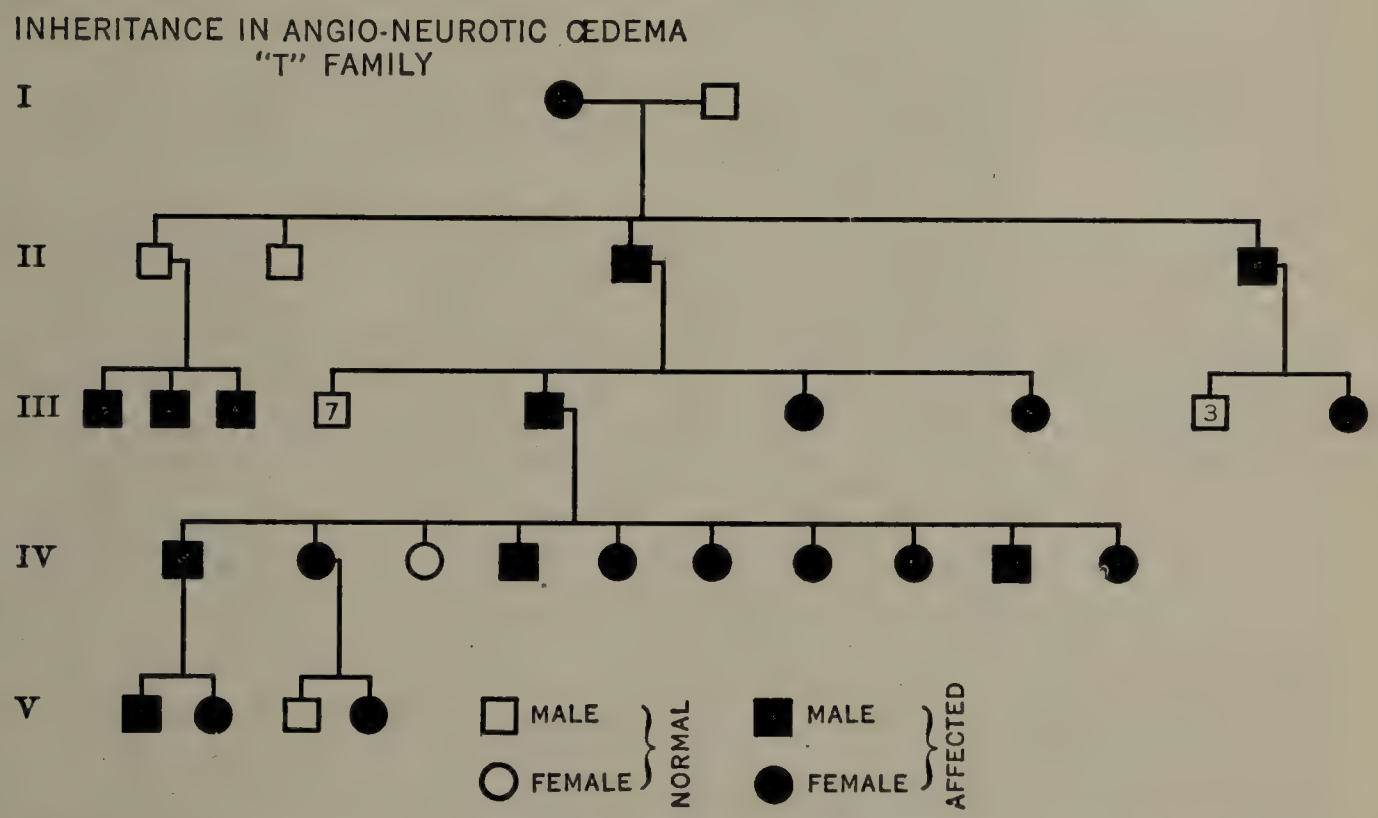


FIG. 71.—Chart showing hereditary transmission in an angioneurotic edema family. (Osler, Am. Jour. Med. Sci., 1888, **158**, 362.)

The *size* of the edematous patches varies greatly. At times very small—one-half inch—they are more apt to be three or four inches in diameter, or at times involve the larger part of a limb. The scrotum may at times swell up to the size of a foot-ball. The penis, in cases reported by Borner, has swollen to double its diameter. The entire body was swollen also in a remarkable case reported by Diethelm. At times the swellings are numerous, polymorphous, semiconfluent. They rarely rise more than one-quarter to one-half centimeter, but swellings two to four inches above the skin occur. The margins of the swellings are usually sharply circumscribed, but at times may shade off imperceptibly into normal areas. The swellings are usually described as circular or sausage-shaped. The swellings invade almost any layer in the skin, the musculature, or they may even invade the periosteum. Some have been termed pseudolipomata.

The *consistency* is semisolid, non-pitting, or slightly so. The *color*

as stated is usually that of the normal skin, or it may be paler, or have a cadaveric hue. Again it is pinkish to red, or even deep red. Often the color disappears on pressure. The color may change during the rise of the swelling.

Local *temperature* varies. At times the skin is colder, again it is warmer than that of the non-affected parts. Exact studies are wanting. It seems not unlikely that there is an initial increase in the local temperature.

Sensory changes are not present as a rule. Certain cases have shown preliminary neuralgic twinges, no definite sensory defect has been noted, but refined methods of examination, such as those demanded by Head, have not yet been made. There is frequently the subjective sense of great discomfort, especially in marked swellings about the face.

There are rarely any *residuals*, although occasionally scaling or peeling has been observed, probably for the more superficially lying edemas.

Secretory symptoms have not been carefully recorded. Local hyperidrosis, dermographia, increased tear secretion have been noted.

The location of the swelling may be almost anywhere, it cannot be said that one place more than another is a favorite site (statistically). Exposed portions of the body seem to be more often involved, but when on the hand or hands, the distribution is not of the glove type, nor are the swellings apt to be symmetrical, nor do they seem to follow radicular or peripheral distributions. There is a distinct tendency for a recurring edema to occupy the position involved during a former attack.

Periarticular swelling constitutes a peculiar type, so also do parotid and salivary gland edemas.

Mucous Membranes.—These are frequently involved. The lips, mouth, soft palate, tongue, pharyngeal pillars, nasal membrane, larynx are all sites of election. The last is particularly frequent and is dangerous to life. In these cases other structures than the larynx are implicated, especially the epiglottis and closely associated structures. In the larynx the mucous membrane is swollen and tense; the edema infiltrates throughout.

When the larynx is involved, the symptoms are apt to be very marked. There is beginning tickling, and rapidly oncoming difficulty in breathing, until marked dyspnea may supervene, with death, unless intubation or tracheotomy is performed. Some of these patients die within a few hours. Many cases, on the other hand, clear up in an hour, after severe dyspneic symptoms. Acute conjunctival or eyelid edema is not infrequent.

Edemas within the bronchi occur in perhaps 20 per cent. of the cases. They make up a certain percentage of the cases of asthma. Certain hay fevers undoubtedly may be arranged in this group. Lung edemas have been described.

In edemas of the *stomach* (gastrosuccorrhea periodica) external signs

are also usually present. There may be intermittent vomiting, or sudden acute pains and anorexia. The attack may last a few hours with severe pain, and finally more or less continuous vomiting of clear or bile-colored watery masses, marked thirst, and gradual disappearance of all of the symptoms. Bits of gastric mucosa have been accidentally dislodged which showed marked edematous swelling.

In *intestinal localizations* profuse diarrheas are present, with colicky pains, meteorism, tenderness of the abdomen, diminished urination great thirst, and collapse. The diarrheas are purely neurotic diarrheas, so-called, and occur in association with other signs of a circumscribed edema.

Rarer localizations present in the tendons have been described, particularly by Schlesinger. Muscle edemas are also rarely described, although it is probable that they are of frequent occurrence. Some lumbagos are possibly of this tpye. Articular edemas have been mentioned. They are frequently of psychical origin, as for instance in the classical association of attacks of gout and of anger.

Optic-nerve edema is one of the rarer localizations, as is also an edema in the labyrinth leading to a Ménière syndrome. (Oppenheim).

The bladder, kidney, and heart structures are among the rarest localizations. Meningitis serosa, aphasia, hemiplegia and monoplegias are among some of the more problematical occurrences reported and periodic paralyses are included here as well as in the previous group allied to the migraines.

Transition forms are common, especially urticaria-like eruptions. Acroparesthesias, Raynaud-like attacks, local asphyxias of the extremities, paroxysmal hemoglobinuria, acroasphyxia chronica, erythromelalgia, periodic paralysis, epidermolysis bullosa hereditaria, synovial serositis, fibrous serositis, herpes zoster are all affections with which attacks have been combined, singly or in groups of two or three. Occasionally edema, acroparesthesia, and erythromelalgia may alternate in one and the same patient.

Prognosis.—In general this is not good. The tendency to laryngeal localization must always be viewed with gravity. A great many individuals have died from edema of the glottis. Remissions are to be expected. Some patients suffer many years, others, but the minority it would appear, have but few attacks. There is some general tendency for the disorder to become milder as the affected individual grows older.

Pathogenesis.—Recent conceptions concerning edema are undergoing such radical modifications that it is practically impossible to interpret the findings here outlined along those lines that regard all edemas as cell phenomena solely, independent of the action of the vegetative nervous system acting on the bloodvessels. The studies of edema made by Fischer and others emphasize only the physico-chemical side of the problem. They neglect the role of the vegetative nervous system in regulating tissue tension and cellular chemism. The statement that the disorder is an angioneurosis by no means clears the situation, although

it is certain that the sympathetics are media from cause to effect. The study of anaphylactic phenomena, especially as seen in the so-called anaphylactic serum reactions, or serum diseases, has offered suggestive glimpses indicating certain analogies with the series of changes here outlined. Wherein are the proteids supposed to cause these related to the endocrinous hormones? It can only be stated that precisely similar processes and appearances are found in the serum reactions, and that it is not without profit to enquire more into the mechanism of their production in an attempt to understand the acute circumscribed edemas here fragmentarily outlined. Unfortunately the mechanisms of the changes in the anaphylactic reactions are still much in the dark. There is a distinct tendency to include the anaphylactic reactions under the phenomena regulated by the vegetative nervous system.[1] "Specific allergy" seems to cover one group.

It is not improbable that there are a series of reactions represented in the acute circumscribed edemas. It is not a unicum, and analysis will show that a number of different causative factors may underlie precisely similar phenomena, be they in any vascular area of the body.

Cassirer adopts this viewpoint, but consents to make only two groups of cases: (*a*) a toxic, autotoxic group, in which the poison works in some mysterious way, which a wealth of language can conceal, better than it can reveal, and (*b*) a heredofamilial or constitutional neuropathic group, which he regards as intimately associated with instability in certain parts of the vegetative nervous system. According to present day knowledge Cassirer's scheme may be slightly advanced and the following types may be distinguished. (1) A general familial or hereditary, constitutional or dispositional hyperreactivity of the capillary nervous system which shows no specific reactions to proteids or other noxæ. (2) Specific anaphylactic reactions arising on the basis of a previous sensitization. (3) Primary specific allergies without previous sensitizations. Allergic constitutional states, etc. This may be, he says, associated in some manner with modifications in the internal gland secretions. Here another dark portal is entered.

After a careful revision of the entire question our own view is to include the participation of psychogenic components in the etiology in which infantile libidinal fixations are to be more seriously evaluated. Whether this is a fundamental factor underlying the sympathetic hypotonus which seems to accompany the whole group (Bolton) is to be tested when more psychoanalytic data are available (Jelliffe).

Treatment.—This is purely empirical. One should not eat any of those things which experience has shown to be hazardous. If one of the more pronounced toxic (anaphylactic-allergic) types be present, careful study must be made of all of the patient's protein reactions, and attempts made calculated to regulate the diet accordingly. It seems

[1] Roncoroni: Ergebnisse d. Neurologie u. Psychiatric, vol. **2**, No. 1. Also see Noël Paton: Nervous Regulators of Metabolism. Bayliss: Principles of General Physiology. Also Smith's Studies on Anaphylaxis, loc. cit. Kammerer, loc. cit (p. 202).

possible that it is through the gastro-intestinal canal that such products gain entry, particularly in food, yet some may enter the respiratory tract, as seems to be the case in the related hay fever reactions which are thought to follow certain contacts, such an ragweed, rose, hay, and other pollens, or even the emanations from lower animals.

From specific exclusion of certain proteins one passes to the general hygiene of the intestine. This means a sort of search in the dark for effective agencies by chemical means. One is justified notwithstanding in trying to bring about altered bowel conditions, which empirically may do some good, when a laisser-faire attitude seems to perpetuate the disturbance. Naturally one should avoid intestinal therapy, should the patient be of an entirely different type, say the intensely neurotic forms with familial hereditary burdens, and emotional shock reactions. These patients need a psychoanalysis.

Of the gastro-intestinal antiseptics so-called, few are of value. Menthol, saline laxatives, carbonated waters, careful dieting (?) may be found among the conventional remedies in the books. The taking of a milk-vegetable diet has been coincident with betterment in some individuals and coincident with getting worse in others.

In certain cases with associated toxemias, such as malaria, etc., a specific therapy is indicated.

On the supposition that the bloodvessels need bracing up to prevent transudation through their walls, also a hypothetical postulate, apparently inadequate, such drugs as strychnine, ergot, arsenic, atropine, morphine have been recommended. While all of these will bring about vasoconstriction it is not apparent whether they can alter a hypothetical transudability or not. Calcium lactate is a modern weapon for this latter. It was mentioned in this place early, and may be of service in preventing transudates, as such are thought to be conditioned by a diminution in the calcium content of the body plasma. Cassirer mentions calcium chlorate. At all events the vasoconstricting drugs have not been of any particular service clinically. Now and then they seem of service; none have been proved of prophylactic value which is a stricter test of their usefulness, since the disease is so self-limited. Atropine is of great value at times. It should always be tried in the cases which seem to show sympathetic let-down—hyperthyroid types.

In cases associated with laryngeal symptoms, intubation may be necessary—even tracheotomy. There are records of certain patients condemned to the persistent use of the tracheotomy tube.

In the more strictly neurotic type—Cassirer's group (*b*)—it is important that the patient be taught a healthy morale. The substitution of reasonable and intelligent actions for purely instinctive and emotional reactions must be acquired by them, if they can hope to in any way control their hair-trigger vegetative nervous system. Perhaps it was so given to them, defective and badly coördinated; even then a rational pedagogy will prove of service. Many will be helped by the

methods outlined by Dubois or Dejerine;[1] others will need a psychoanalysis. Increasing experience and the literature are serving to establish the fact that psychogenic factors amenable to psychoanalysis are responsible for a number of these phenomena. Asthma, hay fever, rose colds, protein sensitizations, urticarias, synovial swellings, arthritis deformans, migraine and other edematous states causing acute, even chronic disorders have been in many instances greatly relieved, in others cured, by the altered emotional attitude taken by analyzed patients. Just how the vegetative pathways become involved in their complex neuro-biochemical relations has been shown in the pages preceding, also in the chapters on the neuroses and psychoneuroses, under which groups also many of these patients are classified.

Bolton would include in the therapeutic resources those hormones which increase the sympathetic tonus. Pituitary extracts are among the most valuable of these. Thyroid and parathyroid therapy also, at times seems indicated. Following, in general, our modification of Cassirer's grouping, the therapeutic attack must be regulated according to the predominance of one or the other factors. In certain hyperthyroid cases bromides or iodides act disastrously, especially in meningeal involvements, when these agents precipitate delirium.

In the second group congenital constitutional factors are reflected in an inferiority of the sympathetic tonus factors. Here those agents which may bring about an increase in sympathetic tonus bring about strikingly valuable therapeutic results.

Genito-urinary System.—Here splanchnic and cerebrospinal controls are in evidence. The former act through the mesenteric, hypogastric and hemorrhoidal parasympathetic sacral ganglia, supplying with non-medullated fibers the involuntary muscles and the mucous membranes, N. hypogastricus to muscles of colon and bladder (sphincters), the plexus cavernosus, and nervus erigens to the genital vasomotors. The latter act through medullated fibers to the voluntary muscles and adjacent skin areas. The nervus pudendus communis supplies the external sphincter ani, external sphincter vesicæ, compressor urethræ, deep perinei, etc.

A series of vegetative reflexes are here met with, the most important being:

1. Scrotal reflex: Stroking of perineum or femoral skin; contraction of dartos.
2. Bladder reflex: Stretching or stimulus (mechanical, psychical) of bladder wall; contraction of bladder (mechanical, psychical).
3. Rectal reflex: Stretching or stimulus of rectum; contraction of rectum.
4. Genital reflex: Psychical or mechanical stimulus; erection and hyperemia; corpus cavernosus.
5. Uterus reflex: Stretching or irritation of uterus; contraction.

[1] Dubois: Pyschic Treatment of Nervous Disorders. Dejerine: Psychoneuroses and Psychotherapy, Philadelphia, 1915. Jelliffe: Technic of Psychoanalysis, New York, 1918.

6. Anal reflex: Stretching of anus; psychical; contracture of sphincter ani.

All of these reflexes act through psychical levels as well as through peripheral, *i. e.*, somatic ones.[1]

Bladder.[2]—The general mechanisms of the bladder pattern after those of the rectum and quite analogous symptoms follow disturbances of homologous relationships of the autonomic and spinocerebral pathways. The chief parasympathetic series travel in the sacral vesical nerves to and from the inferior mesenteric and hypogastric ganglia. Sympathetic fibers are also functionating through the hypogastric, to and from the inferior mesenteric ganglia. Thus the bladder has a vegetative mechanism comparable to that of the pupils.

Emptying of the bladder follows similar lines to that of emptying of the rectum. Section of the cord to above the mid-dorsal region brings about automatic emptying. Psychical influences are here active as in the case of the rectum—urethral erotic with retention and incontinence of purely psychical character. These are discussed under Psychoneuroses, whereas the more mechanical, neurological features are taken up under Diseases of the Spinal Cord.

Sexual Organs.—This chapter in neuropsychiatry would require a library to adequately present. Only the briefest mutilated summary can be here presented. In the phyletic carrying on, the germ plasm (testes, ovaries, in brief) has come to summarize the whole of the possibilities of further evolution. Enormously complex vegetative anomalies are met with which the gynecological and obstetrical specialties are studying. Only in recent years, through the work of Cajal and the Guttmachers, have the neuron connections been definitely analyzed. On the basis of these more accurate morphological studies of the neurology of the organs of generation an entire recasting of the whole physiopathology can be brought about.[3]

Parasympathetic and sympathetic supplies are abundantly present. The former carry stimuli through the nervi erigentes from the sacral cord, producing vasodilatation and erection, turgor in the female, nipple erection, etc. The latter carry stimuli through the hypogastric nerve to cause vasoconstriction and contraction of the unstriped musculature of the sexual glands and discharge channels. In the act of copulation, desire, erection and ejaculation (orgasm) may show separate mechanisms. At the physico-chemical level the concretization of sexual desire usually reaches an active adult stage with the onset of

[1] Dennig: Die Innervation der Harnblase, Berlin, 1926. Peretz, A.: Klin. Wchnschr., 1922, vol. **1**, No. 48 for pharmacology. KIDNEY: Haebler, H.: Ueber die nervöse Versorgung der Nierenkelche, Ztschr. f. Urology., 1922, **16**, 377. Stahl u. Schute: Ueber d. Einfluss d. vegetativen Nervensystems auf. d. Nierenfunktion, Zeit. f. d. g. Exp. Med., 1923, **39**, 312.

[2] See papers by Schwarz, O.: Untersuchung über die Physiologie und Pathologie der Blasenfunktion, Wiener Arch. f. innere Med., 1920, and others cited therein.

[3] Cajal: Trabajos: Vol. **20**. Guttmacher: John's Hopkins Bulletin, 1921, **32**, 394. From the psychological side the works of Stekel afford many penetrating observations, also see Ferenczi, Versuch einer Genital Theorie, 1926. Reich, W.: Die Funktion d. Orgasmus, 1927.

puberty. It is assumed that chemical stimuli—hormones—act at this level to cause tension—tumescence—within the organs themselves, and also possibly working upon higher level nervous structures cause an increase in psychical tension, thereby causing increased sensitization to sensory contacts and to mental stimuli. Thus the erotic impulse springs up at a touch or under the influence of a symbolic expression, as in poetry, or other artistic creation.

The whole impulse of life and of the principle of race-preservation, *i. e.*, immortality, is bound up in the instinct of reproduction. The energy of *this* instinct has been termed *libido* by Freud.

Certain hints obtained from the study of the processes of reproduction in lower organisms—protozoa, protophyta—tend to show that the continuance of the life of the individual and of the species has been obtained through a sacrifice of the ego. Purely individual reproduction gave way to gametic reproduction. The principle of fertilization by sexual processes replaced the principle of individual reproduction, as by budding, for example.

To put the matter in a few words, the process has been something like this: The original unicellular organism was all-sufficient. The amœba, for example, performs all of the functions of ingestion, digestion, egestion and reproduction with practically no structural differentiation. To be sure, there are certain differences in different parts of the protoplasm, but they are relatively inconsiderable, and after all, all of these functions are carried on in the single cell. Correspondingly this single cell is practically immortal, that is, it only dies as a result of accident. The immortality is secured at the expense of differentiation of structure. Immortality can only be attained in simple, all-sufficient, unicellular organisms. In developing from this primary condition one of the first steps is a union of a group of cells, forming a more or less loosely integrated organism. As evolution proceeds, however, this integration becomes much more definite and along with it there goes differentiation in the functions and correspondingly in the structures of the different cells, so that there begin to be cells which are set aside, so to speak, for digestion, others that are set aside for reproduction, etc. The cells so differentiated are very much more efficient in the performance of their several functions than the original undifferentiated cell was, but each cell so differentiated has reached its excellence at the expense of giving up (sacrificing) its other functions and developing in this one particular way. The advantage gained has been that each cell of the group received better service, so far as each function was concerned, than before, but each cell had to sacrifice something of its own independence in order to get this advantage, and in making this sacrifice perhaps the most important thing which it gave up was its practical immortality. Viewed from this angle it is made apparent that death itself has been acquired by natural selection because of its advantages. Highly complex individuals soon accumulate so much past that it is distinctly advantageous to scrap them and make a new start; hence, out of death comes life, a contrast constantly

met with at the symbolic level. In the highest, most complicated organizations, therefore, each cell, while it has a certain individuality, is highly specialized and therefore has only a relatively short span of life. It has given up a great deal in order that the community of cells of which it is a part may profit. In the progress of evolution the process of selection is being, so to speak, slowly transferred from the single cell to the larger group. Each function, therefore, whether of an organ or only of a single cell, may be looked at from the double point of view as to whether it ministers to the preservation of the individual organ or cell as such, or whether it ministers to the preservation of the whole organism, and therefore it may again be seen that both nutritive and reproductive activities are represented at all three levels, the physico-chemical, the sensorimotor and the psychic.

Inasmuch, however, as vital energy acting solely through physico-chemical processes does not afford any adequate explanation for all and least of all for the most important of the phenomena of evolution, an adequate hypothesis must also include similar activities at higher levels, *i. e.*, vital and psychical. The out-and-out materialist stops at the lowest levels, the vitalist midway, the evolutionist argues for the leadership of the psychical, but needs the interrelationship of all. Psychical impotence with intact organs, for instance, is inexplicable on physico-chemical hypotheses.

Seen from another angle this vexed subject of interrelationships is well illustrated in the large disease group of schizophrenia (dementia precox). From the psychical side alone some have endeavored to explain it as a series of reactions to repressed and unconscious sexual activities—repressed and unconscious because of higher cultural demands and inability on the part of the patient to sublimate, *i. e.*, employ his libido in its numerous useful socialized transformations arrived at in the course of cultural development. A compromise situation adopts the interrelatory hypothesis. Reasoning in such terms the interrelationist says that schizophrenia is a disorder occurring in certain inferior individuals; inferior because of certain structural defects of the gonadal or other endocrinous systems (testes, ovaries, pituitary, etc.). These disorders induce changes at the metabolic (physico-chemical) level and thus bring about the disease, which because of inferiority in these endocrinous glands, chiefly gonadal, cause a symptomatology which is largely tinctured with sexual concepts. The Abderhalden pregnancy reaction—ovarian, testicular, hormone changes—shows, in some cases, a peculiar activity with schizophrenics pointing to some disturbance at the physico-chemical level, although all of the confusing contradictions in technic have not yet permitted any positive statements even as to these results. To the student of the problem from the psychical side it is difficult to comprehend how changes in chemical reactions will determine a fairly constant mental picture which psychoanalysis shows to be largely occupied with sexual symbolization with its consequent emotional reactions. The increasing evidence showing that emotional reactions can produce somatic

modifications, as seen in hysterical conversions, compulsive substitutions, and psychotic projections, tends to throw the proof over to the psychical side, with the compromise situation that both somatic inferiority and psychical symbolizations are interrelated and more or less reciprocal phenomena. The student by keeping his mind open and thinking in these various terms will certainly gather more real information from his patient than if he held to one side only of the problem.

In the phenomenon of *erection* one sees these principles at work. The cerebral or psychical is the most frequent source of origin for vasodilation. The pathways are by means of the cord to the upper lumbar segments and by way of the erector nerves. In severe spinal injuries psychical erection may remain intact. Severe continuous priapism is not infrequently of purely cerebral origin, either organic as in encephalitis, non-purulent or purulent, syphilitic (paresis), or possibly purely psychical as in some manic states, some schizophrenics.

Sensorimotor levels respond to the sensory stimuli of the skin of the penis or adjacent organs, and the reflex pathways are made up of the spinal sensory nerves, the second sacral segment and the dorsalis penis and pudendis communis nerves acting through synaptic junctures in the sympathetic ganglion.

The physico-chemical levels respond to the tension stimuli from the bladder, seminal glands, etc., acting through the hypogastric plexus.

In ejaculation, sympathetic and cerebrospinal pathways are utilized. A summation of stimuli, acting through the sympathetic, forces the threshold, setting free a peristaltic contracture of the vasa deferentia with the accumulation of genital secretions in the prostatic portion of the urethra. A spinal reflex causes the contraction of the bulbo- and ischiocavernous muscles with the ejaculation of the semen.

Sympathetic disturbances are rare, spinal ones not infrequent in conus lesions, either being traumatic or due to new growth or infiltrating disease, tumor, syphilis, etc. Ejaculation in coitus, in masturbation, or in pollution dreams is usually accompanied by other autonomic signs, such as mydriasis, hyperidrosis and cardiac palpitation. Pollution dreams have determinants at all the levels mentioned. They are usually not harmful. When frequent and evidently pathological they may arise from lower level stimuli (prostatic disease, etc.), but are more often of psychical origin—usually accomplishing the repressed and unconscious wish for culturally forbidden sexual activities (masturbatory, homosexual, incestuous, or bestiality fantasies). Impotency, partial or complete, is most frequently chiefly a psychogenic complaint. Psychoanalysis shows that copulation is a highly complex act and that complete potency is only attained when all of the partial libido trends are sublimated in a real love act, *i. e.*, one that is essentially constructive.[1]

[1] Reich, W.: Die Funktion d. Orgasmus, 1927. Ferenczi: Versuch einer Genital Theorie, 1926.

Vegetative Skin Syndromes.—The vegetative nervous disorders of the skin is still an unwritten chapter in medicine. It is only possible here to sketch rapidly a few skin syndromes which are spoken of in dermatological literature as "neurotic," or as "trophic dermatoses," etc. The skin is peculiarly under the control of the vegetative nervous system and a careful study of skin phenomena with an eye to their neurological significance will yield many fruitful suggestions. The text-books of dermatology speak of the extreme frequency, and chronicity of such disorders as simple acne, as psoriasis, eczema, and a number of others. They often are treated for decades without success under the present-day dermatological concepts. Scarcely any dermatologist goes behind the term "neurotic," or "nervous," Herein is a rich field for vegetative nervous system disturbances on an emotional basis that can be reached by psychoanalysis. The field of skin symbolisms in the neuroses, psychoneuroses, and psychoses is just beginning to be studied by dermatologists.[1] For the neurologist there are numerous fruitful problems.

From the endocrine point of view a number of definite skin trophisms may be recognized. In hyperpituitary states the hair follicles augment in size, the papillæ hypertrophy, the increased sweat gland activity causes a moist and greasy skin. The fibrosis in the deeper layers causes a dense, boggy type of skin with increase in the furrows, particularly in the hands and face. Hypertrichosis is frequent. In hypopituitary states the skin tends to be smooth and thin and atrophied. It is almost infantile like in quality. Pigmentation is frequent. Pityriasis rubra pilaris has been observed. Hyperthyroid states show a number of anomalies chiefly as hyperidrosis, erythemata, hyperkeratosis, pruritus, urticaria, dystrophy of nails, white spots in nails, increased pigmentation (from suprarenal compensations?). In the hypothyroid states the thick skin of myxedema is classical. Perspiration is diminished, thinning of the hair frequent, premature grayness. The skin anomalies in adrenal disease are often very characteristic. The Addisonian bronzing is classical, while minor grades of bronzing are frequently seen in chronic adrenal exhaustion from toxemias, or continuous psychic conflicts. The hypertrichosis of suprarenal virilism is classic; this is frequently associated with gonadal inferiority states, in the female. Hypogonadal states in the male usually cause defective hair developments with thin delicate skin. The "peaches and cream," complexion is often associated with persistent thymus activity.

These are but a few of the more familiar relationships between skin trophisms and the uniglandular and polyglandular endocrinopathies.[2]

[1] Jelliffe: Psoriasis as a Hysterical Conversion Mechanism, New York Med. Jour., December, 1916. Reede, E. H.: Role of Veg. N. Syst. in Diseases of the Skin. Derm. Sect. Jour. Am. Med. Assn., June 14, 1918. Morris, M.: The Internal Secretions in Relation to Dermatology, British Med. Jour., May 17, 1913. Bachet, P. E.: The Dermatologic Symptoms of Endocrine Dysfunction, Arch. of Derm. and Syph, 1921, **4**, 660. Bunsemann: Ztschr. f. d. g. Neur. u. Psych., 1922, vol. **78**. Böwing, H.: Zur Pathologie der vegetativen Funktionen der Haut. Deut. Ztschr.,f. Neurol., 1923, **76**, 307. O'Donovan: Dermatological Neuroses, Psyche Miniature Medicine Series, London, 1927.

[2] Consult Bloch, Br.: Das Problem der Pigmentbildung in der Haut, Arch. f. Derm. u. Syph., 1917, **124**, 129.

Urticaria, giant urticaria, and related skin affections have a polymorphous etiology. While certain protein sensitizations may account for some, what a protein sensitization really is, is not yet accounted for. The centuries old observation that such phenomena occur in so-called "neuropathic" individuals is probably only another way of saying that psychogenic impulses, *i. e.*, symbolic conditioned reflexes, are operative and that early infantile impressions acting through unconscious mechanisms, see psychoanalysis section—cause these skin phenomena. The vast majority of these reactions cease when the individuals reaches an adult psychosexual development. When they persist into adult life analytic investigation has revealed in many, the presence of fixations in the Freudian sense, and their analysis has relieved, if not cured, many such individuals. The investigation in this field is still in its infancy.

Similarly there are psychogenic acnes, psychogenic eczemas, and psychogenic psoriases. The fact that certain of these are benefited by roentgen-ray or radium treatment, or by thyroid medication, does not change the fundamental nature, and in the refractory cases, psychoanalysis has been of inestimable value, not alone in understanding the dynamic factors underlying their causation, but in curing some of them through a better comprehension of such factors.



FIG. 72.—Scleroderma. (Nammack.)

Scleroderma.—Hippocrates described an Athenian who had a hard, indurated skin all over his body, and Galen spoke of patients whose skin was hard and leathery, with the pores all stopped up. Thirial, in 1845, gave the earliest good descriptions of scleroderma, and the French

school, with Ball, Charcot, and Hallopeau, fashioned the present-day descriptions of this disorder. Grasset and Brissaud, in 1890, were among the earlier advocates that it was a vegetative nerve disorder, while Strümpell first correlated some of the endocrinous cases, observing a patient with scleroderma and acromegaly.

Its description, occurrence, and differential diagnosis, its numerous forms and variations are best to be found in dermatological literature. The neurological interest focusses itself upon the determination of the level of the nervous system involved, for the pathogeny is extremely multiform. Peripheral nerve lesions (trapezius palsy) have been known to be followed by localized scleroderma. Spinal cord injury, involving Jacobsohn's sympathetic nuclei, occasions other cases. Numerous cases are associated with other spinal injuries, as in syringomyelia,[1] poliomyelitis, multiple sclerosis, etc.



FIG. 73.—Scleroderma with sclerodactyly. (S. P. Goodhart.)

As a polyglandular endocrinopathic syndrome, scleroderma has shown a many-sided character. Cases are known with associated hypophysis, adrenal, thyroid, and mesenteric gland disease. The thyroid (frequently syphilitic, also gonadal) correlations seem numerically to outnumber the other in the cases reported.[2]

No psychogenic cases have as yet been analyzed, but inasmuch as many if not most hyperthyroidisms are distinctly psychogenic, at least in the initial stages, it is not an unreasonable hypothesis that psychogenic factors may form a part of the scleroderma pathogeny.

[1] Herzog, M.: Schw. med. Wchnschr., 1921, 667.

[2] Marinesco and Goldstein: Nouv. Iconog. de la Salpêtrière, Paris, 1913, p. 272. MacCallum: Tr. Ann. Phys., 1926, **41**, 190. Waldorp: Semana Med., 1926, p. 1351; Santoinini: Folia Med., 1926, **12**, 727, for luetic hypogenital case.

The chief pathological alteration, increased fibrosis, is largely due to an imbalance of the sympathetic branch of the vegetative system arcs. This increased sympathicotrophic action may result from a number of causes—diminished thyroid secretion, diminished adrenalin secretion being among them. The more exact pharmacological dynamics have not yet been definitely worked out. Timme first attempted to ascertain the resultant pathology of oversympathetic stimuli by cutting off the balancing parasympathetic stimuli in the walls of the stomach. His experiments throw some light on fibrosis in general and should be pursued in any study of a lesion resulting in a chronic fibrosis whether of the skin, the stomach, kidneys, liver, or any organ of the body.[1]

Treatment.—The therapy will depend upon the underlying factors. The endocrinous and psychogenic cases would seem to offer the most opportunity either by carefully balanced opotherapy or by psychotherapy.

Multiple Neurotic Gangrene of the Skin.—The condition described by this title may be mentioned. It is a dermal process in which the vasomotor constriction is not limited solely to the extremities, but to apparently unrelated, isolated patches of the skin.

While exaggerations in neuropaths (hysteria), or in psychoses (schizophrenia) may bring these changes about by artefact, malingering explains only a small number of them, granting that the term is any explanation at all, for why any particular person malingers still remains to be understood.

The disorder sets in with a burning and prickling of the skin. Then after a variable length of time (a few minutes to forty-eight hours) swellings occur, blisters form, with later necrosis. At times only a circumscribed edematous bleb forms.

Sweat Secretory Mechanisms.—These are closely related to, yet may be independent of, the vasomotor mechanisms. They probably have independent ganglion cell representation in the lateral horns. In general those pharmacological agents which increase vasodilatation (autonomic) increase sweat secretions, yet vasodilatation may occur without sweating, and the latter may occur with anemia and vasoconstriction (cold sweat of fear, sweating of face in migraine, epileptic aura, etc.).

Cervical sympathetic and bulbar-autonomic innervation is probable, and cortical connections, though still topographically unanalyzed are certain (unilateral sweating in thalamic lesions, hemiplegia, epidemic encephalitis, neuroses, schizophrenia). Certain anatomists place the cortical pathways among the motor tracts in the internal capsule. The hypothalamus is made an interbrain synaptic station by some.[2]

[1] Timme: Loc. cit. Mosenthin: Arch. f. Derm., vol. **118,** 613. Cassirer: Loc. cit: Lewandowsky: Handbuch. Laredde et Thomas: Arch. d. méd. exp., 1898. Dinkler. D. Arch. f. k. med., 1891, 48. Roux, Rev. Neur, 1902, p. 721. Raymond et Alquier: Soc. d. m. d. Hôp. d. Paris, 1904 (Spinal cord case).

[2] See Müller and Gläser: Deut. Zeit. f. Nerven., vol. **47** to **48,** 365, for literature, also Thomas, Le Reflex Pilomotor., 1921.

Clinically, sweating is increased in certain hemiplegias, and in herpes zoster. Diminution of the sweat is seen in certain cases of poliomyelitis, multiple sclerosis, syringomyelia, myelitis and tumor of the spinal cord.

In spinal cord injuries the work of André Thomas[1] shows that two periods may be distinguished in the clinical evolution of wounds of the cord. During the first called by most writers the period of shock or inhibition, there is complete paralysis of all parts innervated by the segments of the cord below the lesion. The sweat secretions are almost entirely absent in this region, appearing only on the areas belonging to that part of the cord which has preserved connection with the brain. The secretion in these areas thus offers a means for determining the level of the lesion and is called by Thomas *encephalic perspiration.* Later a phase occurs in which activity is manifested in the section below the wound, which, though cut off from the brain, comes under the independent control of stimuli from the periphery, resulting in pilomotor reflexes, sweat reflexes, etc. From clinical and autopsy observation Thomas comes to the following conclusions concerning the localization of the spinal sudorific centers; these spinal wounds show that the sudorific fibers reaching a cutaneous territory belong to much lower segments than do the sensory fibers of the same area. There are perspiration centers for the head and neck and the upper part of the thorax to the third and fourth rib in that part of the sympathetic column which extends from the eighth cervical segment to the third dorsal. There are centers for the upper extremities in dorsal segments 5, 6, and 7, but they probably extend also somewhat lower also. It is very likely that there are no perspiration centers situated below the third lumbar segment. In paraplegics wounded in war the sudoric reflexes behave in a manner very similar to the pilomotor reflexes, which is very natural as their centers are situated near each other in the spine. When the spinal perspiration predominates on one side, the pilomotor reflexes are more likely on the same side, but the study of the pilomotor reflexes gives more precise and trustworthy results concerning the condition of the spinal sympathetic centers than does the study of the sudorific reflexes. Even after the spinal perspiration together with the defense reflexes, has once made its appearance in the region below the lesion, the sudoric secretion may cease again and the perspiration above and below the lesion may alternately recur and disappear. In some cases this phenomena may be referred to a diminution of urinary secretions or to vascular tension, but it is always evident that the two sections of the sympathetic column—the one above and the one below the lesion—do not react in the same manner when under the same influences (elements secreted by the urine). The encephalic perspiration of the uninjured part of the body remains always under the control of the brain—a circumstance which is evidence of the existence of one or several sudorific areas in the encephalon. Thomas' observations,

[1] André Thomas: Encephalic Perspiration and Spinal Perspiration, L'Encephale, April, 1920, **15**, 223.

therefore, seem to explain the mechanism of the sudorific secretion, showing that probably certain conditions of the fluids of the body influence the secretion by their action on areas situated above the spinal cord; and thus the anatomico-clinical experiences are brought into accord with the views expressed by physiologists.

Great variability in the sweat activity is seen in many psychopathic individuals, in vagotonic types and in the psychoneuroses, hysteria, anxiety states, compulsive states and in hyperthyroidism. The reactions of the sweat secretion mechanisms are exquisitely sensitive to psychogenic factors, as is seen by the response to pain, gastro-intestinal cramp, carminatives, nicotine, anxiety and joy. Veraguth's psychogalvanic reflex experiments show that a close relationship exists between the skin secretions and psychical processes. The fineness of registration and the extreme complexity of the phenomena, however, militate against the practical utility of the galvanometer tests.

Pilomotor System.—The smooth muscle fibers of the skin are under sympathetic innervation. The pilomotor fibers run with the sensory fibers (Higier), each sensory nerve carrying fibers from about five sympathetic ganglia (Higier), and have similar topographical (segmental) distributions. (See Plate II). Mechanical, thermal, and electrical stimuli cause contractions. The erector pilæ reflexes (best observed by side light) are particularly responsive to cold. The frequently felt paresthesiæ, acroparesthesiæ etc., of psychoneurotics depends upon these sympathetic reactions, and their exact observation is of much diagnostic importance. Mackenzie's observations should be consulted by the interested student.[1] The feeling of cold over the abdomen after taking cold water in the stomach is an example of the relationship of the sympathetic innervation of an internal organ and a skin area.[2] A large number of analogous phenomena are known. Thus among them, mechanical stimuli of the plexus pudendi, as in cystitis, rectal exploration, prostatic massage, prostatitis, causes distinct gooseflesh or paresthesiæ in the region of the motor lumbar plexus. Erector pilæ crises are known to occur in tabes; they are at times migraine equivalents. The pilomotor cord synapses follow closely in their responses those of the sweat mechanism. (See preceding paragraph.) (Thomas, loc. cit.).

Psychical stimuli may lead to localized, or more often to generalized reactions. A great variety of extremely important skin hallucinatory experiences are known to occur in dementia precox, and in the psychoneuroses, hysteria, anxiety states, compulsive states (pathological blushing, etc.). Their correlation is only just being understood through the results of psychoanalytic research.

Similarly it is beginning to be questioned, if not affirmed, whether a whole group of chronic skin disorders, involving the pilomotor reactions are not also the results of chronic emotional conflicts in the unconscious, mediated through the vegetative nervous system, with or without

[1] Mackenzie: The Signs of Disease.

[2] Head's Zones, Mackenzie.

related endocrinopathies, dysthyroidisms particularly (endocrino-sympathetic). If this be so appropriate psychotherapy may show the causal relations between the skin disease as a symbol of a psychical compromise and thus the scape-goat be removed.

Bony and Ligamentous Syndromes: Osteopathies, Arthropathies.—Bone and joint disorders due to disturbances of nervous functioning are by no means infrequent. The nervous mechanisms underlying bone development are not clear but at least two large groups of neurological bone disturbances may be separated. They are: those dependent upon disease of the vegetative nervous system (endocrinopathies[1]): and those of spinal origin (Petrén, Dejerine et Ceillier). Psychogenic bony and joint changes are also beginning to be recognized.



FIG. 74.—Osteoporosis congenita in a girl aged nine years. (A. Josefson.[2])

The chief *bony endocrinopathies* are achrondroplasia, osteomalacia, the bony changes of acromegaly, gigantism, infantilism, leontiasis ossea, etc. Hutinel, loc. cit. has collected a large number of bony dystrophies occurring in children which he has referred to endocrine anomalies of the thyroid, hypophysis, thymus, suprarenal and gonads. His monographic study is replete with interesting clinical material and physio-pathological discussion.

[1] Hutinel, V.: Glandes endocrines et dystrophies osseuses, Arch. d. Méd. d. Enfants, 1918 Sterling: Ztschr. f. d. g. Neur. u. Psych. Ref. ix, 1924.

[2] Forh. a. d. Gebiet d. Roentgen, 1916.

Achondroplasia.—This consists chiefly in a defective fetal development of the bones of the extremities (micromelia), with comparatively normal development in all other tissues of the body.

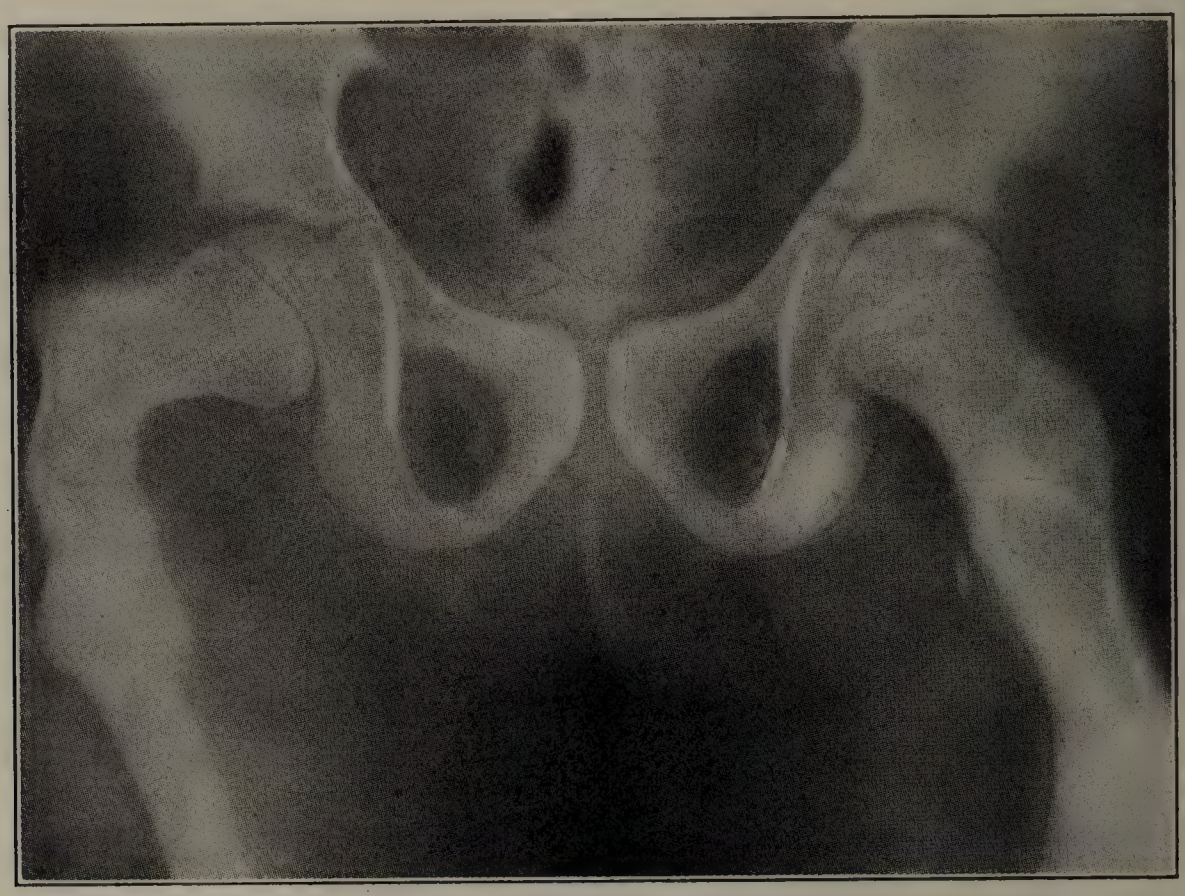


FIG. 75.—Osteoporosis congenita in a girl aged nine years. (A. Josefson.)



FIG. 76.—Micromelia in achondroplasia, showing the trident hand in twenty-one-year. old patient. (A. Josefson.)

Symptoms.—The skull is enlarged, often hydrocephalic, with depression of the nose and prognathism. Shortening of the extremities (micromelia) is characteristic, with exaggerated curvature of the shortened bones. The fingers are nearly all of the same length and radiate slightly, spoke-like, from the metacarpal joints with a tendency to form the trident hand. The thumb, first and second, fourth and little finger, respectively, are arranged in groups. Lumbar lordosis with prominent abdomen is usual.



FIG. 77.—Achondroplasia with micromelia, showing shortened upper extremity with curvatures of bones in twenty-one-year-old patient. (A. Josefson.)

The interpretation of the disorder is difficult.[1] Heredity is prominent, males chiefly being affected.

Osteomalacia.—This is a type of bony softening closely resembling rickets and apparently dependent upon disturbed calcium metabolism through the mediation of the endocrinous glands (thyroid, parathyroids, ovary).

Symptoms.—These are best consulted in text-books of medicine or surgery.

[1] Parhon: Nouvelle icon. d. l. Salp., 1913, **26**, 185.

The bony changes, resulting from pituitary dysfunction (gigantism, acromegaly) are to be referred here. (Studied more in detail under the Pituitary Syndromy.) Thymic gigantism is also to be included in this grouping.



FIG. 78.—Pseudo-epiphysis in second metacarpal bone (proximal). An endocrinous (hypothyroid) stigma. (A. Josefson.)

Vegetative arc disorders are not so well comprehended. The *tabetic arthropathies* have been longest known, although their physio-pathology is still somewhat obscure. Eppinger has interpreted these chiefly as resultants of the cutting off of parasympathetic stimuli through the meningeal implication of the posterior roots. Jelliffe includes in this picture the occasional occurrrence of thyroid defective complicating factors.

Mme. Dejerine and Ceillier[1] have shown that spinal cord injuries may involve trophic synaptic areas and produce osteoarthrophies of a very striking character. Also Petrén, following encephalitis.

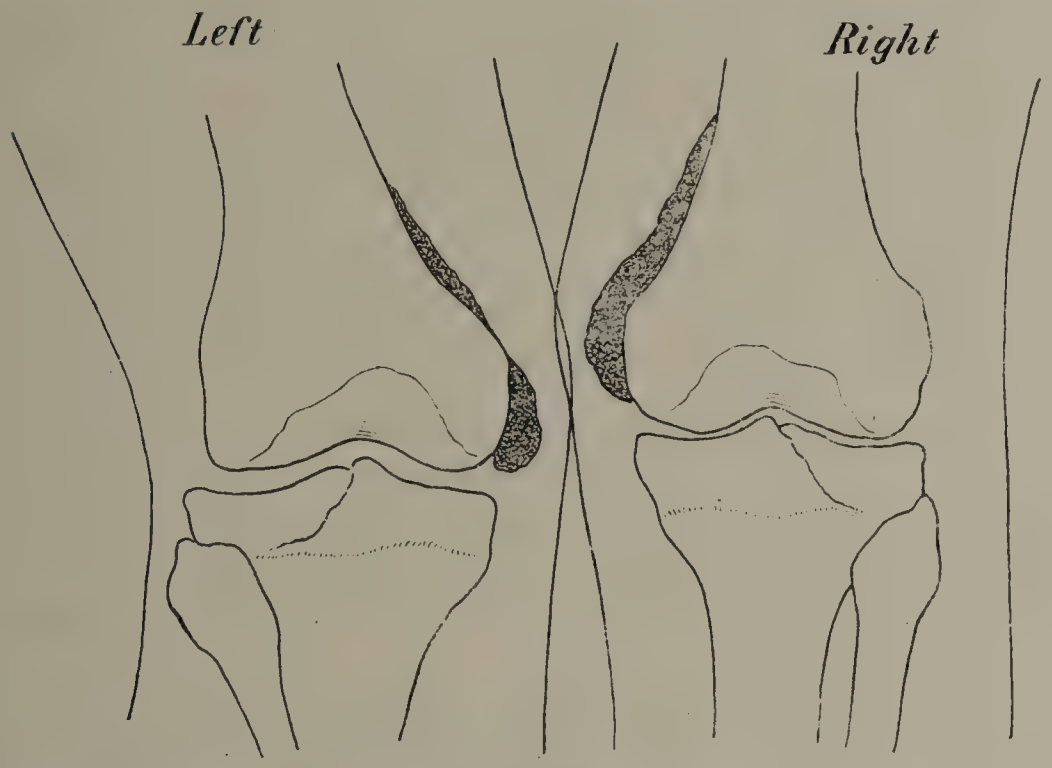


FIG. 79.—Osteoarthropathy of knee after lesion of lumbar cord. Liii Lv with syringomyelic dissociation. Radiograph made six and half months after injury. (Ceillier.)

The numerous bony changes resulting from spinal cord or peripheral nerve injury cannot be followed out here in detail. Some reference might be made to Simon's[2] studies of bone and nerve and their mutual interrelationships. In a series of cases Simon found that as result of purely psychogenic paralysis and contractions a destruction of bone ensued. This destruction was sometimes simply indicated, sometimes slight, but in some cases profound; it involved one bone or several. After complete recovery normal bone structure may develop again. The localization and degree of the changes did not correspond to the sensory supply to the bone, not to the vasomotor disturbances, to the swelling of the tissue, nor to the psychogenic disturbances of sensibility, and the destruction is not a constant occurrence. Neither long continued passive exercise nor massage seem to have any influence on it. Of 12 patients with psychogenic disturbances observed by the author 8 were affected with bone atrophy. Of these, 2 however, had suffered slight bone injury. One had received a wound in the lower third of the tibia which had healed rapidly without plaster cast; eight weeks after the injury a club-foot developed under psychogenic influence. The other patient had sustained a slight oblique fracture of the ulna at its upper part which the roentgen picture showed had healed successfully. If the psychogenic influences had not supervened the hand could have been used shortly after the injury, and the foot of the first patient, two or three weeks after the injury. The degeneration of

[1] Ceillier: Thèse de Paris, 1920. Petrén, Jour. Nerv. and Ment. Dis., 1925.
[2] Simons, A.: Ztschr. f. d. ges. Neur. u. Psych., vol. **37**, p. 36.

bone was much more profound in the foot than on the hand. This is explicable by the relatively greater rigidity of the foot and the greater susceptibility of these bones to functional disturbances. The author is of the opinion that in his two cases the bone atrophy cannot be entirely referred to the continued rigidity of the limbs. The influences of wounds of this sort are more profound than becomes apparent clinically, as is the case in those relatively slight skull wounds which after years cause serious mental disturbances; the bone atrophy primarily due to a deep-seated injury could not get better because of the psychogenic rigidity of the limbs. But in the remaining cases there were no injuries to the bone, and no other influences which could alter the bone structure were active. Here the atrophy is wholly of internal origin. Nonne called attention to the fact that the normal bone findings in many cases of hysteria after prolonged absence of sensory and motor function furnish strong argument against the inactivity as cause of the atrophy, and the author raises the question in connection with his present observations whether there is not a neurotic (reflex) bone atrophy entirely distinct from the atrophy due to inactivity. Nonne's more recent observations together with author's point to a bone destruction of this nature. One of us has also followed closely for a long time this type of interaction and would extend the inferences drawn by Simon and Nonne to include a compensatory hyperactivity which is often observed. Certain of these cases have been diagnosed by roentgenologists as special benign types of giant cell sarcoma.[1]

Arthropathies.—Various types of arthritis constitute very pregnant problems in clinical medicine. It is fairly well recognized that toxic-infectious processes play a predominant role in the causation of many of these. Certain hypothyroidisms (*q. v.*) permit the development of a rheumatoid arthritis, possibly by the reduced capacity of the individual to react efficiently to minimal subinfections, often of cryptogenic origin, teeth, the frontal, malar, and ethmoid sinuses, tonsils, intestines, old vesiculitides, etc.

It is not so clearly understood that other factors may be controlling the pathological processes, and from the hopelessness of much of the heretofore employed therapy in chronic cases it is evident that prevailing conceptions are inadequate. The tooth pulling fad of the early 20th century seems to be a parallel to the ovariectomy fads of the 70s and 80s of the 19th century, and possibly the tonsillectomy enthusiasms of related decades. The epoch making findings of Mme Dejerine and Ceillier show that neurogenic factors play a large part in bony formation, and endocrinological research has also contributed much to the understanding of bone metabolism. The psychogenic discipline is also commencing to show that purely emotional reactions extend deep down in the individual's metabolic processes and must be evaluated in the study of chronic arthritic processes. Syndenham's classic contribution to the relationship between acute gouty attacks and excessive anger outbursts is widely quoted and as widely glossed over. Gout, larvated gout, the gouty diathesis, lithemic gout, etc., made a large

[1] Jelliffe: Neuropathology and Bone Disease, Trans. Am. Neur. Soc., 1923, p. 419.

chapter in the medical literature of the closing half of the 19th century. One looks for them almost in vain in contemporary literature, concealed as it is in a bewildering mass of metabolic anomalies of a more refined chemical technic posited upon an equally confused series of generalizations concerning the thyroid, pituitary, and parathyroid regulations of calcium and phosphorus metabolism.

Certain formulations commence to crystallize out of this mass of bewilderment. In view of the phyletic history of bony formation in its response to geotropic stimuli, and of stress upon bony structure in response to bodily displacement (Apathy, Lane, Semon) through muscular activity (joints), it is no wonder that the questions are hoary in their antiquity and hence difficult of solution. The problems connected with the need and the wish for bodily displacement (labor, etc.) are often too lightly considered by the intellectual worker who does much of his work with language and with purely symbolic concepts. Muscle, tendon, joint and bone work still lie at the basis of productive activity and are inherent in all human effort. Atlas with the World upon his shoulders; Thor with his mighty Hammer; Mercury of the Flying Feet; Jove with his Thunderbolts, these mythical precipitates of a Volksgeist are indices of what *Homo sapiens* would like to be, and failing to be—in a productive—constructive—outlook, may he not lock up his muscle-joint activities in fruitless arthritic bondage? Here *may be* recorded—aided by toxins, bacteria, or other accessory factors, that *lex talionis*, meted out to man, in his effort to supplant the Gods and to wreak vengeance upon a world of reality with which the individual is in fundamental (though unconscious) revolt.

A rapid diminuendo from this philosophic crescendo would attempt to show how particular joints are affected. Clinical experience has seemed to indicate to the authors of this book how the fundamental affective trends finding symbolic expression, at the psychological level, in terms of the unconscious criminal endeavoring to escape from the reality of his desires, have tied up the joints of various individuals in incapacitating arthritic involvements. How the unconscious grasping miserly individual has tied up the hands. The belligerant fist-pounding pugilist has locked up his wrists, elbows and shoulders; the kicking, stamping, hatreds involved the knees and hip, etc. It is here considered highly probable that such factors might advantageously be correlated in the study of the arthritides.

DISEASES OF THE MUSCLES.

The most fascinating themes in modern neurology cluster about the newer concepts of muscle physiology and muscle pathology. Parker's interesting researches, not to mention many others, have tended to show that nerve and muscle have had a simultaneous origin, that stimulus and response have slowly evolved a receptor, a connector and an effector type of organ, which has finally structuralized itself into the anatomical elements which are called nerve and muscle. Out

of these have evolved the earlier primitive unstriped musculature effector organs, glands, etc., and the more complex and duplicated voluntary musculature. This latter, according to these newer concepts is a compound of both elements, which in spite of much histological research is still imperfectly understood.

At the present time, so far as the voluntary muscle system is concerned, it is possible to state that at least three factors are analyzable in voluntary muscle activity: (1) Metabolic activities; (2) tonus (geotropic activities) and (3) kinetic or projicient muscle activities. The metabolic and tonus functions are under the control of the vegetative system, the projicient apparatus under the so-called cerebrospinal system. The functions of metabolic activity are as yet so involved that no adequate generalizations are possible. Myasthenia, as such, whether occurring in unstriped muscle tissue, (spasm of unstriped muscle tissue, cardiospasms, enterospasm, colon spasm, etc.) is not yet completely understandable either in terms of fatigue, toxemia, or emotional (psychogenic) exhaustion. Certain observers have attempted generalizations relative to the tonus and projicient factors which may be referred to.[1]

Striped muscles have a double innervation. The discussion concerning this is extensive. The student is referred to the voluminous literature.

The efferent nervous system (cerebrospinal and vegetative) may be conceived as consisting of two components, physiologically and anatomically distinct: a geotropic system regulating posture, and a kinetic system controlling movement itself. In muscle, the kinetic system innervates the anisotropic contractile mechanism, and the static (Hunt) system the isotropic sarcoplasm. The function of the sarcostyles is movement. The function of sarcoplasm is postural tonus, posture following movement like a shadow. The efferent system consists of three great physiological divisions: (*a*) The segmental nervous system, with components of motility representing reflex movement and reflex posture; (*b*) the corpus striatum, its subordinate spinal systems and cortical connections controlling movements of the automatic-associated type, and (*c*) the Rolandic area of the cerebral cortex and its cortico-spinal system, which is concerned with isolated synergic types of movement. The latter two divisions comprise the suprasegmental nervous system. The essential suprasegmental structure for the control of postural function is the cerebellum. The peripheral nerves and voluntary muscle represent another system. Each of these systems presents anatomical evidences of a kinetic and a static mechanism.

The static and kinetic components of the vegetative nervous system for the control of unstriped muscle are represented in the sympathetic and parasympathetic systems, respectively. These two systems control the posture function and primitive motility of the bloodvessels,

[1] Hunt, J. Ramsey: See Tr. Am. Neur. Soc., 1920. Presidential Address and a group of highly suggestive researches by this author.

glands and viscera. Unstriped muscle, like striated muscle, is composed of fibrillæ and sarcoplasm. The fibrillæ pass from one cell to another, forming a contractile network, and subserve the function of primitive movement. The sarcoplasm is concerned with primitive posture. Posture is the dominant function of involuntary muscle which coincides with the predominance of sarcoplasm. In voluntary muscles, movement is the dominant function, and there is a corresponding differentiation of the contractile mechanism. In both voluntary and involuntary muscles, various types of muscle fiber may be recognized, representing transitions from lower to higher forms.

Contractile function is subserved by two afferent systems, conveying, respectively, sensations of movement and of posture. Muscle sensibility (myesthesia) therefore consists of two components: Kinesthesia, a sense of movement, and statesthesia, a sense of posture. These unite with impulses of similar character from the bones joints, fasciæ and tendons.

A lesion of the kinetic system causes a disorder of movement, and of the static system a disorder of tonus or of posture. This principle holds for both the splanchnic and somatic systems. The kinetic mechanism of the somatic system may give rise to various types of convulsive manifestation; also chorea, athetosis, tremor, etc. Related to the static mechanism are the sudden postural relaxations of epilepsy, myotonia, asynergia, dysmetria and intention tremor. In the psychic sphere, evidences of a dual representation are manifest in convulsions and in catatonia. A similar interpretation may also be given to active and to passive preservation. In the vegetative nervous system, disorders of movement may be related to the kinetic component; *e. g.*, tachycardia, gastric hypermotility and bronchial asthma. Related to the static system are disorders of postural tone; *e. g.*, insufficiency and atony of the sphincters and hypotonic states of the vascular system.

This general viewpoint enables one to distinguish disturbances of muscle function from three general standpoints: (1) Muscle inadequacy as due to (*a*) toxic and endocrine influences; (*b*) psychogenic influences, (*c*) sensorimotor influences.

The vegetative factors are discussed in this section;[1] the the psychogenic factors are taken up in the paragraphs on symbolic neurology, the sensorimotor influences are described in the sections on the cortex, striatum, spinal cord and peripheral nerves.

Myasthenia Gravis.—This is a syndrome. It is still a debatable problem in neurology whether there is one unique condition which may be called by this name in the sense of an Erb-Goldflamm-Oppenheim disease.[2] The earlier descriptions which sought to erect a definite

[1] Curschmann: Das endokrine System bei Neuro-inbesondere bei Myoneuropathien, Ergeb. d. inn. med., 1922, **21**, 467. Deut. Arch. f. kl. Med., 1925, **149**, 129.

[2] See Curschmann, H.: Ztschr. f. d. g. Neur. v. Psych., 1919, **50**, 131, for a valuable discussion of the problem up to 1919 and further Ergebnisse d. inn. med., 1922, vol. **21.** Kuh and Braude: Jour. Nerv. and Ment. Dis., 1913, **40**, 617. Starr: Jour. Nerv. and Ment. Dis., 1912, **39**, 721. Keschner, M.: Arch. Neurol. Psych., 1927,

disease are probably no longer tenable in a rigid sense. Later study has emphasized that a number of different trends or types have been included in the group.[1] At all events, either fatigue, toxic (endocrine), infectious, or psychogenic processes interfere with *muscle function* in such a manner as to produce, according to present rough analytic methods, a fairly homogeneous picture. This is what is called *myasthenia gravis* today.

Thiele-Leschziner, emphasizes the difficulty in handling the proper amount of carbohydrate for muscle action from faulty adrenal-pancreatic symbiosis, and Patrzek has described an atypical case on a definite adrenal defect basis.[2] Marinesco speaks of a suprarenal dysvegetative pathogeny.

The syndrome is infrequent. It was separated from the progressive bulbar palsies of organic nature by Erb (1878) and later studied by Oppenheim (1887), who termed it a myasthenic paralysis without perceptible anatomical foundation. In 1891 Jolly described the characteristic electrical reactions occurring in the muscles, termed the myasthenic reaction.[3]

The early symptoms in the purer type of case which usually come on between fifteen and thirty years of age, usually involve the facial muscles, particularly those of the upper lid, causing ptosis. Diplopia from paresis of an ocular muscle also may be an initial symptom. The two often occur together (asthenic ophthalmoplegia). The patients note the beginning fatigue of the muscles, which perhaps intact in the morning on awakening, show fatigue signs at night. This muscular asthenia then progresses slowly to distinct paresis. Other cranial nerve innervations then show a similar asthenia. Difficulties in chewing or of swallowing, or of speaking develop. The muscles of the neck may also be involved. Whatever group is involved the chief feature is the great fatigue which develops very rapidly upon the use of the muscles.

Calcium metabolism is greatly disturbed. Tests of the blood following muscular actively show marked calcium impoverishment. The potassium, phosphorus metabolism has not been sufficiently studied.

Any muscle or muscle group of the body may be affected. Dyspnea and tachycardia are among the rarities of implication of the respiratory and cardiac muscles. Intestinal, bladder or rectal functional disturbances also occur (Curschmann). Sensory disturbances are not characteristic. Pains may occur.

Leukocytosis is usually present. The reflexes are not implicated. In some instances fatigue of the tendon reflexes has been recorded.

[1] See discussion of paper of Dana by Sachs, Jelliffe and others, New York, Neurol. Soc., November 1, 1921. Jour. Nerv. and Ment. Dis., January, 1922, **55**, 48 et seq. Boudon: Thèse de Paris, 1909. Claude et Lévy-Valensi: in Gilbert and Carnot: Traité, 1922, vol. **33**.

[2] Theile-Leschziner: Ztschr. f. klin. Med., vol. **86**, p. 390. Patrzek: Ztschr. f. d. g. Neur. u. Psych., 1921, **63**, 155. Marañon: Bull. Soc. med. d. Hôp., November 12, 1925, p. 433.

[3] Oppenheim: Die myasthenische Paralyse, 1901.

The chief feature is the rapidly developing fatigue of the muscle. This is best demonstrated by faradic stimuli. These cause a rapid loss in the excitability of the muscle until it no longer reacts to the intermittent faradic current. Hoffmann has shown that this is largely influenced by the rate of the interruptions. With seventy interruptions per second the myasthenic reaction develops promptly, with fifteen it does not. Continuous faradic stimulation produced a similar myasthenic fatigue curve. This myasthenic reaction seems to separate the disorder from other forms of muscular fatigue such as occur in bulbar palsy, medullary syphilis, multiple sclerosis, Addison's disease, exophthalmic goiter and the fatigue of intermittent claudication. There are certain analogies with this last disorder which are not yet cleared up.

Atrophies develop in the affected muscles but there are no definite indications of the reaction of degeneration. Certain transitional cases which show relationships to distinct organic (nuclear, encephalitis) cases may evidence electrical changes approaching R. D. Fibrillary twitches in the affected muscles are not the rule, but they have been observed.

That which has been termed myasthenia gravis runs a very variable course with at times marked remissions. It has been known to develop rapidly in three or four months with fatal issue in from one to three years and, on the other hand, it has been known to extend over fifteen to twenty years The outcome is usually fatal, but certain cases cease to progress.

Little is known of the underlying causes. In certain cases, epidemic encephalitis, influenza, tonsillitis and pneumonia have been shown to be responsible. Status thymicolymphaticus is frequent. Many cases show interrelated thymus, parathyroid and adrenal compensating effort syndromes which do not seem to overcome persisting localized vagotonus of the sacroplastic substance.

Very little is known concerning the unconscious psychical states. One patient examined (Jelliffe) showed highly dynamic father, money and sadistic complexes. Father injustice, relative to money and ineffective fighting were frequent dream symptoms. A nightmare in which sadistic overcoming of the father was prominent was followed next day by grave myasthenic signs, and death. Just what this means must be left for further study.

In the autopsy records the pathological lesions are not constant. In the greater number of cases the muscles are swollen, edematous and infiltrated with lymphoid cells.[1] These changes have not been interpreted. It is possible that they are edemas due to disturbance of the vegetative nervous system control, in which case myasthenia gravis is to be allied with the circumscribed edemas and in some way

[1] Frugoni: Riv. Cirt. d. Cl. Medica, Firenze, 1907, **8**, No. 37. Also consult Claude and Lévy Valensi, loc. cit., for most comprehensive recent study and Keschner's excellent recent review, loc. cit. Marinesco on role of vegetative system, Ann. méd., 1925, **17**, 437.

related to calcium, potassium ion balancing. Thymus tumor cases are known.[1]

The first important therapeutic agent is rest; absolute and prolonged. The second is psychotherapy. Organotherapy has been tried, with as yet little results, but it probably has been entirely too crudely applied. Careful attention should be given to a complete survey of the functions of all of the endocrinous glands, and if a lack of balance be found an attempt should be made to restore the balance. Artificial feeding is at times necessary. Atropine has been of service occasionally as has also calcium. Strychnine, since it is a definite sympathetic stimulant is often of signal service. There are probably syphilitic cases which are cured by specific therapy. Alcoholic preparations are to be avoided, as are also mechanical forms of stimuli, particularly severe massage.

Thomsen's Disease.[2]—**Myotonia Congenita.**—This is a very rare syndrome first described by J. Thomsen in 1876. Its relationships to other myogenic syndromes is very obscure, although related closely to the myasthenic syndrome. Hereditary features have seemed prominent; Barbé has published a recent case without such hereditary factors,[3] also Rimbaud and Jourdan. A constitutionally inferior thoracic parasympathetic control of the muscle metabolism seems to be an outstanding feature.[4] This results in an increased threshold of the synapse preventing immediate passage of the voluntary stimulus. This is often associated in the affected families with other signs of inferiority; neuroses, psychoses, tetany, etc.

The chief anomaly is one affecting the muscles. At the beginning of any voluntary movement the patient finds it difficult to overcome a muscle hypertonus. This makes the muscles stiff and unyielding. After repeated efforts the resistance gradually disappears and in a few minutes or more the muscular activity becomes normal. This limbering-up effect is lost after a cessation of the movements. Any group of muscles may be affected, but the lower extremities are oftenest involved. This makes the beginning of walking difficult. In the upper extremities a similar condition makes manual movement difficult. A patient cannot readily loosen his grasp of an object. Talking and eating, etc., may be similarly affected. Changing the tempo of a movement increases the difficulty and emotional stimuli invariably augment the stiffness and awkwardness. Mechanical stimuli cause welts to appear which subside slowly. Atypical cases are reported, in some of which the disorder appears intermittently. (Compare with periodic paralysis, also paramyotonia congenita of Eulenburg[5] and the dystonic dystrophies of Curschmann.

[1] Bell: Jour. Nerv. and Ment. Dis., 1919, **45**, 130.

[2] Thomsen: Archiv. f. Psychiatrie, 1892. Koch: Ueber Thomsensche Krankheit, Leipzig, 1914. Schlanck: Deut. Ztschr. f. Neur., 1920, vol. **67**.

[3] Barbé: Progres Medicale, 1919, p. 408. Rev. Neur., 1922, p. 26.

[4] S. de Boer: Ztschr. f. Biologie, 1914, **45**.

[5] Jendrassik: Lewandowsky Handbuch, vol. **2**; Deusch. Ueber myotonische dystrophie, Deutsch. Ztschr. f. Nervenh., 1926, **92**, 171.

The pathological changes are slight. Muscle-cell hypertrophy, analogous to that seen in myasthenia gravis, is described.

The disorder begins early, is very chronic, is not fatal in itself, nor does it seem to get well spontaneously.



FIG. 80.—Myatonia atrophica. Note dropped feet, atrophy of hand muscles, and facies. (Abrahamson.)

No therapy has been shown to be effective. Strychnine is temporarily valuable. If the present hypothesis is of value some result should follow from polyglandular therapy, particularly from the use of such substances as influence the bivalent kations, Ca, K, Mg, etc., to regulate the electrical resistances in the motor synapses.[1] No study of the psychical components are published. One personally

[1] Johnson and Marshall: Quart. Jour. Med., 1915. Maas and Zondek: Ztschr. f. d. ges. Neruol. u. Psychiat., 1920, vol. **59**.

observed case presented nightmares in which inability to move was prominent. This is a common nightmare of adolescents. It has some significance to striatal pathology (see epidemic encephalitis and bradykinesis.

Myatonia Atrophica.—*Myotonia dystrophica of Hauptmann.*—This rare syndrome[1] is possibly a definite clinical entity or a variant of Thomsen's disease, as Pelz (1907) first announced. It is characterized by late onset, twenty to thirty years, limitation of myotonic reaction chiefly to the closing of the fist, the tongue movements, myopathic facies, atrophy of jaw muscles, sterno-cleido-mastoid, extensors of arm, especially supinator longus, atrophy of peronei, loss of tendon reflexes (late) with ataxia, cataract, loss of hair, atrophy of testicles, impotency or frigidity, increased reactivity to pilocarpine, vasomotor disturbances, increased tears and saliva, and general emaciation.

It is possibly a vegetative nerve disorder, showing a medley of sympathicotonic and vagotonic signs (due to an hereditary defect in the vegetative nervous system) which causes muscular dystrophies to develop on an endocrinopathic basis.

The Muscular Dystrophies or Myopathies.

This very large and extremely motley group has also been built up of a variety of forms since Duchenne,[2] in 1849, first described the fatty pseudohypertrophies, and later, in 1868, spoke of them as myosceloses. Leyden[3] (1876) and Möbius[4] (1879) described certain hereditary forms, while Erb,[5] in 1883, first brought some order in the confusion of the atrophies and dystrophies by showing that in certain forms the lesion was predominantly muscular and not nervous. He made the first practical synthesis. Landouzy and Dejerine,[6] in 1884, described their well-known form, and separated it from Erb's juvenile type. Since that time the group has been better unified, its limits better recognized, and the various forms within it more thoroughly studied.[7]

The myopathies make a fairly consistent group, although the forms may not resemble one another clinically at different periods of their development, yet they have a number of common clinical factors.

Familial occurrence is frequent. The symptoms usually begin at an early age; the muscles become weak gradually and atrophy in a peculiar manner, in that true hypertrophied fibers are mingled with atrophied

[1] Batten and Gibb: Brain, 1909. Curschmann: Deutsch. Ztschr. f. Nervenh., 1912, vol. **49**; 1915, vol. **53**; 1922, vol. **74**. Hauptmann: Ibid., 1916, **55**, 53; 1919, **63**, 206. Higier; Ztschr. f. d. ges. Neurol. u. Psychiat., April, 1915. Bezold: Schmidt's Jahrb., August, 1919, p. 330.

[2] Union Méd., 1853.

[3] Klinik de R. II, 1876.

[4] Volkmann's Klinik, No. 171.

[5] Neurol. Centralbl., 1883, p. 452. Bachmann: Deutsch. Ztschr. f. Nervenh., 1926, **92**, 28.

[6] Comptes rendus, 1884, p. 53.

[7] Batten, The Myopathies or Muscular Dystrophies, Quart. Jour. Med., April, 1910. Lorenz: Krankheiten d. Muskeln, 1904. Jendrassik: Handbuch d. Neurol., 1911.

fibers. The muscular atrophy may involve all of the muscles equally, or may be irregularly distributed both as to the body in general or within the muscle itself. Reaction of degeneration and fibrillary contraction are usually wanting, although a gradual loss of electrical excitability goes on coincidently with the atrophy.

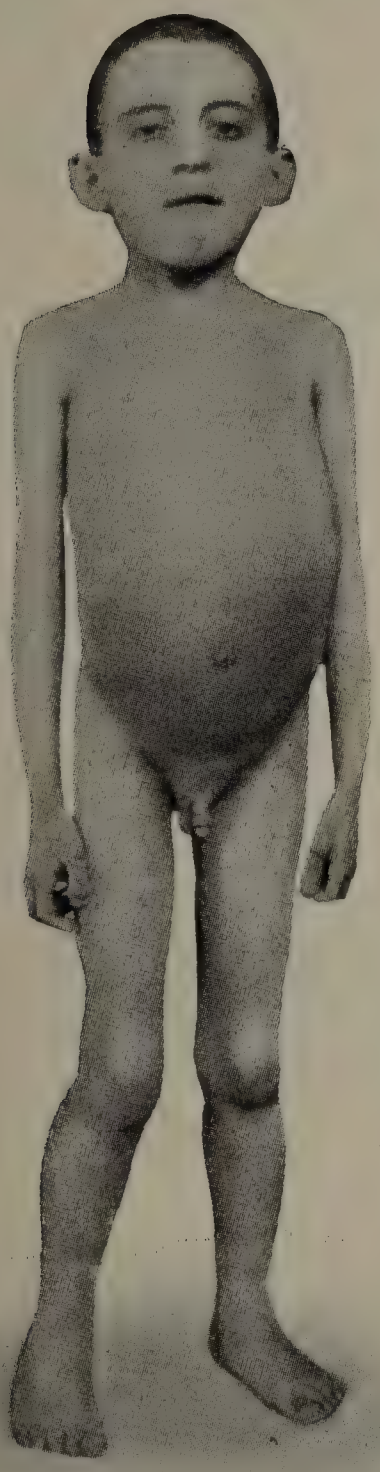


FIG. 81.—Pseudohypertrophic myopathy. Early stage. (Jendrassik.)



FIG. 82.—Pseudohypertrophic myopathy. Stage of atrophy. (See Fig. 81.) (Jendrassik.)

Certain muscles, pectoralis major, rhomboid, serratus magnus, are oftenest the seat of early atrophy. These are also characterized as congenital aplasias (Bing). The muscle electrical reaction curve is striking.

The tendon reflexes gradually disappear, but the Achilles is apt to persist, or occasionally be increased, especially with much pseudo-

hypertrophy. Sensory disturbances are usually absent, likewise bladder and visceral disturbances. Pseudocontractures with limitation of movement are frequent, causing peculiar positions. The patients hop like frogs. Bony dystrophies are also frequent, mostly showing in thinning of the long bones, with cranial deformities, deformed



FIG. 83.—Pseudohypertrophic myopathy. Later stage of atrophy. Compare Figs. 81, 82. (Jendrassik.)

hands, short hands and short feet. More recent biochemical researches are developing a large number of anomalies in metabolic relations. Among these impaired creatine transformations are striking.[1] A number of accessory lesions have also been described such as acromegaly, gigantism, idiocy of thyroid type, leukoplakias, vitiligo, diseased

[1] Gibson and Martin: Jour. Biolog. Chem., 1921, **49**, 319. See for study of creatine anomalies and literature to 1921.

pineals, etc., all indicating muscle metabolic disturbances dimly related to neuroendocrine disorder.[1]

Pathology and Pathogeny.—Erb based his synthesis upon the changes he found in the muscles, but at the same time was inclined to attribute them to lesions in the sympathetic cells of the cord. These muscle changes consist in the main in hypertrophy and atrophy and splitting of the muscle fibers, proliferation of the nuclei, new connective-tissue proliferation with hyperplasia of the vascular tissues and fatty deposition. Macroscopically the muscles have lost their normal color, varying from pale pink to dark red. In places where the muscle substance has entirely disappeared white connective tissue is apparent. The muscle plates are frequently missing.

Loss of cells in the ventral horns has been described by Holmes[2] and others. Whether these are primary or secondary cannot yet be determined.



FIG. 84.—Pseudohypertrophic myopathy. Later stage. Compare Figs. 81, 82, 83 (Jendrassik.)

The changes in the bones is in the nature of a trophic rarefaction.

The newer studies on the vegetative nervous control of muscle metabolism affiliates this entire group to the vegetative nervous disorders, in which an endocrinous myogenic disorder is at the present time the most practical hypothesis. Progressive vegetative muscular dystrophy is the term Tcherbak would use.[3]

No work on the symbolic levels has yet been done.

[1] Timme: Arch. of Internal Medicine, 1917. Janney, Goodhart and Isaacson: The Endocrine Origin of Muscular Dystrophy, Arch. Int. Med., 1918, **21**, 188, for complete discussion of the problem. Curschmann: Erg. d. inn. med., 1922, **21**, 467. Schaefer: Mitt. d. g. Grenzg. M. u Ch., 1923, **73**, 128 (bibliog.)

[2] Rev. Neur. and Psych., 1906, **6**, 136.

[3] Rev. Franco-Russe-de med. et de Biol., June-September, 1925.

Forms.—The forms are many and show considerable intermingling. They are:

1. Pseudohypertrophic (Duchenne).
2. Juvenile (Erb).
3. Facio-scapulo-humeral (Landouzy-Dejerine).
4. Atrophic myatonia congenita (Oppenheim).
5. Distal (Gowers).
6. Mixed and transitional forms.

1. *Pseudohypertrophic Type* (Duchenne, 1849).—Semmola, in 1834, and Costa and Gioja, in 1836, antedated Duchenne in describing these cases, but pictures of earlier centuries give evidence of its presence. It is the type most frequently observed. It is more common in males (3 to 1) and usually begins during childhood. A hereditary history is very frequent. The parents first notice a certain clumsiness



FIG. 85.—Pseudohypertrophic myopathy. Later stage. Compare Figs. 81, 82, 83, 84. (Jendrassik.)

in the gait of the child, then the position of the body is peculiar, the head being bent forward, and the cervical vertebræ are particularly prominent. There is an early beginning lumbar lordosis. The patient waddles then commences to find it hard to go up stairs—often trips and falls. On rising from a recumbent position the arms are called in to aid, and the mode of rising is unique. The patient climbs up his legs, as it were, with his arms. In the final stages the patient is unable to raise himself at all.

The shoulder-blades are freely movable and rise with the elevation of the arms. Atrophies are apparent.

The gait varies somewhat according to the muscles chiefly involved. It is often wabbly, like a pregnant woman; at times it has a high step character; again the patient walks on his toes. The lower limbs often show marked hypertrophy in the early stages, the calves are plump and firm.

The atrophy advances unequally. The muscles most affected in the lower extremities are the psoas, glutei, quadriceps, sartorius,

adductors, gastrocnemius and soleus; in the trunk the rectus abdominis, latissimus dorsi, erector spinæ, rhomboidei, infraspinatus, serratus, trapezius, and pectoralis major, while in the upper extremities the muscles mostly implicated are the deltoid, biceps, brachialis, and brachioradialis.



FIG. 86.—Pseudohypertrophic myopathy. (G. Hammond.)



FIG. 87.—Pseudohypertrophic myopathy. (G. Hammond.)

In advancing cases all of the muscles go, save perhaps those of the face. In rare cases the face is involved (myopathic facies), and in a few cases the vagus is implicated. In the less advanced cases many of the distal muscles can be utilized. The patients usually die of intercurrent disorders after many years of illness.

2. *Juvenile Form* (Erb).—This usually develops about the age of puberty, with weakness and atrophy in the shoulder girdle. The deltoid may show hypertrophies. The arm is usually thinner and more

atrophic than the forearm and typical winged scapulæ develop. In walking the patients not infrequently bend forward from weakness of the trunk and support the back by holding the thighs. Pseudohypertrophy of the calves is not uncommon. Timme[1] has reported on a benign type due to a polyglandular disturbance in which predominant pineal disturbance was inferred. Other polyglandular endocrinopathies may be complicating factors.

3. *Facio-scapulo-humoral Type* (Landouzy-Dejerine).—Here the facial atrophies usually develop early, particularly the orbicularis oris. The sphinx-like face develops, the patients are unable to whistle, tapir mouth is often present, and the smile is distorted. The eyelids hang and cannot be closed completely. The shoulder-girdle

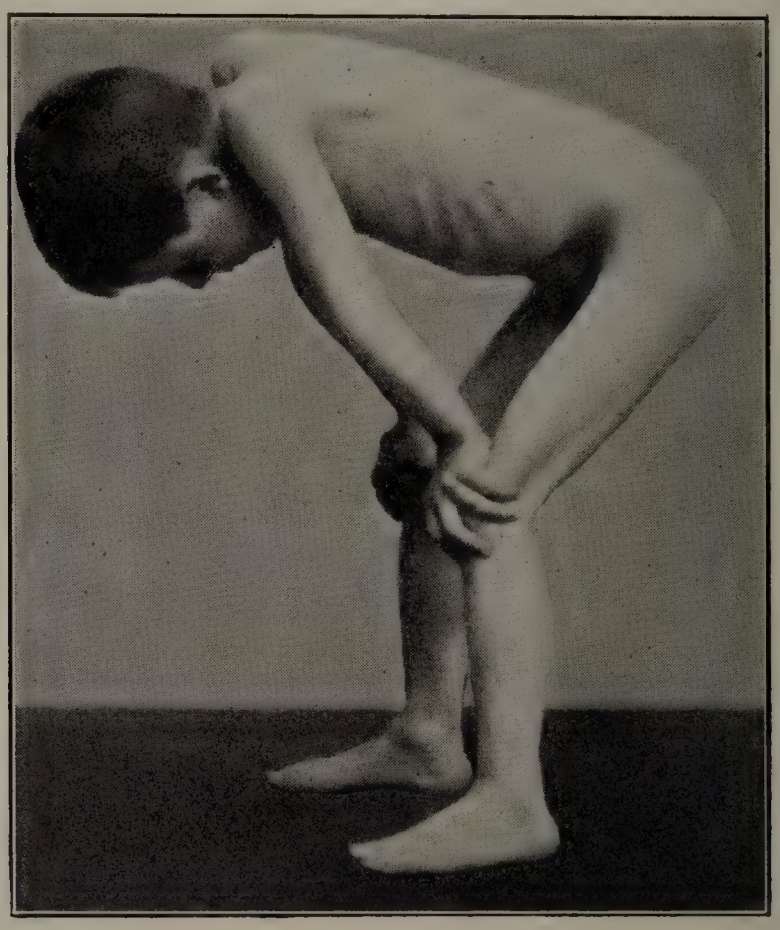


FIG. 88.—Pseudohypertrophic myopathy. (G. Hammond.)

atrophy then advances, the waist is small and wasp-like and the chest flattened; finally the dystrophic process becomes universal.

4. *Amyctonia Congenita*—Myatonia Congenita—Oppenheim[2] (Wilson).—It is not certain whether this disorder should be included among the dystrophies or not. Pathologically it seems identical, clinically it is quite at variance. Spiller made the first autopsy. The number of cases known (about 60—1911) prevents a definite answer at the present time.[3] Rothmann is inclined to ally it with the Werdnig-Hoffmann spinal nuclear atrophies as a congenital variety.

[1] Arch. Int. Med., 1916, **19,** 79.

[2] Monats. f. Neurologie u. Psychiatrie, 1900, **8,** 232.

[3] Latest literature: Collier and Holmes, Brain, 1909; Batten, loc. cit.; Cassirer, Handbuch d. Neur., 1911; Griffith, Arch. Kindhk., 1910; Griffith and Spiller, Am. Jour. Med. Sci., August, 1911. Hübner: Neur. Ztbl., 1918, **37,** 370. Brouwer, B, and Schippers: Psch. en Neur. Bladen., 1914, No. 45, with complete literature to 1914. Kerley and Blanchard: Arch. Pediat., 1927, **44,** 26. Stransky: Monatsschr. f. Klin., 1926, **34,** 24. Davidenkow: Ztschr. f. d. ges. Neurol. u. Psychiat., 1926, **104,** 596.

Symptoms.—The disorder is usually congenital, hypotonia is characteristic, with loss of tendon reflexes. Active motion is impaired by reason of weakness, but the limbs are not paralyzed. Usually the lower limbs are involved, in half of the cases the upper, and in a few those of the trunk and neck. These little patients kaleidoscope as it were. The facial muscles are usually spared. The intercostals are but little affected.

Electric reactions are normal, or show quantitative reduction. The knee- and Achilles-jerks are usually absent; those of the upper extremity less constantly gone. Atrophy is not definite, nor is pseudohypertrophy present. Mechanical irritability and fibrillary contractions are absent.



FIG. 89.—Landouzy-Dejerine myopathy.



FIG. 90.—Landouzy-Dejerine myopathy.

Contractures are not uncommon. The sphincters are intact. Sensibility is intact, also the special senses, and the children are usually bright mentally. The general condition is good, and vasomotor disturbances are absent.

The disorder has some tendency to improve, although Batten claims that not one has gotten well. Some of the patients learn to stand, but rarely unaided.

Intercurrent disorders, particularly respiratory, cause death in the majority.

5. *Distal Type.*—Gowers-Spiller,[1] and Spiller[2] first definitely separated this myopathy from the apparently related Charcot-Marie-

[1] Brit. Med. Jour., 1902.

[2] Jour. Nerv. and Ment. Dis., 1906.

Tooth atrophy. It varies little from this latter save in the absence of sensory disturbances.

Treatment.—This has been very unsatisfactory thus far in this entire group. The pathogenesis is still to be worked out. The most hopeful of the newer suggestions, as yet only tried out in a few cases, is the use of muscle substance preparations. Carnot has experimented with fetal muscle substances trying to find possible regenerating elements. Testicular and suprarenal lipoids at times stimulate muscle growth. These are purely empirically used remedies. A careful study of the heredity for endocrinopathic organ inferiority may help in the use of the glandular products. Thymus and pineal have been used. Until the activities of the sympathetic and vagus fibers in their control of muscle metabolism are better understood, medicine will remain in the dark respecting this group. These patients should be systematically studied by the vegetative methods relative to neurometabolic factors.

BLOOD SYNDROMES.

Our reliable knowledge is only beginning to dawn relative to the control of the vegetative system upon the blood formation. This is a most promising field in vegetative neurology. The vegetative nerve supply of the bone marrow has just begun to be investigated[1] and important results relative to the mechanism of the formation of the different blood constituents are accumulating. The reaction to toxic agents, the mechanism for eosinophilia,[2] of various anemias, of the various leukopenias and leukoplasias are being comprehended through the vegetative nervous system supply of the blood-making organ.[3] The best studied and apparently most widely observed blood syndromes of vegetative disorder are chlorosis and eosinophilia.[4]

Chlorosis.—From the time of the early hypotheses of the retention of menstrual blood as the cause of chlorosis to the present this problem of the etiology of chlorosis has been extremely obscure. Vegetative nervous disease hypotheses have not been neglected, however. Sydenham (1705) and Grawitz among modern hematologists, called it hysteria. What they understand by hysteria is hard to envisage. Copeland, Hoefer, Eisenmann, Brinton Hicks and the Genoa school (Giovanni) teach other views. The general formula adopted by them is that chlorosis is an endocrinosympathetic dystrophy, and exists chiefly in two forms, as a thyroid and a suprarenal chlorosis. It is originally ovarian, the hormones of the interstitial ovarian cells (?) being implicated. What the real dynamics of the situation is, is still obscure.

[1] Drinker: Am. Jour. Physiol., June, 1916. Drinker, C. K., Drinker, K. P. and Lund, C. C.: Am. Jour. Physiol., 1922, **62**, 1.

[2] Schwenker u. Schlecht: Zeit. f. klin. Med., 1912, **76**, 77.

[3] Skorazewski: Zeit. f. exp. Path., 1912. Borchardt: Deut. Arch. f. klin. Med., 1912, **106**, 182. Carpi: Berl. klin. Wchnschr., 1910, 2059. Burr: Jour. Nervous and Mental Disease, 1899, 30. Kaplan: Am. Jour. Med. Sc., 1911, **142**, 702.

[4] Schwarz: Eosinophilie, Lubarsch u. Ostertag's Ergebnisse; Vagotonic Leucopenia, Deutsch. med. Wchnschr., 1923, No. 8.

Eosinophilia.—Many forms are observed. The best types known are those due to various parasites (uncinaria, tæniæ, trichinæ) and to increased activity of the adrenal glands. Eosinophilia is a frequent accompaniment of vagotonic states and is found widely in the agitated depressions of depressed manics, anxiety hysterias, and anxious compulsive states. Here fear, conscious or unconscious, is the etiological factor. Fear also induces the increased adrenalin activity.

Klinkert,[1] Wollenberg, Engelking, Breuer and other workers have brought out much important information which can only be referred to here. Breuer comments on the constantly increasing importance of the blood as an index of vital processes. In constitutional diseases the blood shows changes which seem to suggest pathological influence on the vegetative nervous system, acting through the endocrine glands. Lymphocytosis is the rule in constitutional diseases. The influence on the vegetative nervous system from neurotropic products of bacteria also has to reckoned with. Neurological hematology is in its infancy. One of us (J) has been studying certain forms of chronic pernicious anemia and finds psychogenic factors at deep unconscious levels indicating the need for psychoanalytic research in this profound type of blood alteration.[2]

FATTY SYNDROMES.

Obesity.—The exact mechanisms underlying fat metabolism are not completely understood,[3] but that the vegetative nervous system control of fatty trophism is specific is fairly certain. From a chemical point of view the synthesis seems to start with glucose which on oxidation forms pyruvic acid. A hormone action then converts this into acetaldehyde and carbon dioxide. Condensation produces higher ketone acids which finally by further condensation and polymerization build up fatty acids. The whole process is reversible. What the hormones are and how controlled is unknown.[4] Endocrinous disorders—gonads, hypophysis, possibly pineal, are known to modify the oxidations and permit fat storage. These take place in very characteristic fashion and some have been discussed under the heads

[1] Klinkert, D.: Das Problem der Konstitutionellen Eosinophilie. Zeit. f. d. klin. Med., Heft 1 and 2, 89. Wollenberg, H.: Vegetativen Nervensystems and weisse Blutbild, Zeit. f. d. klin. Med., 1921, Heft. 1-3, 92. Breuer: Ibid. Deut. Zeit. f. Chirurgie, 1921, Heft. 4-6, 164. Engelking. Polyzythämie als vererbbare Störung der innerere Sekretion. Deut. med. Wchnschr., October 7, 1920, p. 1140.

[2] Jelliffe, S. E.: Psychopathology and Organic Disease, Am. Neur. Assoc. Tr., 1922. Archives of Neurology and Psychiatry, December, 1922. Multiple Sclerosis and Psychoanalysis, Am. Jour. Med. Sci., May, 1921. Paleopsychology and Somatic Disease. Psychoanalytic Review April, 1923. Psychopathology and Somatic Disease, loc. cit. Groddeck, G.: Psychische Bedingtheit und psychoanalytische Behandlung organischer Leiden, Leipzig, 1917, Ueber die Psychoanalyse des Organischen im Menschen. Internat. Ztschr. f. d. a. Psychoanalyse, 1921. Das Buch von Es. Archiv of Psychoanalysis, 1927, Deutsch, Schwarz and others. The work of Schwarz on Psychopathology of Organic Disease is specially valuable.

[3] Goering: Zeit. f. Konstitutionslehre, vol. **8**, Heft 4.

[4] Maclean: Lecithin and Allied Substances, 1918. Fraenkel: Arzneimittel Synthesis, 1927.

of adiposis genitalis of pituitary and of gonadal origin, eunuchism and eunuchoidism. Other syndromes will be taken up here.

Lorand described an exogenous—possibly pancreatic—type, occurring in big eaters and developing diabetes from overwork of the oxidizing mechanisms—pancreatic suprarenal(?), and an endogenous type of endocrinous origin. Thus von Noorden differentiated a pancreatogenic obesity and a thyrogenic obesity. Fröhlich separated his hypophyseal type. Kraus has described a variety of this, pilous cerebral adiposity. In addition a group of localized lipomatoses are to be recognized. These are the various types of lipomatoses of which the adiposis dolorosa of Dercum is an extreme form rather than a separate disease since in many of the milder types of symmetrical and circumscribed lipomatoses pains, psychic and neurotic disturbances are seen.

Adiposis Doloroso.[1]—Dercum (1888) first named one of these syndromes. It is characterized by adiposity, pains, general muscular weakness and psychical changes. The patients, mostly women (6 to 1),



FIG. 91.—Adiposis dolorosa. (Dercum.)

averaging between thirty and fifty years, with a tendency to family obesity, slowly develop fatty deposits, often enormous in size. This adiposity may show as nodular deposits (symmetrical lipomatosis), varying in size from a bean to an apple. They may be circumscribed or the adipose deposits may be generally diffused throughout the entire body. The hips, shoulders, upper arm and abdomen are predilection sites. The skin is tense. The fatty deposits in the well-developed types are often painful to pressure, especially at nodular points, and even at times before there is much fatty infiltration. Hyperesthesiæ and paresthesiæ in the form of tingling, burning, numbness, etc., are frequent. Spontaneous pains occur with some. They are sharp and intermittent, localized in the skin or more deeply, and often increase with motion. Asthenia is a marked sign and psychical alterations are the rule. These latter are mostly in the nature of depressions with, at times, suicidal ideas, irritability, capriciousness and other signs

[1] Hirschfeld: Ztschr. f. d. ges. Neurol. u. Psychiat., ref., vol. **6.**

closely resembling manic-depressive states (*q. v.*) with flight of ideas and confusions. Other patients show marked deteriorations.

Various anomalous conditions have been observed with different patients, such as vasomotor signs with edemas, ecchymoses, hemorrhages, and pigmentation. Hyperidrosis, anidrosis, and trophic changes also are recorded. Various combinations with other members of this vast collection of chemical metabolism anomalies are numerous. Some of these have been hyperthyroidism, myxedema, scleroderma, Raynaud's disease, pluriglandular dysfunction, etc.

The disorders usually progress slowly and vary greatly in intensity from year to year. The patients recover spontaneously or finally die of intercurrent disease.

The organs involved have been the hypophysis and the thyroid chiefly, but the exact relationships are still uncertain. Hypophysial tumors are not infrequently found, ovarian disease is present in some, vascular neurotrophic disorders are present in still others.

Lipodystrophies, of which that just spoken of is but an accentuated type, occur in a great variety of forms. See special monographs.[1]

Treatment.—Opotherapy with thyroid has been of service in those cases in which a diseased thyroid has been associated. Ovarian extract has helped the ovarian defect types. Electricity, hydrotherapy, diet and general hygienic control has been of service in others. Here as in other of the endocrinopathies a careful survey of the heredity may give useful clues as to the probable constitutional defects and to their partial alleviation by properly selected opotherapy.

[1] Boissonas, L.: Revue Neurol., October, 1919. Smith: Johns Hop. Bull., 1921. Long et Bickel: Ann. f. Med., 1924, **16**, 107. Marañon: Endocrinology, 1926, vol. **10**.

CHAPTER III.

THE ENDOCRINOPATHIES.

INTERNAL SECRETIONS.

MEDICAL biology has not yet arrived at hypotheses sufficiently comprehensive to conceive satisfactory schemes which can explain all of the relations of the vegetative nervous system to the control of the viscera. Some suggestions have been offered concerning the action of the vegetative nervous system upon the gastro-intestinal tract, and also some ideas relative to the intricate adjustments of internal and external organs brought about through the bloodvessels have been reviewed. Special neurological problems in which direct modification of the vegetative systems in the eyes, nose and throat, lungs, heart, skin, blood organs and bones have been touched upon, all too hurriedly. The problems of cellular adjustment now demand attention. These concern the vital phenomena of anabolism and katabolism, and the adjustment of all of the organs of the body involved in the elaboration of special substances, which are of importance to the metabolism of the rest of the organism.[1]

Thus, what part is played by the nervous system in the carbohydrate oxidations of the body, acting chiefly through the lungs, the liver, the pancreas, and suprarenals? The substances of the hypophysis, its hormones or active principles, what are they; and what is the interrelationship between them and growth as seen in the clinical phenomena of infantile dystrophies, of acromegaly, and a number of related conditions? Similar questions arise for discussion concerning the thyroid, the thymus, and the adrenals. The pineal gland is also a glandular formation, likewise the parathyroids, with their problem of regulating the calcium metabolism not yet certainly disposed of. What nervous mechanisms keep the other constituents of the body plasma in a state of equilibrium, so that all types of functioning may go on, physico-chemical, sensorimotor, and psychical?

This entire group of questions cannot even be *asked* here. It can only be said that in their consideration, one sees an entirely new country opening up which promises to greatly modify the geography of our present neurological schemes, since Brown-Séquard in 1889 reached out for immortality by his use of testicular substances. It may be recalled that as early as 1775 Theophile de Bordeu[2] founded the vitalistic school and taught that each organ of the body elaborated a specific substance. So far as space permits the more essential

[1] Weil, A.: Die Ergebnisse der Lehre von der inneren Sekretion für die normale und pathologische Physiologie, Deutsch. med. Wchnschr., April 28, 1922.

[2] Neuburger, M.: Wien. kl. Wchnschr., 1911, No. 39 and 40 has written an excellent study of Theophile de Bordeu of Monpellier.

interrelationships and correlations will be brought out in the discussion of the various diseases. They are at present grouped under their respective glands. Varying points of view are developing rapidly.

The student of the endocrinopathies should constantly keep in mind the fact that any clinical picture seen at ony one time represents only a cross-section of a dynamic process which may be modifying daily or possibly have come to a temporary or permanent standstill. Thus a lymphatic giant picture at the age of sixteen or seventeen may be converted into a hyperpituitary syndrome a few years later from failure of thymus involution; an inferior adrenal system failing to compensate throws the balance to its chief adjunct, the pituitary. Thus mutual interactions and compensations are to be found in this field as in all other fields of medicine where complex dynamic activities are constantly in play and shifting from year to year in individual development. As in manifest bodily adjustments the expressed symptom may not be due directly to the disorder of the organ showing the most evident sign of disease. Thus a respiratory difficulty is not always due to a lung disease. It may arise from distant and elusive sources, kidney, heart, emotional, etc. Thus an endocrinopathy is not necessarily a disease of the gland itself. If this principle had been kept in mind much meddlesome surgery of the thyroid would have been avoided, as so frequently a hyperthyroid activity may be a functional activity trying to make good for a deficiency elsewhere. To cut out the organ apparently diseased may destroy the effort Nature is making to bring about some compensating mechanism in the body's adjustment to some difficulty, physico-chemical, sensorimotor, or symbolic.

The chief available literature summaries are Biedl,[1] Sajous,[2] Falta,[3] Lewandowsky,[4] Parhon and Goldstein, Levy and Rothschild, Weil, Pende,[5] Laignél-Lavastine[6] the encyclopedic work on Endocrinology and Metabolism (1922) edited by Barker, Hoskens and Mosenthal,[7] Max Hirsch's *Handbuch der inneren Sekretion*, Zondek, Gley, Schaefer, etc.; and special monographs to be mentioned under the separate syndromes, such as Cushing on the *Pituitary*, Klose and Vogt on the *Thymus*, Morel on the *Parathyroid*, Sattler on *Basedow's Disease*, Bell, *Sex Complex*, Harms, *Keimdrusen*, etc.

The glands in question may be grouped in general according to their phylogeny and embryology as follows:

1. From the buccal cavity:
 - (*a*) Thyroid (phylogenetically also gonadal).
 - (*b*) Hypophysis (anterior lobe of pituitary).

[1] Internal Secretions. [2] The Internal Secretions.

[3] Die Erkrankungen der Blutdrüsen, exhaustive and suggestive.

[4] Handbuch der Neurologie, vol. **4**, Special Neurology, a collection of valuable monographs.

[5] Endocrinologia, 1920, 2 Edit.

[6] Nervous and Mental Disease Monograph Series, 1919, No. 30.

[7] In the Barker, Hoskens, Mosenthal Encyclopedia Dr. F. H. Garrison has written a very valuable historical summary of the development of Endocrinology. A good historical retrospect is to be found also in the Max Hirsch, Handbuch, also see Hopf, Janus, 1898, 1899.

2. From the nervous tissues:
 (*a*) Infundibular process (posterior lobe).
 (*b*) Chromaffin tissue (suprarenal).
3. From the branchial arches:
 (*a*) Parathyroids.
 (*b*) Thymus.
4. From the intestine:
 (*a*) Parathyroids.
 (*b*) Mucosa of small intestine.
5. From the mesothelium of the genital ridge:
 (*a*) Gonads (sex glands).
 (*b*) Interrenal bodies.

Asher[1] has arranged the organs according to their physiological function as follows:

1. Internal secretions serving as foods.		Glucose.	Liver.
		Fat.	Intestinal mucosa.
		Fibrinogen.	Panniculus adiposus and liver.
2. Harmozones. Substances regulated by chemical processes.	1. Those playing a role in metabolism.	Substances regulating sugar production.	Pancreas.
	2. Those tending to fix inner milieu.	Antithrombin.	Liver.
	3. Morphogenetic substances modifying growth.		Intestinal gonadal, thyroid, hypophysis, thymus.
3. Hormones.	(*a*) Acting chemically.	Substances activating trypsin.	Spleen.
	(*b*) Acting physiologically.	Substances modifying nitrogen and gaseous metabolism.	Thyroid.
		Secretin.	Mucous membrane of duodenum and jejunum. Uterine glands. Placenta.
		Galactagogues.	
4. Parhormones.		Carbon dioxide, Uric acid derivative.	Fetus? Muscles, glands, liver.

It is highly important to remember that an endocrinous gland rarely represents in itself the entire material which may function, as such. Accessory glands, or distributed bits of specific glandular substances are widely distributed. Failure to bear this in mind vitiates many of the results obtained from physiological extirpation experiments. Notably is this true for many studies of the adrenals.

Among the earlier attempts at correlation of the group of disorders of the blood glands or internal secretory glands were those of Claude and Gougerot, and Laignél-Lavastine.

A comparatively recent systemization of this latter author is suggestive. In the first place, one can distinguish (*a*) neurological symp-

[1] Asher, Leon: Allgemeine Physiologie der inneren Sekretion. Max Hirsch: Handbuch der inneren Sekretion, 1927.

toms as a part of an endocrinopathy; (*b*) endocrinous disturbances in neurological syndromes, and (*c*) double forms of endocrino-neuro-endocrinopathies and neuro-endocrino-neuropathies. The endocrinopathies, as outlined by Laignél-Lavastine, may thus be rapidly reviewed. It is still to be proved how valid certain of these conclusions are. They are suggestive, however, and entitled to be tried out.

I. *Uniglandular Endocrinopathies:*

1. Thyroid.
 - (1) Myxedema—hypofunction.
 - (2) Exophthalmic goiter—hyperfunction.
 - (3) Thyroid insufficiences.
 - (4) Thyroid instabilities.
2. Parathyroids:
 - (1) Tetany.
 - (2) Paralysis agitans (?).
3. Thymus:
 - (1) Vagotonic symptoms of exophthalmic goiter.
 - (2) Myasthenia gravis.
 - (3) Thymoprivous idiocy.
4. Suprarenal:
 - (1) Addison's disease—hypofunction.
 - (2) Genito-adrenal syndrome.
5. Sympathetic paraganglia.
6. Pancreas:
 - (1) Diabetes mellitus.
7. Hypophysis:
 - (1) Adiposogenitalis of Fröhlich.
 - (2) Acromegaly (hyperfunction).
 - (3) Gigantism.
 - (4) Hypophysial insufficiency.
8. Pineal:
 - (1) Macrogenitosomia.
9. Gonads (ovary, testicle):
 - (1) Infantilism.
 - (2) Acquired ovarian insufficiency.
 - (3) Hypoövarianism.
 - (*a*) Infantilism.
 - (*b*) Acquired testicular insufficiency (castration)
 - (*c*) Eunuchism.
10. Prostate:
 - (1) Hypo- and hyperprostatic syndromes.

II. *Polyglandular Endocrinopathies:*

1. Thyroid predominances.
 - (1) Exophthalmic goiter with thymic hypertrophy.
 - (2) Myxedema with thymic hypertrophy.
 - (3) Acromegalies with ovarian insufficiency, at times resembling exophthalmic goiter, at times myxedema.

2. Ovarian predominance:
 (1) Thyroid reactions with ovarian insufficiency.
 (2) Dyshyperovarianism of hypothyroidism.
 (3) Thyroövarianism.
3. With hypophysial predominance:
 (1) Infantile giants. Feminism, eunuchism, cryptorchism, pseudohermaphroditism.
 (2) Acromegalics with defect symptoms. Infantilism, amenorrhea, obesity, asthenia.
 (3) Acromegalics with hyperactivity symptoms, exophthalmos, arterial hypertension, atheroma.
4. With suprarenal predominance:
 (1) Addisonians with amenorrhea, frilosity, tetany, or goiter.
 (2) Goiter, acromegalies, giants with diabetes.
5. Without marked predominance.

The French students have carried these suggestions to great lengths, and have developed an organotherapy of startling complexity, but one which should be carefully reviewed and checked up.

Here we purpose to discuss those disorders of the internal secretions with pronounced disturbance of the nervous system, beginning with those best known. The student is reminded that a text-book can only deal with the most pronounced types, and those concerning which there is a certain unanimity of opinion, hence for further study of the mass of material reference should be made to the literature here quoted.[1]

We shall therefore take up: (1) the thyreopathies, (2) the hypophysial disorders, and (3) the disorders of the parathyroid, thymus, adrenals, and sex glands. Finally, some suggestive relationships between disease of certain viscera, liver, kidneys, spleen, etc., and the nervous system.

Before passing to the detailed study of these forms a word may be said concerning the interrelationship of these various endocrinous glands. It is highly probable that these glands work in unison and that such regulatory synchronism is mostly brought about through the vegetative nervous system. Pure chemical regulation may take place, but it is becoming more and more evident that the reactions which bring more or less hormone to the blood are mediated by the vegetative nervous system and the electrolytes. Extremely minute quantities are capable of producing definite reactions. Zondek and others have shown, for instance, that thyroxin 1 to 10,000,000 modifies the metamorphosis rapidity of tadpoles.[2] These hormones modify the electrical carrying capacity of the fibers and the resistances at the synapse chiefly through the Ca and K, ions and thus modify function. The whole series of processes are too highly complex and as yet too little

[1] Rosenbloom: Functional Methods for Studying Diseased Conditions of Certain Endocrinopathies, Interstate Med. Jour., 1919, vols. **10, 11, 12, 25, 26.**

[2] Zondek: Deut. med. Wchnschr., 1924, No. 12, Zondek and Reiter: Ztschr. f. kl. Med., 1923, **99**, 139.

known to be discussed in a text-book. Heidenhain (1921) has studied these from the morphological aspect. The student is referred to special works.[1] To refer to but one mode of explanation the scheme of

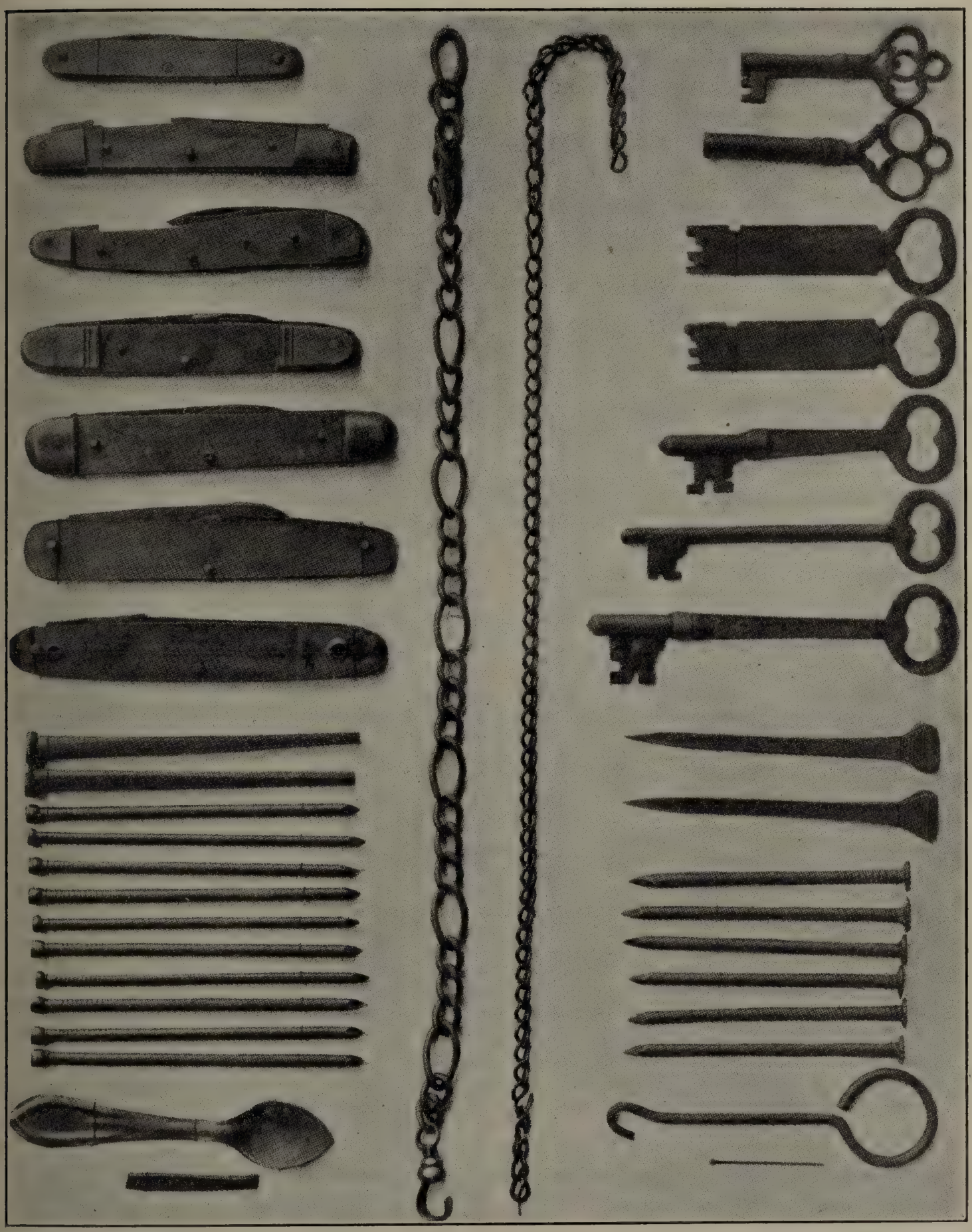


FIG. 92.—Objects removed from stomach af patient suffering from gastric tetany. (Warbasse.)

[1] Noël Paton: Nervous Regulators of Metabolism. Higier: Vegetative Nervous System, Nerv. and Ment. Dis., Monograph Series, 1919. Laignel-Lavastine: Internal Secretions and the Nervous System, Nervous and Mental Disease Monograph No. 30. Asher: Harvey Lecture, 1922, 1923, **18**. Zondek: Die Krankheiten d. endocrinen Drusen, 1923. Weil: Die innere Sekretion, 1923, third edition,

Zondek is of interest. (See Fig. 93.) These hormones in turn modify the electrical carrying capacity of the fibers and the resistances at the synapse and thus modify function. The whole series of processes are highly complex and the student is referred to special works with the express suggestion that science is dependent upon hypotheses for its advance but these should be constantly checked up by the objective findings, and put into the scrap heap so soon as better functional tools, *i. e.*, more valuable hypotheses, are advanced.

The interrelationships of the various endocrinous glands have been ably illustrated by Noël Paton, a reproduction of certain of his charts or diagrams being given here.

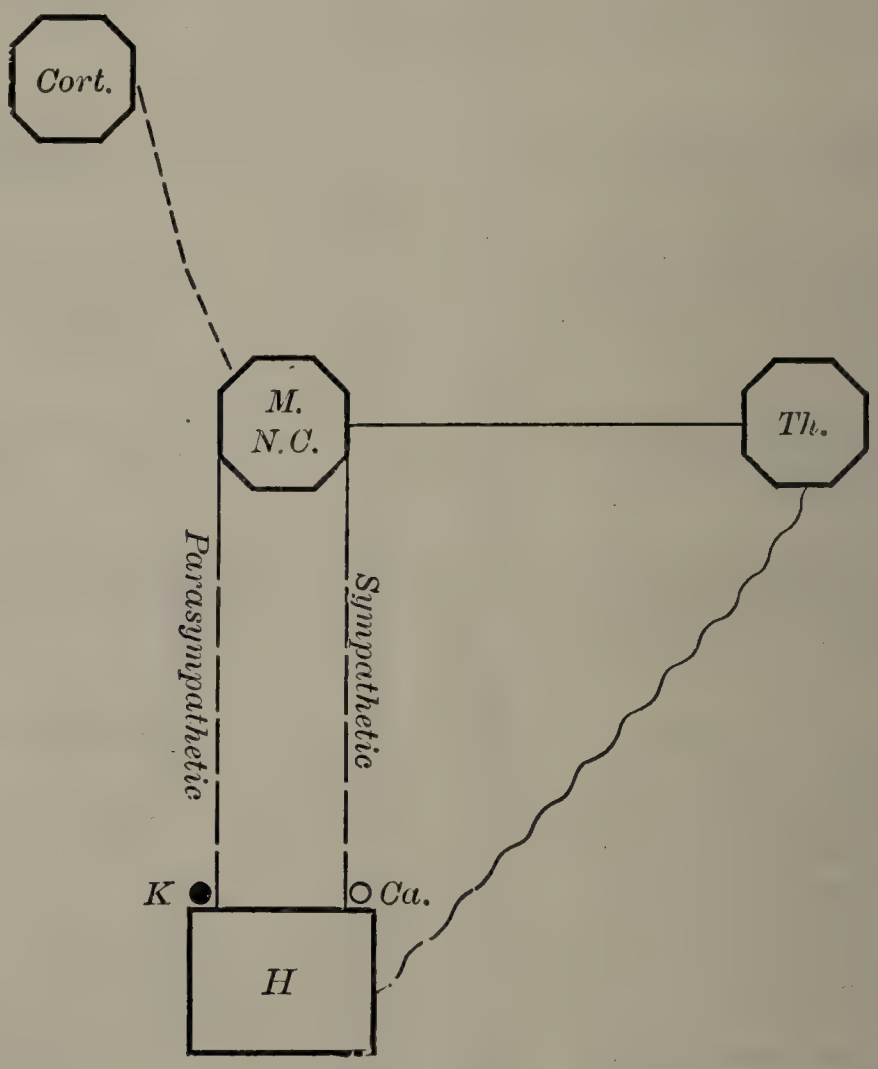


FIG. 93.—Zondek's scheme of the action of hormones. *Cort.*, Brain cortical association paths with mid-and interbrain vegetative nuclei, *M. NC.* These are in connection with the incretory organ, *Th*, here thyroid; and the stimulus passes through the sympathetic and parasympathetic fibers to the peripheral organ, here represented as *H*. Heart: through the electrolytes, Ca and K ions, the character of these impulses is regulated and hence the activity of the peripheral organs of the entire body. (Kraus, Zondek.)

As Paton well remarks, these may well be a grotesque parody of what will ultimately be found to be the relationship of the activities of these organs. "They are probably as near the truth as those quaint ancient maps of the Indies with their 'here be much gold' scrawled across them which served as the charts of our forefathers, but if, like them they merely indicate the direction which further investigation should take and suggest lines of attack, they will have served their purpose."

The direct and profound action of the secretions of the sexual glands (gonads) upon the body is seen in every tissue of the body. How far their action is facilitated and how far checked by other endocrinous organs is not yet entirely worked out. The thymus supplements the action of the testes secretion. Its relations to the ovaries is not so

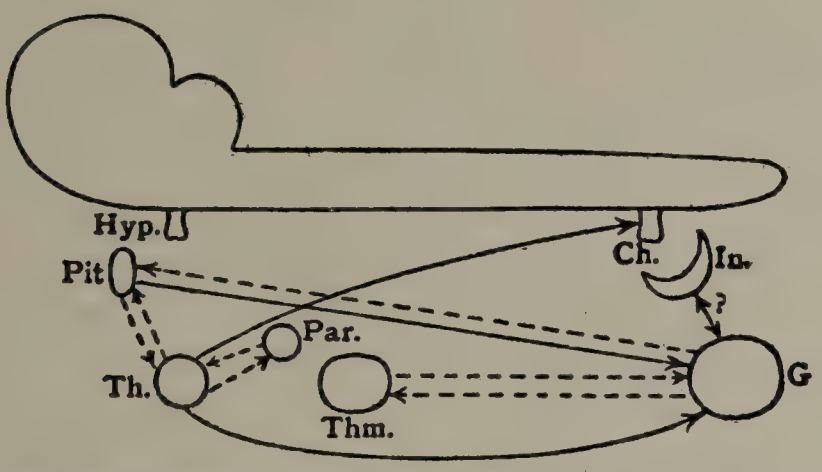


FIG. 94.—To show the possible influence of the various endocrinous structures on one another. The following explanations apply to this and to the three succeeding figures: ——— stimulation; ------- inhibition. The arrow indicates the direction of action. *Hyp.*, hypophysis; *Par.*, parathyroid; *Ch.*, Chromaffin system; *Art.*, artery; *Pit.*, pituitary; *Thm.*, Thymus; *Th.*, thyroid; *G.*, gonads; *B.*, bone; *In.*, interrenal; *Pan.*, pancreas; *M.*, muscle. (Paton.)

certain. It exercises a checking action on the male gonadal activity which in its turn acts reciprocally on the thymus. (See Figs. 94 to 97.)

The removal of the thyroid checks the growth of the gonads. Castration acts less on the thyroid, although menstruation, childbirth, and the menopause cause definite increase in thyroid activity. (See Thyroid.

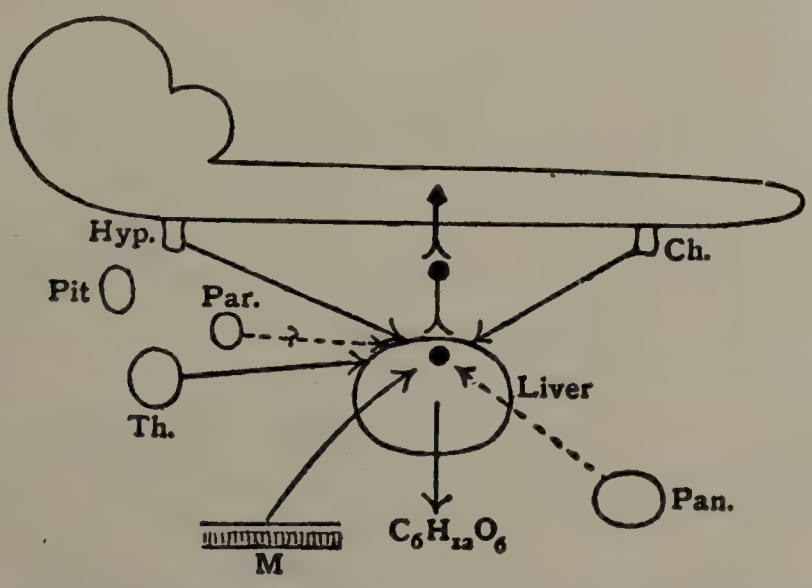


FIG. 95.—To show the possible mode of action of the various internal secretions on the mobilization of sugar in the liver. (Paton.)

The destruction of the pituitary leads to gonad atrophy and reciprocally castration causes hypertrophy of the pituitary. The secretion of both stimulate the growth of the long bones, the uncontrolled activity of the former leading to gigantism and acromegaly. The action of the gonadal secretions is to check the pituitary activity

and the increase in size of the eunuch is possibly a response to this unchecked hypophysial activity. The gonads are not alone in hindering the pituitary action.

Suprarenal and gonadal activity are closely related and suprarenal loss is usually accompanied by genital aplasias or anomalies. Paton has suggested the identity of certain elements of these tissues and that

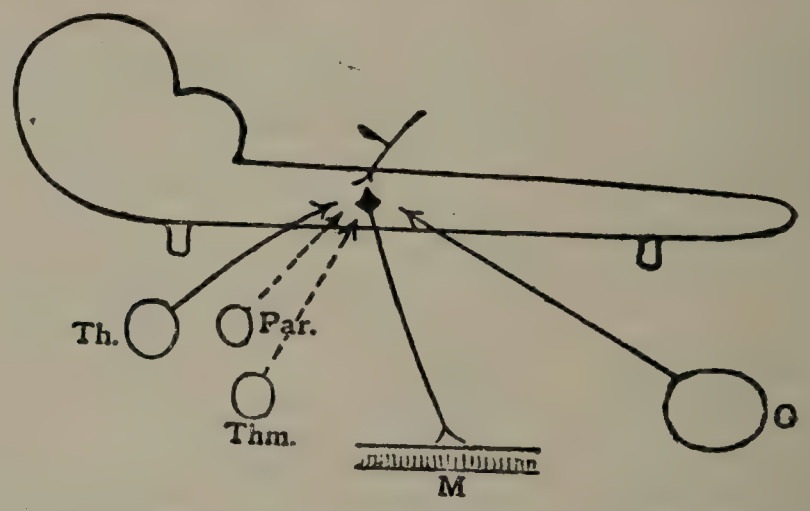


FIG. 96.—To show the possible mode of action of certain of the internal secretions upon the spinal reflex arc. (Paton.)

the suprarenals constitute a sort of bridge or intermediary between the bodily and the sexual cells.

The thyroids and pituitary are closely related. Removal of one causes hypertrophy of the other. (Fig. 94.) They thus mutually check each other in part and are also coöperative, the pituitary needing

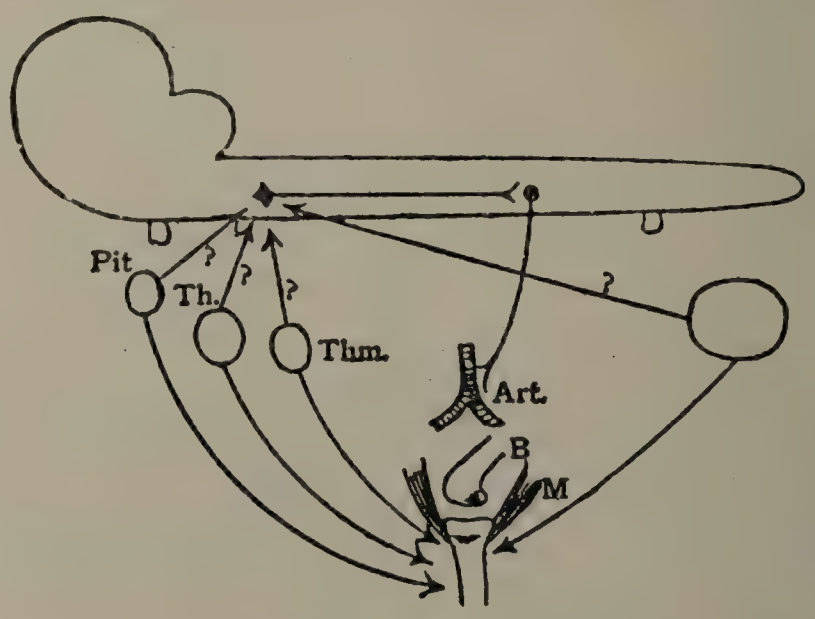


FIG. 97.—To show the possible mode of action of the internal secretions on the growth of muscle and of bone and of the connective tissues. The possibility of this being a vasomotor reflex mechanism is indicated by lines marked ? (Paton.)

the thyroid to complete its activities. Hyperthyroid activity does not lead to hyperplasias of connective or bony tissues as does hyperpituitary action; the reciprocal parasympathetic and sympathetic nerve activity is not exactly similar; although diminished activity of both substances may lead to diminished bony growth—atrophy. The vegetative

mechanism of this, however, has not yet been elucidated. See previous section on Bony Syndromes.

The action of thymus on thyroid is far from clear, but the tendency is to show a reciprocal checking action especially on the neuromuscular apparatus. The problem of myasthenia gravis has been thought to lie behind this reaction. Feeding thymus to salamander larvæ induces parathyroid atrophy and the development of tetany, possibly related to the diminished calcium regulation of the parathyroid substances. Dustin sees in the thymus a glandular reservoir for nuclein metabolism mechanisms. The pituitary undoubtedly plays a large part in phosphorus regulation.

The thyroids and parathyroids have distinctly different and even antagonistic activities: The former seems to be related more distinctly to the iodine, the latter to the calcium metabolism of the body. Just how they are regulated through the vegetative nervous system is unknown. Calcium is of pronounced value in neuromuscular activity as the phenomena of tetany show. Myasthenic states in general and myasthenia gravis in particular are more directly related to disordered thyroid and thymus activities. Lundborg suggested early that the parathyroid function plays some part in the reaction.

The thyroid acts on the pancreas chiefly through its action on the liver sympathetic fibers. Sugar mobilization and release are brought about through modified thyroid and pancreatic action, which latter prevents the mobilization of sugar in the liver. Thus glycosuria is frequent in hyperthyroid states. (See Fig. 95.)

The complicated interrelationships cannot be entered into more fully here. The chief available literature has been indicated. One point, however, should be emphasized and that is that the activities of the internal secretion organs are all under vegetative nervous system control. The active substances, hormones, if one wishes, are not entirely independent chemical activators, they are under sympathetic and parasympathetic (autonomic) control. The output of iodine, of calcium, of adrenalin, of hypophysin and of all of the substances thus far known or named is controlled almost exclusively by the nervous system. The hormones act principally through the nervous system. While it may be shown that within an organ itself purely humoral regulation may be effective—thus one must explain the positive and negative tropisms within the cells of an organ in its initial response to a disturbance of cellular adjustment—yet the chief activities of the internal secretions are brought about by neuro-chemical regulators, as Paton terms them.[1]

Just as the complicated sensorimotor integrations are effective in governing the muscular activities of the human body, so the integration of neuro-chemical regulators, taking place at the physico-chemical level, is effective in adjusting the metabolism of the body cells. Hormones as such, do not stimulate or inhibit but act through the vegeta-

[1] For complete discussion of the chemistry of the hormones, see Guggenheim in Hirsch's Handbuch, loc. cit.

tive nervous system according to the electrolytic milieu. All of the endocrinopathies are really polyglandular syndromes. Emotional reactions constantly influence their activity and when prolonged can modify the structure of the glands. This as a fact has been known for centuries but is now being demonstrated experimentally in the physiological laboratories. (Pavlof, Cannon, etc.)

THE THYREOPATHIES; THYREOSES.[1]

The Thyroid.

In the lowest vertebrates the thyroid was intimately connected with the genital ducts, they were uterine glands, but from Petromyzon upward it has lost that connection, although it is known that an intimate (polyglandular) association still persists (menstruation, pregnancy and swelling of gland, genital excitement and hyperfunctioning and other relationships to be discussed). In the human embryo it seems to be cut off from the foregut. It is intensely vascular and its chemistry is unique. It contains a comparatively high percentage of iodine, also phosphorus, arsenic, bromine and sulphur. The chief hormone Kendall names thyroxin.[2] In crude state it is associated with colloid material the presence of which is a general though not a certain index of its activity.

Kottmann,[3] in 1920, described a reaction of serum to colloidal iodide of silver which offers quantitative data concerning the activity of the thyroid.

The nerve supply, thyroid nerves, arises from the cervical sympathetic, the fibers, mostly non-medullated, passing from the middle (inferior) cervical ganglion from the thyroid plexuses whose fibers penetrate the gland. Its chief visible supply is parasympathetic (vagus) but sympathetic fibers also are present. The fibers go to the bloodvessels and also to the glandular cells. The character of the receptor and effector cells of the glandular cells is not yet known but there is some evidence that the visceral reflex passes by means of the sympathetic fibers and not by the cranial parasympathetic ones.[4] Section of the sympathetic causes marked atrophy of the gland, vagus section none.

[1] Sattler: Basedow's Krankheit, one of the most complete monographs on the subject. Collected papers. Mayo Clinic, 1915. McCarrison: The Thyroid Gland, 1917. Crotti: The Thyroid and Thymus Glands, 1922. Richardson: Thyroid and Parathyroid. A. Josefson of Stockholm has contributed many important papers and a small valuable monograph. Rossi: Disfunzione Tiroidea, see Report of Thyroid Dysfunction from Milan Serotherapeutic Institute, 1924. The periodical literature will be best consulted in Endocrinology, a quarterly devoted to this branch and in the French, Spanish and South American special endocrine journals, and in the Current Literature department of the Journal of Nervous and Mental Disease and Ztschr. f. d. ges. Neurol. u. Psychiat. Also consult monograph edited by Barker, Hoskens and Mosenthal on Endocrinology and Metabolism, 5 vol., D. Appleton & Co., 1922, and Hirsch's Handbuch d. inneren Sekretion, 1927, and French monographs.

[2] See Guggenheim: Hirsch Handbuch, for Chemical Structure.

[3] Schw. med. Wchnschr., 1920, **1**, 644, also see Petersen, Jour. Am. Med. Assn., 1922, **78**, 1022. Raphael and Smith: Am. Jour. Psychiatry, 1924, **4**, 161.

[4] Cannon and Cattell: Am. Jour. Physiol., 1916.

The chief endocrinopathies produced by thyroid disease are those due to lessened function, hypothyreoses, and those due to an excess of function, hyperthyreoses. While the iodine is the most striking ion in the secretion it is probably not the only one in producing the striking metabolic disturbances of thyroid disorder.

Certain hyperfunction cases, even with toxemias, are not due essentially to thyroid disease. They are not primary hyperthyreoses. They are frequently overcompensatory reactions chiefly to inferior adrenal situations. These latter are frequently conditioned by exhaustion (physical or fear states) and thus caused the great amount of hyperthyroid states seen in the soldiers in the World War of 1914–1918. Many of these and related types are often quickly relieved by pituitary extract 1 to 3 gr. daily, under careful scrutiny. The pituitary may compensate for the adrenal and thyroid. A large thyroid with defective adrenals and tightly bound in pituitary, Timme has claimed is the type which reacts better to pituitary extract than to surgical procedures. The *x*-ray of the sella turcica shows the small restricted pituitary which cannot compensatorily hypertrophy for the defective adrenals, this throws the work over to the thyroid, which enlarges. Surgical removal of the thyroid here is worse than useless unless the overcompensation is threatening life.

Hypothyreoses; Myxedema.—The chief pronounced *hypothyreoses* are grouped under the symbols *myxedema* and *cretinism*. There is an important group of cases in which less marked insufficiencies are found and which need close study. Three main types of the former, congenital, idiopathic, and operative, are described, while sporadic, endemic, and irregular types of cretinism are distinguished. The whole group may be considered as one, *i. e.*, hypo- and athyreoses. The various subgroups have grown up clinically since Gull, in 1873, first called attention to myxedema. They are still in great need of clearer differentiation and description, especially the milder and the variable types.

Operative myxedema (cachexia thyreopriva) has been the best studied type since Kocher, in 1883, called attention to it, one year after Haddon had shown the relationship between myxedema and the thyroid.

Symptoms.—The chief symptoms in the pronounced types are present in the skin, nervous system, thyroid, circulatory apparatus, temperature, digestive tract, blood, urine, bones, and general metabolism.[1]

Skin.—Here there occurs a general gradually increasing swelling, most marked in the hands and head. It seems edematous and yet does not pit on pressure. The thickness of the lips causes an eversion or hanging down of the same. The folds of the skin, especially of the forehead, are more distinct than is usual. Irregular, flattish, fat-like deposits are present in different parts of the body, often being most marked in the supraclavicular region.

[1] Bolton, G. C.: Ueber Hypothyroidea (Sympathicus hypotonia), Deut. Zeit. f. Nerv., 1917, **97**, 119.

The hands are apt to be clumsy and thick, the skin of the back of the hand being much thickened. The feet may show similar changes. The whole skin is whitish and dry; it scales readily and rarely shows



August 3, 1914. August 17, 1914. November 27, 1914.
FIG. 98.—Infantile myxedema. Ten years old. Treated by large doses of thyroid tablets. (A. Josefson.)

any perspiration. Diaphoretic drugs even are unable to bring about any marked perspiration, and the mucous membranes are apt to be dry and not easily irritated to cause exudates. Yellowish pigmentation may occur. The hair breaks easily and is apt to be badly and

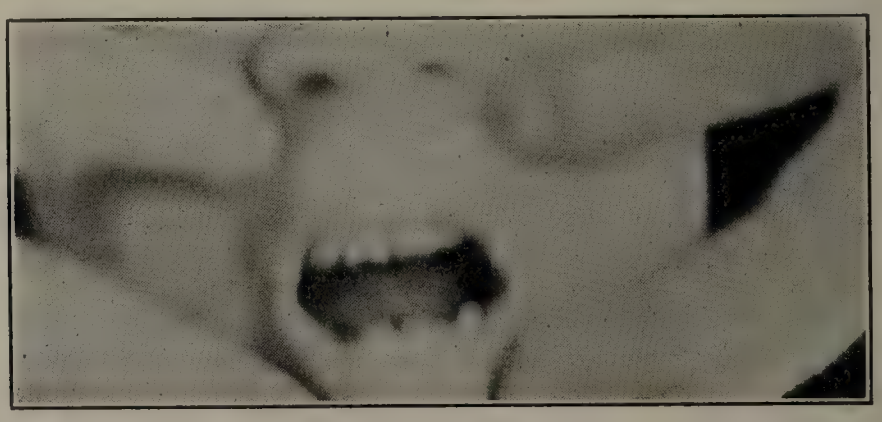


FIG. 99.—Hutchinson teeth, nonsyphilitic, hypothyroid stigma. (A. Josefson.[1])

sparsely developed. The nails are brittle, develop slowly, and show irregular markings. The teeth also develop badly. (See Fig. 99.)

[1] Dermat. Wochenschr., 1914.

There is a sense of tension in the skin, and coldness of the extremities seems universal. This is made much worse in winter, with marked tendency to chapping and frost-bites.

The *nervous system* shows a number of defects varying with the grade of hypothyreosis. Any of the cranial nerves may show defective development. The cerebrospinal nerves may be deficient. The motility as well as the sensibility is diminished both as a result of peripheral and central maldevelopment. The changes in sensibility are furthermore augmented by the localized skin changes. The reflexes are not markedly changed. The gait is usually wide-based and clumsy and due in part to the mental dulness. Defect in thyroid substance seems to hinder the regeneration of cut or injured peripheral nerves.

Mentally a great variety of changes may be observed. They are usually in the nature of defect. There is defect of memory, attention is diminished, thinking goes on more slowly, but may be of fair capacity. There is usually a loss of initiative, and emotional dulness goes along with the sensory losses and motor reluctance. The speech is apt to be slow, as are other motor acts. It is monotonous and the thickened lips further contribute to make it at times unintelligible. The whole appearance of the patient is one of gradually advancing stupidity which, if there is no relief, goes on to more profound defect states—dementia.

The *thyroid* itself is usually much diminished in size, or not at all palpable. Though palpable, its active secretory substance is usually defective.

The *circulatory* apparatus not rarely shows anomalies. Zondek (1918 described a definite heart syndrome.[1] Electrocardiogram changes, dilated heart, and congestive failure are present.[2]

The *cold* sensations are not subjective alone, as there seems to be a fairly constant diminution in the bodily heat, as is also seen in hypopituitarism. Digestive discomforts from dry mouth and enlarged tongue are frequent. There is not infrequently diminished muscular tone and deficient secretions in the entire digestive apparatus with obstinate constipation.

Genital anomalies are frequent, consisting of irregular or suppressed menstruation or diminished potency. The organs themselves—testes, ovaries—may be diminished in size and infantile; there is defective genital hair development.

The *blood* shows fairly constant eosinophilia, the clotting time is increased, and the fibrin content above the average.

The *urine* is not characteristically altered, save that its quantity is usually decreased.

Metabolism is slowed down in many directions. Oxygen exchange is reduced, the calories consumed being markedly diminished. The nitrogen output is less, as well as that of the purin derivatives. The calcium-magnesium metabolism is not modified save in those operated

[1] Fahr: Myxedema Heart, Jour. Am. Med. Assn., 1925, **84**, 345.
[2] More recent studies by Read, J. M., Jour. Am. Med. Assn., 1927, **89**, 493.



FIG. 100.—March 5, 1913.



FIG. 101.—January 5, 1915.



Fig. 102.—March 5, 1913.



Fig. 103.—January 5, 1915.

Figs. 100, 101, 102 and 103.—Hypothyroidism. Effects of thyroid in large doses on skeleton. (A. Josefson.)

upon and in whom the parathyroids are also disturbed. Carbohydrate tolerance is high.

The *bony* system is variously altered. Here the amount of change and its diversity depends largely upon the age of the patient at the onset of the disorder. The long bones fail to grow normally and those with late ossification centers fail to undergo complete development. The skull is apt to be macrocephalic, but the thickness of the bone may diminish the interior capacity a great deal. In hypothyroid cases fractures are apt to heal slowly. The fontanelle in young patients is apt to remain open. The changes in myxedema are not those seen in rachitis or chondrostrophia.

Types.—The *idiopathic* form usually begins with changes in the skin, and is often accompanied by neuralgic pains. A patient recently seen was diagnosed as a tabetic. The skin of the face is often first affected and usually the extremities are implicated very gradually. The symptoms may all come on within a few weeks, but usually their development occupies months. Women are much more frequently affected, and usually about the menopause period. In these cases the hypothyreosis may be diagnosed as a "menopause neurosis." A not unusual result of the cessation of the menstrual function is an overactive thyroid, but in other cases the reciprocal stimulation which is pronounced between ovary and thyroid suffers with the diminution of the ovarian function.

Operative myxedema is now comparatively rare, since the essential relationships have been pointed out. The tetany symptoms often seen in the earlier and badly operated cases were due to the parathyroid removal.

Congenital forms, thyreoaplasias, occur in children usually of normal birth and average development up to about the time of weaning—if not breast-fed usually earlier (thyroid in mother's milk). The symptoms then develop rapidly, and, as a rule, are very extreme. The irregular, imperfectly developed, cretinoid pictures are not the usual ones in congenital myxedema; as Eppinger has remarked, there are few "halfway" congenital thyreoaplasias. The female sex preponderates and there are no geographical limitations as in endemic cretinism. Neither is there, as a rule, any goitrous family history as is often found in cretinism.

These little patients forget to suckle and to swallow. The skin becomes folded, the nose broad, the eyes deeply sunken, the nasal wings widely spread apart. They are mouth-breathers, with swollen, not infrequently protruding, cyanotic tongues. Salivary increase is often present. The hair is badly developed, the face that of an old person. The head grows in size, but the rest of the body stays behind with marked disproportion in length and breadth throughout. The epiphyses do not ossify, and the centers of ossification, especially in the hands, fail to develop. The fontanelles remain open perhaps until the twelfth or fifteenth year, and the teeth are tardy in appearance. The body is apt to be fat and the abdomen especially swollen, in part from

gas and obstinate fecal accumulations. Umbilical hernia is frequent. Other defects are often present in heart, palate, and other

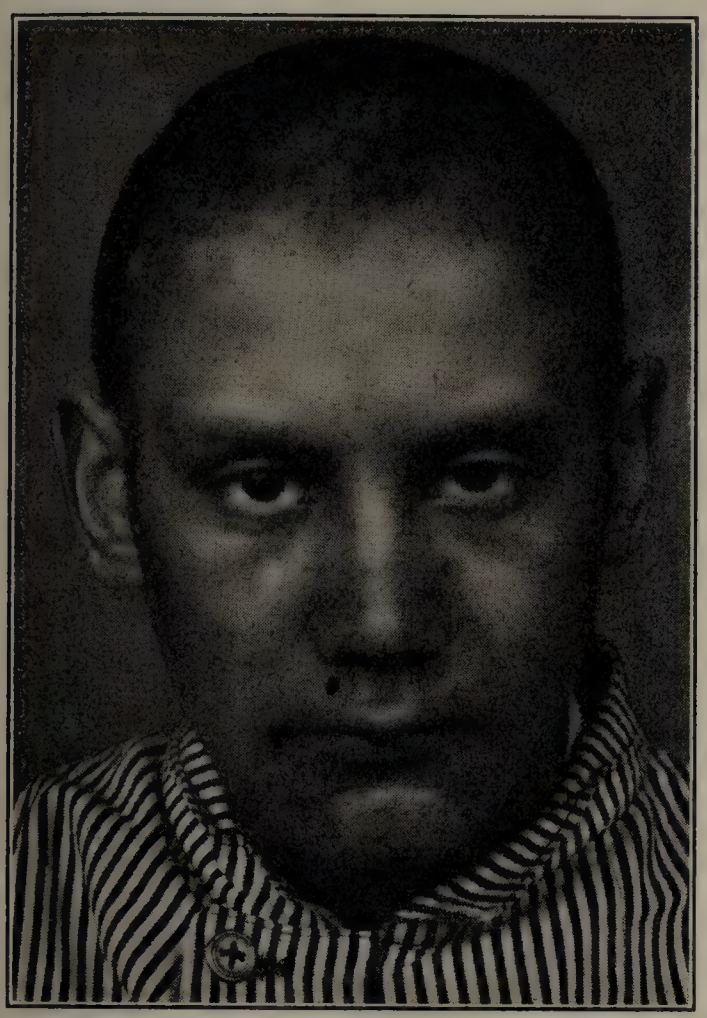


FIG. 104



FIG. 105

FIGS. 104 and 105.—Myxedema treated with thyroid in great doses. (Josefson.)

structures, and they die early with the general mental symptoms in part described.

Diagnosis. — Nephritic edema and other skin edemas must at first be ruled out, especially ovarian pseudoedema of the menopause and rare forms of syphilitic or familial neurotrophic edema. Chondrostrophia must be separated from the cretinoid complications. The relations to cretinism are close. There are differences in the skin and perspiration. Deaf-mutism is rare in myxedema, frequent in cretinism.

Cretinism.[1]—This is a broad, general term applied to a combination of somatic and psychic changes which, in the young, result



FIG. 106.—Myxedema showing failure of ossification in epiphyses of the bones of the hand. (Siegert.)

[1] Finkbeiner, E.: Die krentinische Entartung, Springer, 1923.

from loss or diminution of the thyroid functions. Such a loss may occur sporadically, *sporadic cretinism*, from causes to be enumerated, where the picture is analogous to that seen in the adult from removal of the thyroid, cachexia thyreopriva adultorum, or it may occur as a localized or *endemic* degeneration, affecting the thyroids of a large number of individuals, causing a hypothyreosis which may show a number of tendencies. When these are pronounced they are spoken of as *goiter*, *goitrous heart*, and *endemic cretinism*.



FIG. 107.—Cretinism. Woman, aged thirty-four years; mentally, seven years by Binet-Simon test; height, 49½ inches; protuberant abdomen, typical facies, supraclavicular pads of fat.

These three fairly well separated conditions may be discussed to advantage under the head of cretinism. In the first place to call cretinism a type of idiocy is misleading. There are numerous very intelligent cretins. Cretinism, as here described, is a complex of different conditions due to a lack of development of one or more elements in the body and chiefly conditioned by defect or loss of the thyroid functions.

The historical chapters on cretinism are full of interest. The disorders were known in early days. Pliny has left indubitable evidence of their presence in early Roman times. Vogt, in his admirable monograph in the Lewandowsky *Handbuch der Neurologie*, tells of Marco Polo's descriptions of certain types he had seen in his Asiatic travels.[1] During the past two centuries the disorders here included have been observed throughout the world. In certain lands the disease is very widely distributed, certain mountainous districts of Switzerland, Northern Italy, etc.—endemic cretinism—while in other regions it occurs rarely—sporadic cases. In the United States endemic cretinoid states are being found in greater and greater numbers. The Great Lakes' region has shown a high incidence of goiter. It has been observed in California, among our native Indians of the Southwest, in Vermont, and such patients have been seen in New York State (Adirondacks). In certain regions it has been a veritable

[1] Garrison records that Fodéré, 1792, first published a treatise on Goiter and Cretinism.

plague. Thus in Switzerland between the years 1875–1884, 7 per cent. of the recruits in the army showed some form of cretinoid degeneration. In ten years 2500 men were lost to the Swiss army from this cause alone. Certain valleys, especially those of Berne and Wallis, are overthickly populated with individuals showing cretinoid degeneration. In the school years 1899–1904, of 336,000 children fit for school, 15,000 had one or another type of cretinism. Similar conditions existed in Styria, Austria, and in certain Italian provinces. F. Bircher has contributed an important study to the distribution of cretinoid degeneration.

So far as the etiology is concerned, it seems certain that the conditions are fundamentally due to a defect of the thyroid substance—the thyroid hormones. This is secondary to various types of inflammation or of aplasias of the thyroid. There are certain limitations which must be discussed in their respective paragraphs.



FIG. 108.—Case of sporadic cretinism, aged twenty-one years. Before treatment.



FIG. 109.—Case of sporadic cretinism. After four months' treatment. (H. Sanderson.[1])

Sporadic Cretinism. — *Infantile Myxedema of some Authors.*—The clinical picture in an extreme case—*i. e.*, fully developed—in contrast to the *many irregular or incomplete forms*—is that of a normally born child who about the end of the first or the beginning of the second year begins to show the characteristic changes in development. The little

[1] Sanderson: Sporadic Cretinism, with Report of Three Cases in One Family, Michigan State Medical Journal, April, 1906.

patient falls behind in his normal bony development. This is due to a defect in the development of the long bones. The epiphyses fail to lay down bone even after twenty to thirty-five years, and in twenty-year-old cretins the anterior fontanelle may still remain open. There is a proportionate loss in bony substance throughout; thus a characteristic dwarfism results save perhaps in the development of the skull, which grows larger in proportion to the rest of the body giving rise to the "full-moon" face. Other cranial bone defects, sphenoid, nasal, etc., give rise to the widely separated eyes, the pug-nose. The eyelids are thickened, the ears have a waxy appearance. The bony defect is in the nature of a sclerosis and the bony tissue is unusually hard, which is the reverse of that seen in rachitis.



FIG. 110.—Case of sporadic cretinism, aged four years. Beginning of thyroid treatment.



FIG. 111.—Case of sporadic cretinism, five years old. Thyroid treatment for one year.

Dental deficiencies (diminished calcification) go hand in hand with the bony defect. In severe athyroid cretins the teeth do no develop for a number of years, and the first or milk teeth may persist far beyond the normal period. Other defects appear in a high palatine arch, which with large adenoids and tonsils and a chronic hypertrophic rhinitis cause the child to snore and sniffle, often with copious excretions from the nose.

There is usually a short, thick neck. A fairly constant finding is that of umbilical hernia. The abdomen is usually puffy, the navel sunken (frog-belly). The skin is myxedematous in the young, but becomes

atrophic in later years, the supraclavicular and facial swellings remaining for many years.

The facial habitus is characteristic. The hair line begins low. The nose is sunken, the zygomatic arches prominent, the eyelids swollen, the face puffy, the tongue enlarged and often protruding between the swollen lips, in the mild cases giving one the impression of a child whose whole countenance is puffed up with crying.

There is usually an enlargement of the liver. Respiration is unusually slow in the severe athyreoses. The genital organs show marked changes. The labia are small, the external not covering the internal ones. The uterus and ovaries are usually small, and the mammary glands are atrophic or hypoplastic. The penis is apt to be small, the testicles undescended and small. Genital and axillary hair is absent or scanty. In boys the pubertal changes in the voice are lacking.

Blood changes are present. The hemoglobin is reduced and is out of proportion to the erythrocytes. The leukocytes are increased, the polymorphic neutrophiles being markedly diminished and the lymphocytes correspondingly increased. Large numbers of granular cells are observed. These changes appear as a result of defective thyroid substance.

The metabolism of calcium is markedly diminished (one-third of its normal amount in the studies made by Haugardy and Langstein and the required calories are far below that of the normal child's need. The assimilation of carbohydrates seems high, and adrenalin injections, without increased sugar intake, do not result in a glycosuria. There is a definite hypothermia. In many cretins there is a widening of the sella turcica. Hypoplasia of the thymus is also not infrequent.

Nervous system defects are present with the others and apparently conditioned by the endocrinous gland insufficiencies. These show at sensorimotor levels in defects of sensory and motor-nerve structures, and at psychical levels in various grades of stupidity, mental weakness (moron), imbecility or even idiocy. These words are here used in accordance with the arbitrary scale of the Binet-Simon tests.

Thus smell is at times defective; the eyesight poor; hearing is frequently disturbed, and with it speech, so that many patients are deaf and dumb. The vestibular function is frequently involved, so that these patients balance badly, often showing unsteady gait, with wobbling of the head, and nystagmus.

Some sporadic cretins may show little involvement of nervous structures.

Cretinoid Degeneration.—Mention has been made of the widespread character of this type of degeneration related to defective or absent thyroid secretions. The statistical study of the conditions, particularly in Switzerland, in France and in Italy (Bircher) has shown that goiter, goitrous heart, endemic cretinism, endemic deaf-mutism, and endemic feeble-mindedness are closely allied. The cretins are almost all goitrous, or nearly always have goitrous parents. Exophthalmic

goiter (hyperfunction) is rare with cretinism, but very frequent with goitrous heart conditions.

The causes of this particular type of hypothyroidism are not definitely settled but there seems to be a constant relation between it and certain elements in the water supply, and goitrous springs are known. Just what the noxious element may be is still conjectural, but it apparently is related to mineral constituents found in certain geological formations, notable in the trias and tertiary. The disease is absent in regions fed from waters flowing through crystalline formations. As a result of Bircher's suggestion of supplying a goitrous region in Rupperwill from Jura water coming from an adjacent valley, the disease disappeared. Similar results followed in the town of Asp. Animals may be made goitrous from drinking water from certain springs. They also develop goitrous hearts, and are delayed in their development. The thyroid shows degenerative changes. The agent passes through a Berkefeld filter, but is modified and made non-active by being heated to 70° C. It does not dialyze and is thought not to be an organized plant or animal substance, but to be of colloid nature. Bircher held that the disease was of an infectious nature. Our own inclination is to ascribe it to the iodine ions held in peculiar colloidal states.

Goiter.—Here Falta describes those enlarged hyperplastic, non-inflammatory thyroid formations, with degenerative changes in the struma. The hyperplasia invades the parenchyma and the vessels. Notwithstanding histological hyperplasia there is physiological diminution in function. Functionating parenchyma, however, does not exist. Histologically one finds parenchymatous, vascular, or fibrous hyperplasias, with circumscribed or diffuse goiters. A relationship between goiter and uterine myomata exists, and with a diminution of one there is a decrease of the other at the menopause.

The etiology of some goiters is ascribed by McCarrison to chronic fecal infection from contaminated water supplies.

Goiter Heart.—See Exophthalmic Goiter.

Endemic Cretinism.—Here there is a richer and much more variable picture than obtains for sporadic cretinism. Whereas a typical habitus is described, there are many anomalies and variations. The head is usually broad, but may be small and flat instead of large and broad, at times very large. The nose is usually wide-spreading and flat, the eyes wide apart. The neck is short and thick, the features swollen, the early impression, especially due to the prognathism, one of moroseness or stolidity. The bones are shortened, various anomalies as scoliosis, anchyloses, etc., being present. Great variation in dwarfism is observed. Certain cretins are under three feet six inches, but full cretins have been observed seven feet in height. As a rule they die young, but Kocher reports cretins seventy and even one hundred years of age.

The general coördination of these patients is poor. They are usually short, clumsy, inelastic with badly developed musculature. The skin is loose, lax, anemic, marked with folds and wrinkles, giving

a peculiar appearance of old age. The lips are swollen, the tongue enlarged, and not infrequently protruding. The breasts are flat or badly developed, the abdomen flat or pendulous. Short, stumpy fingers and toes give an ugly appearance to the extremities and contribute to clumsiness. The entire activity is apt to be heavy and awkward, although a few athletes and acrobats may be found among them.

The changes in the bones have been mentioned in the paragraphs on sporadic cretinism. Here, however, the variations are more marked and Weygandt's study of Virchow's material shows that many bony anomalies exist among cretins not mentioned in Virchow's classic which has remained a standard for writers for many years.

The skin has a peculiar cachexia. It is swollen and flabby, whitish or yellowish, folded and soggy. The general appearance of old age is striking. The hair and nails are badly developed, both breaking easily. Thick, underlying, fatty masses are unevenly distributed, usually in the neck, back, upper chest regions, occasionally over the hands. Variable states of tension occur in these fatty masses; at times they are hard, again like empty sacks. The mucous membranes are also pale and gray, often folded but look different from a typical anemia.



FIG. 112.—Fetal adenoma of thyroid with enlargement of the pituitary body. (Josefson.)

The sexual organ changes have been touched upon in the description of sporadic cretinism. They are characteristically infantile. Menstruation is scanty, wanting, or develops very late. Fecundation may take place, but the results are miscarriages, dead children monsters, etc. The secondary sexual characters are all delayed in their development.

The majority (63 per cent., Ewald) of cretins show a swollen thyroid, but it is not an overfunctioning one, nor do they all show athyreosis, or hypothyreosis. Schonemann has reported the findings of strumous changes in the glandular portion of the hypophysis. In 112 autopsies on endemic cretins he found a normal hypophysis in only 27 instances. These individuals had no goiter. He states that in individuals with struma of the thyroid almost invariably there was an increase in the connective tissue, also the chromaffin cells, struma of the vessels, hyaline degeneration and swelling of the cell strands and finally goiters with colloid formation. It is highly probable therefore that the goiter

poison works deleteriously upon the hypophysis (Falta). The parathyroids show no changes.

Most of the internal organs show reduction in activity. Digestion is usually slow, constipation is marked. The metabolism is modified as already indicated. The urinary secretions are apt to be diminished, and of high specific gravity.

Mentally cretins show marked variability. A few are practically normal, but most show a characteristic combination of mental traits, which is in marked contrast with many other defective mental states. (See chapters on Feeble-mindedness.)



FIG. 113.—Two cases of hypothyroidism. Elsa, aged fourteen years; Lisa, aged sixteen years. (A. Josefson, Deut. Arch. f. klin. Med., 1914.)

As noted, the great majority suffer from impairment of the chief sensory tracts. Hearing seems to suffer most. The defect in hearing is associated with speech defects. Taste and smell are also defective. They take little interest in their food or drink. The feeble-mindedness is accompanied by great slowness of all reactions, with marked retardation of motion, with apathy, and indolence. This indolence is a marked feature. Many cretins will lie in the sun all day long, and in the hospital or other institution will sit around and do nothing for weeks or months. In the milder grades there is often great shyness which makes them unapproachable and serves to make them appear more feeble-minded than they really are. It is with the greatest

difficulty that they can be trained to the simplest of performances. With many, in spite of the marked general stolidity of their average mood, they may show great excitement and emotional outbreaks.

The sense of sight is frequently diminished. It is highly probable that the receptors and conduction paths are less involved than the perception areas in this diminution in sensory intake. The hearing seems to be affected both as to its receptors and the conduction paths. Pain, touch and thermal sensibilities are all dulled. Motility is extremely retarded. The reflexes are active (60 per cent.). The field of vision is reduced in many, although the fundus is usually normal (Hitschmann).

Aberrant and abortive types are to be expected. In the former one may find patients with striking development of one or more features, in the latter a very great shading off to almost normal states, *i. e.*, endemic goiter with mild mental signs.

Endemic Deaf-mutism.—This combination is extremely frequent where endemic cretinism is present (29 per cent., Scholz). It may constitute one of the aberrant types just mentioned with striking development of single features, or it may be associated with all the grades of a complete cretin picture. According to the studies of Kocher, the loss of hearing is due to a bony defect which has destroyed the possibility of normal cochlear development. Bad hearing is reported at 32 per cent. among cretins in Scholz's investigations.

The changes found in the brain which may account for the feeble-mindedness have been variable. Meningeal inflammation and mild grades of hydrocephalus have been found by Scholz and Zingerle. The brain is often asymmetrical, small, or single lobes are diminished in size. Often the brain's development is arrested at an infantile stage, the pallium or the ganglia being involved alone or together. The cerebellum is often imperfectly developed, which fact stands in correlation with the marked incoördination and possibly in relation with defective labyrinthine development.

The ear difficulties are numerous. Peripheral, conducting and central mechanisms are found to be at fault, but at all events seem secondary to the developmental anomalies induced by the action of the poisonous substance on the thyroid. The speech defects usually go hand in hand with those of hearing, but this is not universal. A cortical developmental defect is explanatory for many of the cases.

Mild and Mixed Hypothyroid States.—Under the general title of abortive or mild hypothyroidism may be grouped a very large number of individuals, rarely considered sick, but who nevertheless are not up to concert pitch. They show one or more symptoms which are due in part solely to a mild thyroid deficiency, or to a polyglandular syndrome with hypothyroid predominance. These show themselves at various ages. Thus in the nursing child the absence of appetite, constipation, obesity, and somnolence have been referred to. In the older, the premature loss of the hair, irregularities of dentition, wide spacing, non-emergence, etc., precocious graying, somnolence, constipation.

Anorexia as a syndrome is often conditioned by mild thyroid defect. Certain studies of families have shown in the different members graded series of hypothyroidisms from myxedema to the mildest involvement, and again in others the gamut from the most severe types of myxedema on the one hand to the most severe types of exophthalmic goiter on the other may be observed.

The *hypothyroid type* is usually smaller than he should be, with a tendency to obesity. There is a trend toward facial puffiness, the eyelids, particularly in the morning, being swollen. The complexion has a tendency to be sallow and varicosities are frequent. The hair is apt to be dry, and the hair line high. The hair over the brows is

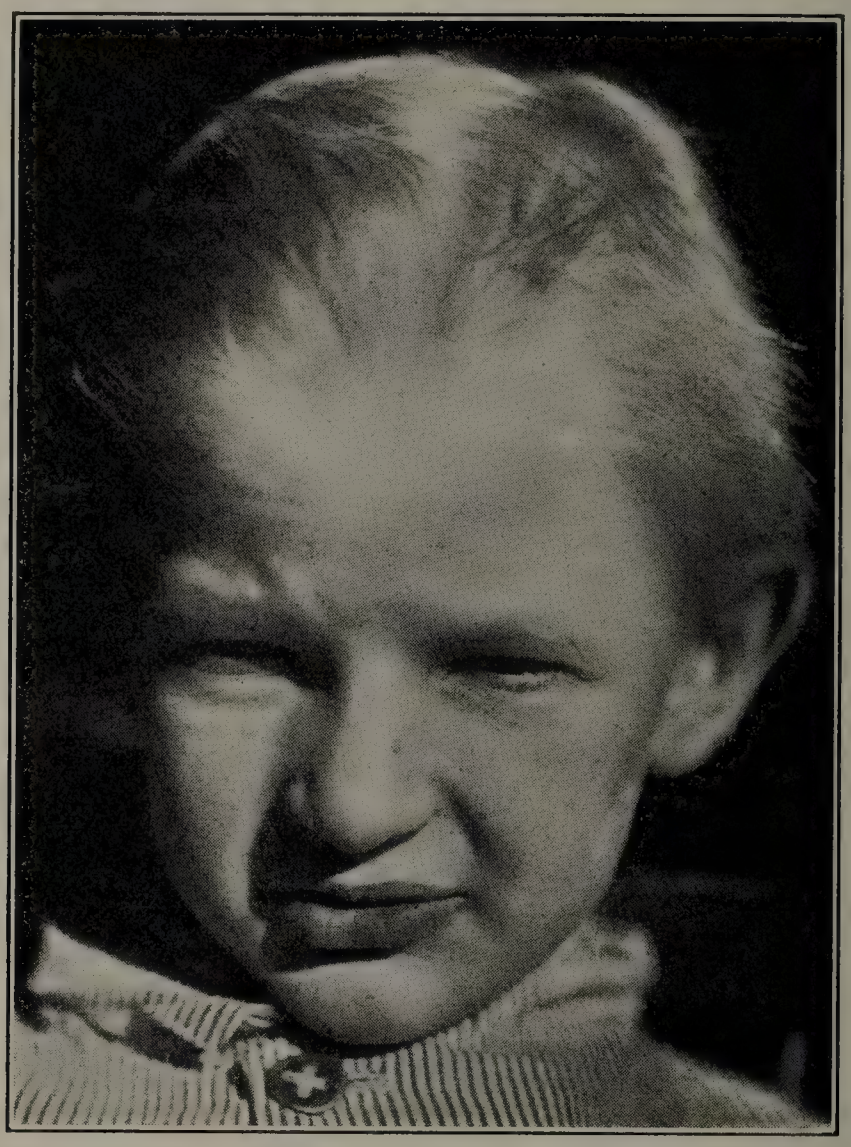


FIG. 114.—Lanugo hair. (Hypothyroid disturbances). (A. Josefson.)

scanty, particularly at their outer edges. The moustache may be scanty. The eyeball is deep set, often lacking luster and without expression or listless. The palpebral fissures are narrowed, frequently unequally so; the teeth irregularly developed. Napoleon was a classical illustration.

Gingivitis is not rare. The nails are brittle or frequently very hard. The extremities have a tendency to be cold, the hands cold, bluish, slightly swollen and moist. Chilblains are not infrequent in winter.

This is a general schematic summary of the chief minor hypothyroid signs. These symptoms may be found entire or in groups. Those most likely to be constant are the defects in development, the hairy anomalies

and the coldness of the extremities, which latter is frequently associated with a hypothermia, with frilosity and tendency to shivering, goose-flesh, chattering of the teeth, etc. A marked hyperesthesia to cold is often found in these types which leads readily to coryzas, to bronchitides and to neuralgic pains.

Hypothyroid constipation is probable. It is a marked feature of the myxedematous and its opposite, diarrhea, is recognized in exophthalmic goiter. The explanation probably lies in the lowered tone of the unstriped muscle fibers, as an altered parasympathetic response to the diminished thyroid. In some mixed types where thyroid lability is



FIG. 115

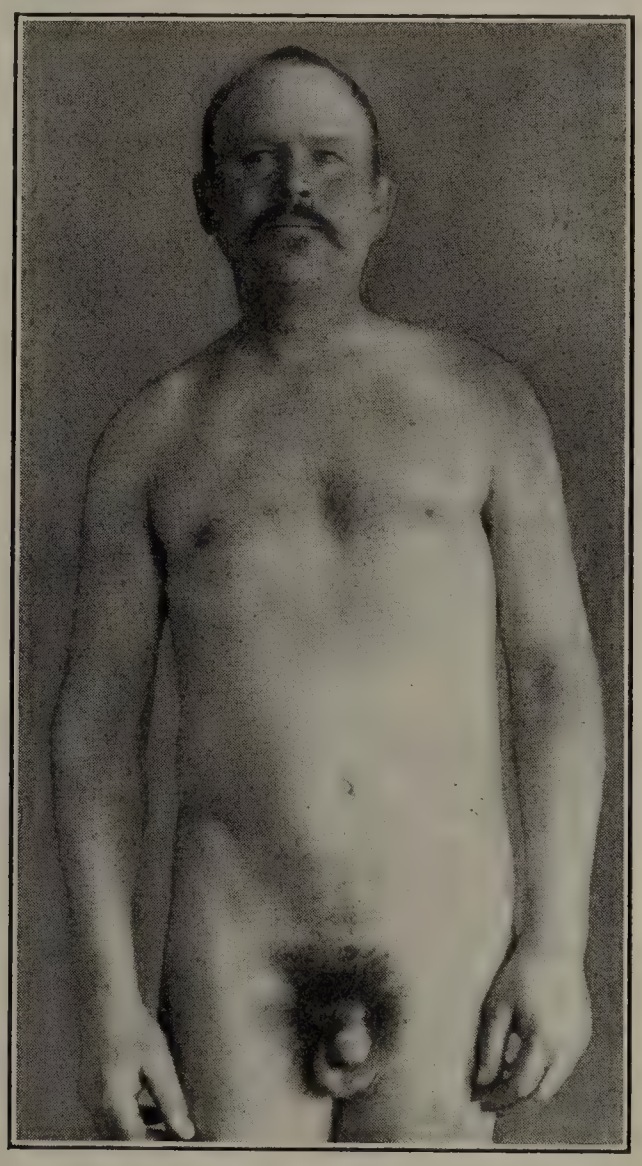


FIG. 116

FIGS. 115 and 116.—Hypothyroidism before and after treatment. (A. Josefson.)

marked (chiefly secondary to marked emotional lability—the so-called nervous, neurotic or hysterical types) alternations of diarrhea and constipation are frequent. This is related to an especially significant type of intestinal movement syknokenosis, *i. e.*, increased frequency of movements, not diarrheal, but soft and frequent. From the vegetative side these are related, particularly by Levi and Rothschild, to thyroid instability, from the psychical side they are related to the symbols of impatience, haste, tenseness, unconscious rather than conscious. They frequently subside on thyroid therapy but are equally amenable to rest and quiet. A more fundamental psychotherapy is called for in others.

A number of other syndromes, much emphasized by Levi and Rothschild may be mentioned. Personal experience has not yet confirmed these completely. They are hemorrhoids, mucomembranous enteritis (undoubtedly thyroidal secondarily, but primarily emotional). Bradycardia is frequent. Transitory edemas, tendency to colds, nasal asthma, respiratory oppression are among the minor signs.

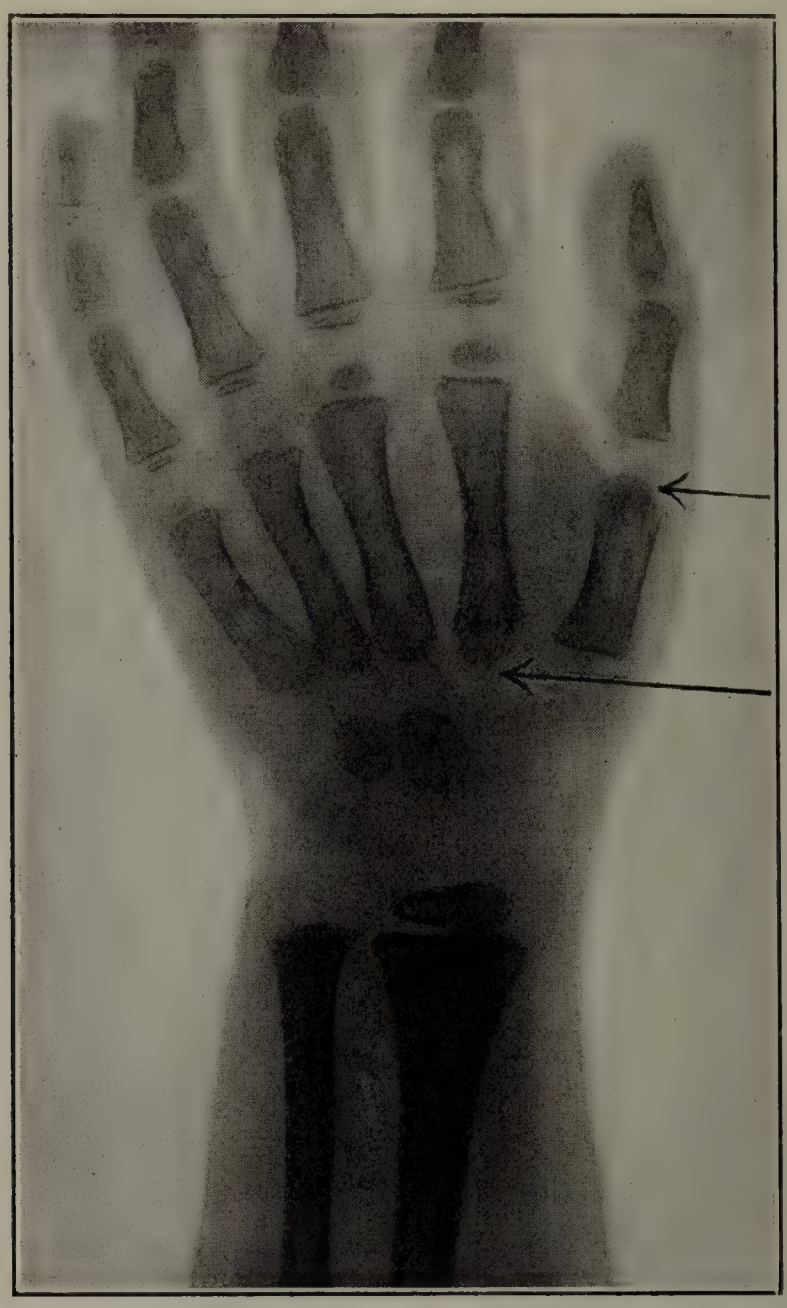


FIG. 117.—Pseudoepiphysis. An endocrinous (hypothyroid) product. (Josefson.)

Certain skin syndromes are closely related to mild hypothyroid states. The skin is usually dry, rough, apt to be thick. It is frequently scaly, even advancing to ichthyosis. The vegetative instability leads to acrocyanosis, to transitory edemas, at times to eczemas on the flexor surfaces, to psoriasis on the extensor surfaces. Urticariæ are not infrequent hypothyroid signs and are closely related to gastro-intestinal inferiorities (vegetative) usually spoken of as anaphylactic reactions to certain (usually) proteids. Acne, herpes, eczema, psoriasis, scleroderma should always be studied with the thyroid in view.

The bony or joint inferiority which may result from defective development on a hypothyroid basis is frequently responded to by chronic arthritides. Sometimes the specific overthrowing lesion is an

infection, again metabolic inferiority is registered by a chronic arthritis. Cases of *chronic rheumatoid arthritis* then should be carefully analyzed with the possibility of hypothyroid states in view.

Pseudoepiphysis, Josefson has shown to be a hypothyroid stigma. (See Fig. 118.)

In the paragraphs on myxedema attention has been directed to the sense-organ deficits. These may show in benign hypothyroid states as corneal opacities, opacities in the vitreous, interstitial keratitis, iritis (so-called rheumatoid iritis).

Pathology of Hypothyroid States.—The study of the histological changes in the thyroid lies outside of the purposes of this work. The most important defect states result from infections, causing acute and chronic thyroiditis, from syphilis and from various aplasias. The changes in the nervous system related to or possibly due to hypothyroid states have been extensively studied. Kojima's work in relation to that carried out by Mott is noteworthy.[1]

The nerve-cell changes are most strikingly seen in certain cases of myxedema and in experimental hypothyroidism. Chromatolysis, particularly within the vegetative nuclei groups, is marked. Vagus and glossopharyngeal nucleus chromatolysis was extreme in certain of Mott's cases. In those patients with marked mental symptoms—myxedematous psychoses or in certain manic-depressive states of the menopause, possibly related to dysthyroid activities, either due to age atrophies or emotionally induced unbalances, there are found extensive cortical and bulbar changes. Precise correlations between the destructive lesions found and the symptomatology are still in too crude a stage to be didactically formulated.

Therapy for Hypothyroidism.—Many contradictions may be found in the literature concerning the use of thyroid substance in various types of myxedema and cretinoid degeneration. This is to be expected since so many observers use their diagnostic terms so lightly. Age differences are not recorded—stage and intensity of the disease—and grade of defect are overlooked, and hence no uniform basis for comparison exists.

Among the best reported results are those of v. Wagner who obtained the following results: a diminution in the myxedematous swelling of the skin, the genitals developed rapidly, the tongue diminished in size, there was loss of the umbilical hernia, development of new hair, dentition was hastened, closure of the fontanelles occurred, and there was an increase in bony development. The psyche was less hopefully modified, but there was a diminution in the apathy, and slight increase in the intellectual capacity was noted.

Early therapy is naturally the main feature. According to v. Wagner small doses of iodine in addition seem to stimulate the thyroid activities still further. Magnus Levy, v. Eysselt, and others also report excellent results, complete cure resulting in some patients still in their teens.

[1] Proceedings Royal Society of Medicine, Psychiatric Section, vol. 8; Mott, ibid., Pathological Section, February 13, 1917.

A widespread state experiment was carried out by v. Kutscheras in Styria by treating 1011 cretins. A large number were neglected by the parents, *i. e.*, treatment was not kept up. In 2.4 per cent. the thyroid tablets could not be well borne. All idiots and severe grades of deafness and dumbness were left alone. Of 440 of the cases 10.2 per cent. showed slight increase in bony development, 4 per cent. showed definite change, 85 per cent. showed an increase well above the average. The increase in bony growth was marked with the younger individuals, but also persisted into the third decade.

A careful revision of 677 cases showed 42.8 per cent. marked improvement, 48 per cent. some definite improvement, 8.6 per cent. no improvement.

Scholz's experiences with 100 cretins in an institution were disappointing. He used as many as eight tablets a day. Von Wagner claims the doses were too extreme, hence the bad results. Emaciation, weakness, loss of appetite, vomiting and diarrhea, and other symptoms of hyperthyroidism developed. Von Wagner recommends the use of only one-half or one tablet (thyroidinum siccum, Merck = gram 0.1); 0.4 gram corresponds to the activity of an entire gland. (Burroughs Welcome Co., 0.1–0.3 gram = gr. iss–v.)

Thyroxin has also been utilized. It seems that the iodine content is not the only factor in the activity of the thyroid substance and it is not as yet definitely demonstrated what the combination is that is most effective.

Surgical implanting of the thyroid gland itself would seem to be the ideal therapy, and experiments directed to this end have been carried out since 1889, when Bircher was one of the first to attempt it. The gland has been implanted in different portions of the body—the neck, under the breast, in the spleen, even in the bony substance. As a rule, however, the implantation has not been as successful as was hoped, the gland itself undergoing retrograde changes. Implantation of human glands has not been brought about with certain therapeutic efficiency.[1]

One is compelled, therefore, to resort in most cases to the dried or liquid preparations of the thyroid itself or to such biochemical products as laboratory research has provided through the utilization of the glandular substance itself or that portion of it which presents its chief hormone activity, Kendall's Thyroxin for example.

The general results of thyroid medication in typical cases are fairly constant. Especially is it of value in the aberrant and minor forms of the disease of which one of the most chronic of symptoms is the persistent anemia. This may be in part overcome by the simultaneous use of small doses of arsenic, which have been recommended by a number of investigators. Alcohol and morphine work disadvantageously, and should be carefully avoided. The use of small doses of sodium bicarbonate and bismuth work advantageously in diarrheal states.

[1] Autotransplantation and Homotransplantation of the Thyroid Gland, Archives of Internal Medicine, 1917.

Thyroid medication for the sporadic cases varies somewhat from its use in the endemic cases. In the sporadic cases of the light or mild type the action is quite similar to that seen in the endemic ones, but as a rule sporadic cases by reason of their longer involvement and the less rapid development of the symptoms, their more hidden or obscure nature with their great mixture of syndromes make them less responsive to the therapy. Nevertheless, many of them respond very kindly to it, the same dosage being utilized.

In the benign hypothyroid states carefully selected thyroid therapy has been of excellent service. Massive doses are at times required to bring about the desired effects; again very minute dosage is sufficient. The use of the endocrinous glands at the present time seems to be going through the characteristic cycle of all enthusiasms. Thyroid is now a universal panacea. Out of this hypertherapeutic activity careful discriminations will come and proper means adapted to help the syndromes which have been here rather hastily summarized. Hormone therapy has its place, the limits of which are being outlined by a careful scrutiny of the vegetative reactions and their relations to the endocrinous hormones.

Hyperthyreoses.—Exophthalmic Goiter.—C. H. Parry (1755–1822), in 1786, and Flajani (1802) were feeling the way to a generalization which Graves, in 1835, Basedow, in 1840 and 1848, rounded out more in its present form. Möbius, in 1886, insisted on the relation of the disorder to changes in the thyroid gland, which Kocher and other surgeons had also pointed out previously.[1]

Exophthalmic goiter is a disorder conditioned by a modification of the activity of thyroid gland substance which in turn leads to an increased activity of the vegetative nervous system with a series of cardiovascular signs, tachycardia, exophthalmos, tremor and increased metabolic activity. Under some circumstances the increase in the glandular activity is primary, the vegetative symptomatology, secondary, under others the reverse. In every case the cause for the hyperactivity should be ascertained before therapy is inaugurated. To operate for an acutely disturbed thyroid due to a severe mental shock is hasty and mostly meddlesome therapy. To attempt a psychoanalysis for a septic or adenomatous thyroiditis is equally farcical. As has been pointed out certain forms of hyperthyroid functioning is a healthy compensation for defective endocrine activity of other glands, adrenal or pituitary. Many of the hyperthyroid states observed among soldiers were due to this interrelationship. A lessening of the adrenal load (from exertion, fear (fight and flight), infections with pituitary therapy, 1 to 3 grains daily, often very shortly restores the impaired

[1] Kocher, A.: Morbus Basedowii. Spec. Path. u. Ther. d. inn. Krankheiten. Kraus-Brugsch., 1, 2. See recent study by Souques in which he states that Parry, an English physician of the eighteenth century, whose posthumous works were published by his son in 1826, described the first recorded cases of exophthalmic goiter. He quotes a passage from Parry's description that is very accurate, including his account of the symptom complex, goiter, tachycardia and exophthalmia.

balance and the thyroid swelling and symptoms of a Basedow's syndrome subside.

The disturbance is widespread and presents many variations. Indeed incomplete and irregular forms are among the commonest of the manifestations of hyperthyroidism. Women much more often than men show this particular type of disturbance. In Sattler's great monograph 3200 of 3800 cases reported were females. Hereditary types are known.

Symptoms.—The chief symptoms are found in the thyroid, eye, heart and bloodvessels, skin, and muscles. Psychical, gastro-intestinal, respiratory, genital, and metabolic changes occur as well.



FIG. 118.—Exophthalmic goiter, showing marked exophthalmos and enlarged thyroid. (Courtesy of Dr. George W. Crile.)



FIG. 119.—Same patient four months after operation (extirpation.) Greatly diminished exophthalmos and change of facial expression. (Courtesy of Dr. George W. Crile.)

It must be emphasized that the symptom groups which are here described apply to the more evident hyperthyroid states. It should constantly be borne in mind that a great many persons, women particularly, as a result of emotional disturbance, often unconscious, suffer from mild toxic thyroid states. The literature is enormous. Sattler's monograph is classical. The more recent enclycopedic résumé on Endocrinology and Metabolism (1922) *loc. cit.* and Kocher's résumé on Endocrinology and Metabolism, and Kocher's studies and Hirsch, *Handbuch,* give summaries of the present situation.

An enlarged thyroid is fairly constant, although occasionally absent. It is soft and elastic, rich in new bloodvessels, pulsates, and varies in volume (often very rapidly). Its variations in volume are fairly com-

mensurate with the intensity of the symptoms. Auscultation of the enlarged thyroid often gives a marked bruit.

The heart action is rapid (tachycardia), and the pulse is very variable, reacting excessively, particularly to psychical influences. The heart sounds are increased in force, the beating being felt in the neck, and the whole chest wall is at times moved by the cardiac tumult. The blood-pressure is rarely raised and the radial and other vessels show marked hypotonus with reddening of the face, ears, and finger extremities.

The *eye* symptoms consist of a marked and variable protrusion of the eyeball, with widened orbital fissure, sometimes greater on one side than the other, and felt as a disagreeable pressure and tension by the patient. The eyelids are at times swollen, and the upper, and possibly the lower lids largely retracted (Dalrymple, Stellwag) independently of and often preceding the protrusion of the eyeballs. The upper lid also does not move synchronously with the lowering or the raising of the eyeball (von Graefe's sign), following more slowly or receding more rapidly (spasticity) than the moving eyeball, in the presence or absence of protrusion. Both signs may be unequally present. The relative infrequency of winking (Stellwag) is a frequent sign. Eppinger shows in tabular form the relative frequency of these ocular phenomena.

Symptom.	Wilbrand and Sänger. Per cent.	Sattler. Per cent.	Eppinger. Per cent.
Protrusion, wide, v. Graefe	23.0	37.3	35.6
Protrusion, v. Graefe	36.0	19.8	16.4
Protrusion	10.4	17.6	19.2
No eye signs	15.3	13.3	4.8
Wide, protrusion		2.2	6.7
v. Graefe, wide	2.6	5.4	9.6
Wide	5.1	1.1	3.8
v. Graefe	7.6	3.3	3.8
Number of cases	39.0	91.0	104.0

Löwi's sign (dilatation from adrenalin) is frequent, but inconstant, and made uncertain because of emotional complicating factors. Occasionally mydriasis is present, less frequently miosis. Irregular or stiff pupils may be observed as well as loss of the accommodation reflexes. Optic nerve atrophy is infrequent, Increased tear secretion is often observed early, dryness late in the disease. Mechanical complications —pus, ulcerations—are met with.

Insufficiency of convergence, without double vision (Möbius's sign), is frequent and is independent of protrusion.

Cardiovascular symptoms are among the most constant and early signs of exophthalmic goiter, and are due to the action of the thyroid secretion. Tachycardia (occasionally paroxysmal) is the most prominent single sign. The pulse may beat from 100 to 160 times a minute —even 200 has been recorded. Undoubted cases may show no rise above 100, and great variability is the rule, especially in response to

psychical stimuli. During sleep, and also on lying down, the pulse frequency slows down.

Angina-like attacks, with hyperalgesia in the left ulnaris region are not infrequent, and in most patients the feeling of distress and anxiety over an increased sense of heart oppression (apart from the tachycardia) is one of the most annoying symptoms. Cardiac dilatation, with later hypertrophy, without valvular defect, may or may not (50 per cent.) accompany the disease and disappear at its termination. On auscultation the first sound is usually accentuated, and systolic murmurs at the base are frequent. Valvular insufficiencies occur under special circumstances, and are often of serious moment in operative cases.

Strong pulsation of the carotids is frequent, and though the large vessels are often prominent, and apparently arteriosclerotic, the walls are usually soft and yielding. Vasomotor instability is frequent. Marked reddening alternates with paleness. Irregular erythemata also are not infrequent and many patients complain of surface heat, seek cold places and light clothing, even in winter, and yet show no temperature anomalies. Dermographism is also a frequent vasomotor phenomenon, and epistaxis is not infrequent, Rarer urticariæ, irregular circumscribed edemas, pruritus, etc., are to be expected.

Skin symptoms are frequent. Increased perspiration is not rare and the skin is always moist with some patients, possibly only on one side, or in isolated (head) areas or smaller spots.

The electrical resistance of the skin (Veraguth-Vigoureux) is diminished as a result of this increased secretory activity. Goose-flesh develops readily with these patients, and changing pigmentary anomalies, chiefly chloasma spots, are present on the skin, not on mucous membranes, more especially on the eyelids, neck, nipples, armpits, and genitals. In many patients (23 per cent., Sattler), there is a tendency for the hair to fall out (sometimes unilaterally) with the development of the disease, and usually there is renewed growth of the hair with improvement.

The nails frequently show deformities and dystrophies (marbling).

Tremor is an early and frequent symptom, and may involve the entire body. It is usually fine, varies from seven to forty vibrations per second, and occasionally is intermingled with wider, irregular choreic-like movements. Psychical rather than physical stimuli increase the tremor greatly; lying down tends to diminish it. It is more apparent in the upper than in the lower extremities and is marked in the eyelids (Rosenbach), in the tongue, and when in the vocal cords produces a peculiar staccato breathing (Minor.)

Digestive disturbances are frequent. Dry mouth may alternate with excessive salivation. There is a marked tendency to chronic alvine discharges (30 per cent.) and to vomiting without anorexia (15 per cent.). Both occur in paroxysms, somewhat resembling tabetic crises. Hour-glass contraction of the stomach may be demonstrated by the roentgen-rays. Both the vomiting and diarrhea are obstinate,

are accompanied by mucus or colloid, at times bloody material and the movements may occur as often as four or five times a day. Fatty stools without diarrhea may occur. With both diarrhea and vomiting the patients are in grave danger. Obstipation of spastic type may also occur. In many patients there are enlarged lymphatics, tonsils, tongue follicles, thymus, and lymphatics of the intestines.

Respiratory symptoms, dyspneic in character are usual. Normal breathing is frequently irregular in depth and rhythm, and seems strained. The swollen gland may cause relative stenosis. Asthmatic tendencies are present, and the general sense of air hunger is striking with nervous pseudohysterical coughing.

Menstrual irregularities are common. The flow is usually small in amount and infrequent in occurrence, with occasionally the direct reverse condition. Thinning of the breasts, and other atrophies (testicles) have been recorded, and seem to be coördinated with thymus anomalies.



FIG. 120.—Exophthalmic goiter. (Hammond.)



FIG. 121.—Exophthalmic goiter. (Hammond.)

Metabolic anomalies are characteristic. The patients become markedly emaciated and get very weak. This is related to a definite nitrogen loss which may be checked by proper amount of hormone, and also to a marked overoxidation of carbohydrates and fats (see Fatty Stools). The carbon dioxide output is increased. This sudden loss of flesh and strength may come on in attacks, and then a distinct improvement takes place. Eppinger speaks of these patients as individuals who, not doing any work, need the carbohydrates of a hard-

working individual. In mild cases an increase in carbohydrates keeps the patient at a normal weight. The increased oxidation also shows in a mild hyperthermia. Alimentary glycosuria is frequent, and gradually disappears on recovery; hyperglycemia, 0.1 per cent. and over, is a common accompaniment, and not infrequently the blood-sugar findings may be utilized as a test for the severity of the disorder. True diabetes is an infrequent complication. Polyuria is frequent (13 per cent., Sattler), less so albuminuria (11 per cent., Sattler).

Studies in the so-called basal metabolism, a much exaggerated generalization, inaugurated chiefly by Magnus Levy, and followed by a host of others seem to point to a relationship between toxicity and the basal metabolism.[1] Means and Aub have followed this line of investigation and show that the relationship is of great prognostic value and further show the great value of roentgen-ray treatment, comparing it favorably to surgical therapy.

Boothby in a recent (1922) study of the basal metabolic rate in exophthalmic goiter and adenomas of the thyroid compared with the rate in conditions other than thyroid in some 7000 cases reports that the basal metabolic rate was determined in 2332 patients having exophthalmic goiter; of these, 94 per cent. had basal rate above + 20 per cent.; 5 per cent. were between + 20 per cent. and + 11 per cent., and in 1 per cent. the rates were normal. In adenoma with hyperthyroidism, the basal metabolic rate was above + 20 per cent. in 68 per cent. of 1425 patients, and between + 20 per cent. and + 11 per cent. in 32 per cent. of the cases. In adenoma without hyperthyroidism the basal metabolic rate was within normal limits in 100 per cent. of 1111 patients. These figures are to be contrasted with those obtained in 2417 patients who had diseases which did not involve the thyroid gland. In this group only 3 per cent. had basal metabolic rates above + 20 per cent.; 11 per cent. had rates between + 20 per cent. and + 11 per cent.; 7 per cent. had rates between − 11 and − 20 per cent.; 2 per cent. had rates below + 20 per cent.; while 77 per cent. of individuals had normal rates between + 10 and − 10 per cent. Of this entire group, 90 per cent. had basal metabolic rates within ∓ 15 per cent. of the Du Bois standards.

The *blood* shows anemic changes frequently at the very outset. The number of red cells is not markedly lowered, as a rule, save in those patients with marked cachexia. The leukocyte count is usually low; the percentage of cells is altered. Lymphocytosis is marked (60 per cent.). The polynuclear neutrophiles are markedly diminished. The eosinophile cells are increased (8 to 20 per cent.). The large mononuclears are normal. Thyroidectomy changes the entire blood picture more toward normal, as does also ligation of the thyroid arteries and according to Kocher the blood picture is a valuable prognostic index. The coagulation time is increased. In the young, hyper-

[1] Magnus, Levy: Berl. klin. Wchnschr., 1895, **32**, 650; Dubois, E. F., Arch. Int. Med., 1916, **17**, 1915; Means-Aub: Arch. Int. Med., 1919, **24**, 404; ibid., 1920; Janney: Arch. Int. Med., 1918, **22**, 187; Hirsch: Handbuch gives the latest literature to 1927.

thyreosis leads to increased growth of the bones, and young exophthalmic patients are apt to be very large and very fat.

The mental symptomatology of hyperthyreosis is of great importance, since from the studies of Parhon and others it seems possible that like others of the symptoms the mental signs may develop almost exclusively. In many cases the psychical signs are mild. The tendency is to both psychomotor and emotional irritability. Moodiness and sudden changes are frequent. In marked cases distinctly manic phases may develop; again acute and deep depressions (often suicidal) take their place. Thus the picture approaches very closely at times to the type of Kraepelin's mixed manic-depressives, or the more typical circular forms of this psychosis. Toxic epiphenomena may take place with ideas of reference, of persecution, even hallucinations, principally of sight. The general picture of an acute delirium is a grave sign.

The analysis of the psychical pictures in exophthalmic goiter is far from complete. Sattler advocates a catholic attitude, saying there is no one typical hyperthyroid psychosis. The present tendency is to ally the mental phenomena of the hyperthyreoses with the manic-depressive group, and to separate certain manic-depressive cases as largely conditioned by hyperthyroid activity on a polyglandular basis.

General Etiology and Pathology.--General considerations relative to the athyreoses, dysthyreoses, and hyperthyreoses as seen in the clinical pictures of congenital and acquired hypofunctioning as in cretinism and myxedema, and in hyperfunctioning as seen in exophthalmic goiter point to paralytic and irritative phenomena of the sympathetic and parasympathetic (autonomic) nervous systems.

The blood changes in exophthalmic goiter and in myxedema are similar, *i. e.*, there is a relative lymphocytosis with diminution of the neutrophile leukocytes. In exophthalmic goiter the coagulation time in increased, in myxedema diminished. In exophthalmic goiter the sympathetic irritation explains the exophthalmos, tachycardia, loss of weight, and the alimentary glycosuria. Autonomic irritability causes the von Graefe, the lymphocytosis, the diarrhea, the increased secretions. The influence of the thyroids on the carbohydrate metabolism, as seen in the rapid emaciation and alimentary glycosuria, possibly acts through the pancreatic retardation or through a relative increase in adrenalin action. That the thymus is involved in the blood picture formation seems certain.

Thus one comes to a combined neurochemical theory in that exophthalmic goiter is dependent upon hyperactivity of the thyroid secretions, which increased secretions act through the visceral or vegetative nervous system. Both autonomic and sympathetic systems are thus in a state of hyperexcitability—a condition the anatomical foundations for which are found in a certain type of individual termed vagotonic by Eppinger.

The detoxication hypothesis of Blum, Ostwald, Kocher, Klose and others, in which a dysthyreosis is assumed with a type of iodine poisoning from insufficient detoxication is ingenious, but not yet

satisfactory. A complete discussion cannot find a place in a text-book and the student is referred to the literature cited.[1]

The full etiology is still very dark. Many apparently healthy individuals suddenly develop the disorder following a shock. This shock frequently involves the complex of the fear of death or the loss of money. There is a characteristic fear reaction. Minute analyses from the psychoanalytic school are not yet sufficiently numerous to permit generalization, but the psychical import of shock is undoubted. In certain personal experiences chronically increased thyroid activity and certain definite unconscious trends have been uniformly associated.[2]

Infectious and toxic types are also recognized—acute thyroiditis, etc., and also forms of acute exacerbation of the thyroid intoxication in individuals who have taken iodides or bromides even in minute doses.

The histological changes in the gland itself are of great moment for the interpretation of the disorders.[3] The adenomatous type is one of great significance. A toxic non-hyperplastic type is emphasized by Plummer. A great variety of variations from the average have been described, chiefly of hyperplastic type. A hyperplastic type associated with liver cirrhosis is one of the anomalies. Apparently perfectly normal glands are at times associated with severe types of the disease. The gland is usually enlarged, elastic, the vessels dilated, and new proliferating bloodvessels are found. Kocher has described the goitrous type as parenchymatous hyperplastic struma, poor in colloid and in iodine; other changes are largely due to complications.

Forms and Diagnosis.—If the classical triad, exophthalmos, tachycardia, and goiter be present, there is little question as to diagnosis, but still all may be absent and yet the patient be suffering from severe hyperthyroidism. Hence great variability may be expected. Eppinger and Hess distinguish two chief groups according to the predominance of the sympathetic or parasympathetic irritative phenomena. These deserve more detailed study. The parasympathetic group, in particular, is often overlooked. These show von Graefe's sign, diarrhea, lymphocytosis and increased perspiration and marked anxiety. They are not infrequently taken for cases of anxiety neurosis or other neurasthenoid hybrids. Severe gastric or enteric crises have led to a mistaken diagnosis of tabes. Undeveloped forms may be readily overlooked, especially when the more classical triad just noted is not present.

Particular attention should be focussed on the thyroid itself. Its rich and increased vascularity tends to give it a peculiar consistency, even when not markedly enlarged, which is very characteristic. Kocher has compared it to the general fulness of the breast of a pregnant or nursing woman.

In a very large number of patients, especially those showing the

[1] Barker, Hoskins and Mosenthal, Claude, 1922; Laignel-Lavastine, 1923; Hirsch, 1927; recent studies by Plummer from the Mayo Clinic, also.

[2] Marañon: Ann. de med., 1921, **9**, 81; Jelliffe: The Psyche and the Vegetative Nervous System, New York Med. Jour., April 5, 1922.

[3] Farrant, R.: Royal Soc. Med., Path. Sect., February 17, 1914; Loeb and Hesselberg: Jour. Med. Research, 1920.

parasympathetic irritation (vagotonic) signs discussed, hypertrophy of other lymphatics is to be observed. These are chiefly to be sought in the thymus, tonsils, tongue and rectal lymphatics. There is a tendency to elongated extremities, scanty beards in men and badly developed genitals in women. Marked lymphocytosis is also present. This relative lymphatism possibly plays a very important compensatory role in the disease.

The patients with marked psychical signs are apt to show both autonomic and sympathetic symptoms. Certain patients show only cardiovascular signs. Such are those described as goitrous heart. They show tachycardia, dilated heart, some respiratory arrhythmia. The eyes are often shiny, pupils dilated, and striking even if not protruded. Dermographia is frequent and dizziness is often complained of. Other closely related forms suffer from dyspnea and bronchial catarrh, bleeding from the nose and congestion of the upper air passages. Neurotic goitrous heart from pressure is another special type often overlooked. There is also unilateral mydriasis, at times tachycardia, and the eye on the pressure side protrudes and recedes. Rarer cases are disguised under mild diabetics, and F. Müller had described a group of pseudosclerosis cases of hyperthyroidism.

The iodine toxic cases form another group. Running from the nose, bronchial catarrh, salivation, stomachic distress, nausea, diarrhea. sleeplessness, headache, and skin eruptions are the more frequently found symptoms which may develop with but small doses of iodides or bromides.

That the blood of patients with exophthalmic goiter contains an increased adrenalin content which in some cases may be experimentally demonstrated in animals, is a point of hypothetical diagnostic value. It also points to the possible relation between this disorder and unconscious "fight or flight."

Occurrence and Course.—The disease is comparatively rare. The proportion of men to women is 1 to 6 on the average. Between fifteen and thirty are the most frequent years of incidence.

The course is usually chronic with ups and downs and many variations. Emotional shocks, behind which simple term there may lie the profoundest and most tragic events of life, almost invariably bring on an attack or increase an existing one. Acute infections frequently bring on exacerbations, while pregnancy frequently acts advantageously. The length of time that the disease persists is extremely variable—from three months to thirty years. The prognosis also varies with the severity of the hyperthyroidism. With healthy individuals the prognosis is relatively good, with distinctly nervous (especially vagotonic) individuals it is less hopeful.

Lymphocytosis, with normal numbers of white cells, is a better prognostic sign than lymphocytosis with leukopenia.

Treatment.—This may be surgical, by internal remedies or by psychotherapy. Surgical treatment is the most radical and should be the last to be considered. It is frequently necessary to save life. It

is often meddlesome and unnecessary. The statistics of various operators have shown improvement in from 6 to 76 per cent., death in from 2 to 22 per cent.[1] Kocher has reported 76 per cent. good results. These figures are probably high, if ultimate results are meant. The operation of choice is the successive elimination of thyroid substance, with minimal handling of the gland. This is advocated particularly by Kocher, whose lethal results have been from 3 to 7 per cent. Death frequently is preceded by narcosis, with a very characteristic symptom-complex. The face gets red, the whole body becomes tremulous and breaks out in perspiration, diarrhea supervenes, the temperature rises and the heart action becomes excessively rapid, and death with cyanosis and dyspnea takes place. Status thymolymphaticus is possibly responsible. Inferior adrenals are also to be considered.

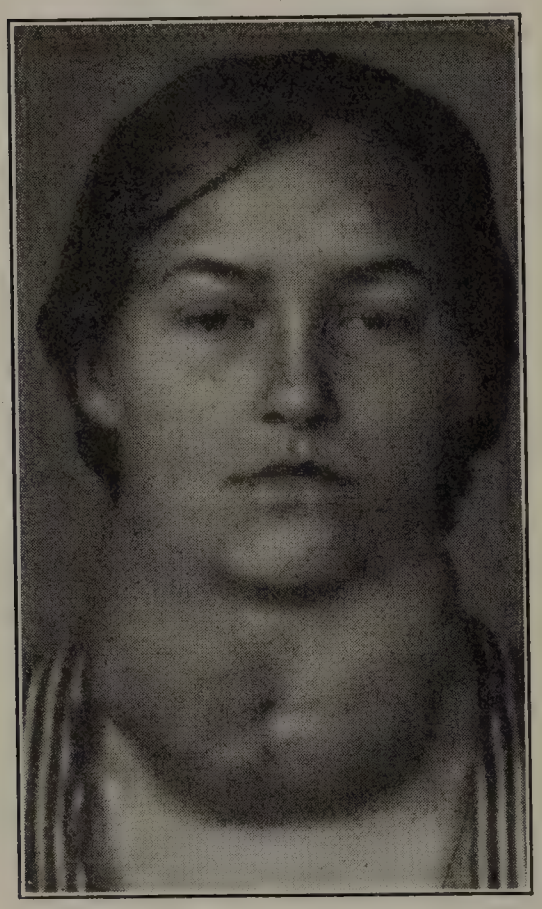


FIG. 122.—Before treatment.



FIG. 123.—After five weeks treatment with great doses.[2]

The operations on the cervical sympathetic are not to be recommended. They help the eye symptoms possibly, but the disease is not one of the cervical sympathetics alone.

Internal therapy is still unsatisfactory. Rest in bed is primary and essential. Any remedy increasing the thyroid secretion is bad, hence thyroids, iodine and bromides are to be avoided. Thymus has been tried with best results in the sympathetic types. The fresh gland is given by mouth. Heliotherapies, roentgen-rays, violet rays, as at present developed offer even better results than surgery and are less radical in removing a necessary organ from the body. The studies

[1] Eppinger, loc. cit., p. 70.
[2] A. Josefson: Hygiea, 1915, p. 517.

of Means and Aub (loc. cit.), offer certain criteria, from the basal metabolism curves, when operation seems alone justifiable in the face of failure by light therapy. The chronic infectious, hyperplastic goiters do best with light therapy.

Pharmacological agents which act to diminish the thyroid secretion have been used. Chief of these is belladonna. It is the most reliable of the internal remedies and can be given in fairly large doses. Adrenalin in solution by rectum is of service at times. Vagotonic cases react best to its influence, the tachycardia and diarrhea being well influenced by it. The digitalis group of glucosides are not to be recommended, neither is iron of any service. Certain cases react very advantageously to extremely minute doses of thyroid (Psychotherapy?). Pituitary is useful in thyroid overcompensations for adrenal deficiency. A combined thymus and pituitary therapy helps a great many neurotic unsatisfied women (sex repressions usually with an unconscious masturbatory basis) whose dissatisfactions and childish impatience with reality keeps them in a constant state of adrenal exhaustion in which an attempted thyroid compensation causes chronic hyperthyroid signs of a minor grade.

Psychotherapy is above all of great value, especially as applied toward an education of the patient concerning his fears, in the sense of Dubois. This is a combined rest and reëducation therapy which Dubois claims has been successful in the vast majority of cases. Psychoanalysis is of the most signal service in a large group of cases, especially in readjusting the patient to his unconscious wishes, revolts and rebellions. Psychotherapy is of value in the initial stages particularly, especially to determine why the thyroid is overfunctioning. It is particularly valuable as a follow-up of a surgical operation which has been performed to save life.[1]

Serum treatments aiming to exert a lytic action upon the secretory cells of the thyroid have been devised. As they do nothing but destroy structure they are not particularly useful.

PARATHYROID SYNDROMES.[2]

The parathyroid glands arise in man from epithelial outgrowths on the third and fourth branchial clefts. That from the third cleft usually lies free from the thyroid in most animals save in man. Accessory parathyroids are present in different neck structures and parathyroid tissue is frequently found in the thymus gland. In man the chief parathyroid masses are imbedded in and blended with the thyroid tissue, although distinct from it. In general the removal of two or more of the parathyroids gives rise to the disorder termed tetany.

[1] See Lewis, Jelliffe, Schwarz and others on psychoanalytic psychotherapy in hyperthyroid states.

[2] Richardson: Thyroid and Parathyroid. Morel: Les Parathyroides, 1912. Pepere Paratiroidee, and papers by Dustin, Uhlenhuth and others on the reciprocal relations of thymus and thyroid and parathyroid. Noël Paton and L. Findley: The Parathyroids, Quart. Jour. Exper. Physiol., 1916–1917, **10**, 203.

Definite knowledge concerning parathyroid functioning is slowly accumulating. It is undoubtedly a general reservoir of hormone-containing substances which aid in the proper distribution of calcium in the body. This calcium may be ionized or combined as it circulates in the blood. Collip[1] and associates have isolated the hormone; also claimed by Berman.

Disturbances of calcium metabolism, which is not improbably regulated from other sources as well as the parathyroid, are of much moment to internal medicine. One of the greatest factors in resisting infection seems to be bound up in this function.[2] This function we cannot take up here although it is a neurological problem of far reaching significance.

Calcium as an essential element in the proper distribution of nervous impulses is most strikingly studied in the disorder known as tetany. Jelliffe has shown that in the analysis of certain epileptics faulty parathyroid functioning may play a part in certain examples of this syndrome.[3] He has found that in those epileptics in whom spasmophilic signs are present, as in various tetanies, etc., that parathyroid, given by rectum, not by mouth, has been beneficial.

Tetany.—Corvisart first used this term "tetanie" in 1852. Frankl-Hochwart, in 1887, clarified the conception, and since his striking description the term tetany has had universal recognition.

As early as 1815 J. Clarke described the symptoms, Cheyne in 1819 Steinheim in 1830 and Hall in 1841 also gave sufficiently detailed histories of convulsive states to permit the recognition of tetany.

Tetany is most satisfactorily interpreted as a disorder of the vegetative control of the calcium metabolism of the body, alterations of which modify the electrical permeability of the neuron membrane of the synapse, causing marked hyperfunctioning of the sensory and motor spinal mechanisms. As the parathyroids are possibly the chief regulators of the calcium ions in the bodily fluids tetany is preëminently a result of parathyroid hypofunctioning.[4]

Symptoms.—Tetany has, as its main symptoms, tonic, intermittent, bilateral, often painful cramps, which, without, for the most part, any loss of consciousness, involve the muscles of the upper extremities, particularly the hand, which is held in the obstetrical position. The muscles of the lower extremities may also be involved, those of the

[1] Jour. Biol. Chem, 1925, **63**, 395, 439, 1925, **66**, 133. Berman, L.: Proc. Soc. Biol., 1923, **21**, 465 Asher, in Hirsch Handbuch, vol. **2**, 254.

[2] Grove and Vines: Calcium Deficiencies: Their Treatment by Parathyroid, British Med. Jour., May 20, 1922. Elias, Kornfeld and Weissbarth: Beiträge z. Path. u. Klinik d. Tetanie, Wien. Arch. f. i. Med., 1922-1923, 1924, et seq. IV, V, VI.

[3] Jelliffe, S. E.: Parathyroid and Convulsive States, New York Med. Jour., December 4, 1920.

[4] See Osterhaut: Botanical Gazette, 1915, et seq., for valuable studies on the alterations of electrical permeability of cell membranes, due to bivalent kations. Also see excellent summary of situation by Collip: Jour. Biol. Chem., 1925, **63**, 395; also in Asher: Hirsch Handbuch, 1927, **2**, 254. Turpin: La Tétanie infantile, Paris, 1925, with excellent bibliography to 1925. Hyperventilation tetanies (Rosett, Foerster, *et al*) should be recorded here. Also see Spiegel: Ztschr. f. d. ges. Neurol. u. Psychiat., **70**, 13. Adlersberg and Porges, Monograph, also Wien. Arch. inn. med., 1924, **8**. Jelliffe: Tetany, in Osler's Mod. Med., 1928, vol. **6**.

larynx, of the face, and of the jaw, seldom those of the chest, abdomen, neck, diaphragm, or tongue. In rarer cases the eyeball muscles are implicated, as is also the bladder. In the sensory sphere paresthesiæ and pains are present, while hyperesthesiæ occur now and then. Pressure upon the brachial plexus may give rise to an attack (Trousseau); hyperexcitability to electrical currents is present (Erb); mechanical hyperexcitability of the muscles and motor nerves is observed (Chvostek), while the sensory hyperactivity to mechanical and electrical stimuli is also present (Hoffmann). The psyche is rarely uninvolved, and following operative removal there has developed extreme anxiety with the sense of impending dissolution.

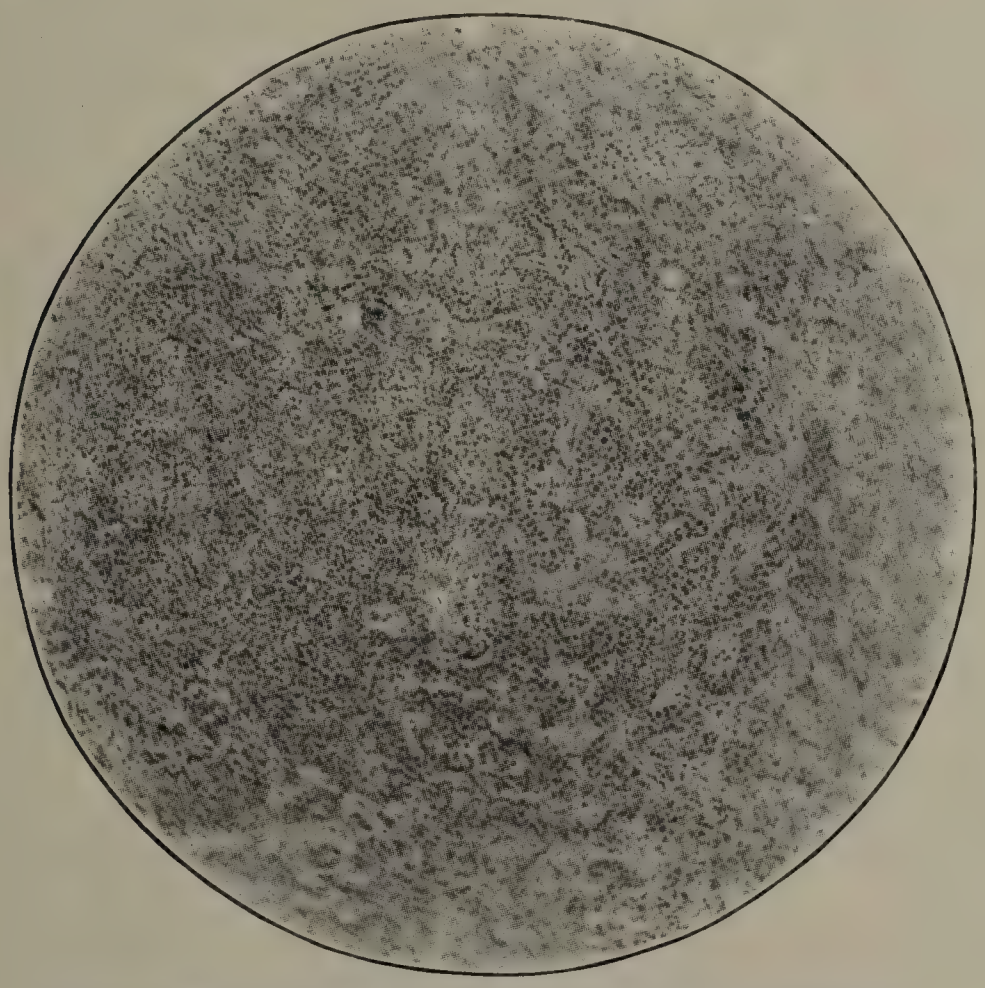


FIG. 124.—Adenoma in the parathyroid. (Josefson.)

In chronic and repeating forms secretory and trophic disturbances occur, such as increased perspiration, reddening of the skin, swellings of the joints, mild edema, falling out of the hair (alopecia) and nails, discoloration of the skin, urticaria, and herpes. Dyspnea may intervene; polyuria and glycosuria are rare accompanying symptoms. Incomplete forms have been designated "tetanoid" by Frankl-Hochwart.

For didactic purposes Frankl-Hochwart divides tetany into simple and acute forms and chronic recurring forms. A further division of forms occurring in children and in adults is made. Tetany of the adult he groups into seven classes: (1) Tetany idiopathica—tetany of otherwise healthy individuals—workman's tetany. This is the form which seems to occur epidemically as an acute, or acutely recurring affection in certain cities, notably Vienna, Heidelberg, etc., principally

in the early spring months, and among certain handworkers—tailors, shoemakers, etc. (2) The tetany of gastric and intestinal affections. (3) The tetanies of acute infectious diseases, typhoid fever, encephalitis, measles, scarlet fever. (4) The tetanies of acute poisoning, chloroform, morphine, ergot, phosphorus, renal, and gonadal substances. (5) The tetanies of maternity (pregnancy, parturition and lactation). (6) The tetanies of parathyroid involvement. (7) The tetanies accompanying other nervous diseases, exophthalmic goiter, brain tumors, cysticerci, syringomyelia, etc. (8) Hyperventilation tetanies.

Incidence.—Tetany in its different manifestations is very rarely seen, and is even less frequently reported. In undeveloped phases the tetanoid reaction is comparatively frequent in children. Gastric tetanies are probably the most frequent, while the pure epidemic form has not been encountered in the United States. In Griffith's study only 77 cases were found recorded, while Howard's later collection brings the American cases to 154 in 1907.

Etiology.—Whether the work of MacCallum and Voegtlin[1] has solved this problem is to be determined, but it would appear that an essential factor has been found in the relation of the parathyroid to the calcium metabolism of the body. The hyperexcitability of the neuromuscular apparatus is primarily due to a change (chiefly a deficiency) in the amount of the calcium in the blood, and this is thought to be due to a relative or absolute insufficiency of the parathyroid glands. Changes in the calcium content (bivalent kations) alter the permeability of cell membranes to electrical stimuli, and the tetany reaction may be due to a lowering of the synaptic threshold to sensory stimuli (analogous to strychnine). Stimuli summation with overaction then occurs.[2] Hyperventilation can induce tetany in health.

Uhlenhuth has shown in the tadpoles of certain salamanders that thymus feeding brings on tetany. He argues that the thymus contains a tetany toxin. We believe the thymus feeding causes regression in the thyroid and parathyroid glands (proved by Dustin and others); this causes a defective parathyroid functioning and thus disarranges the bivalent kations of the body fluids (*Ca. Mg.*). Greenwald and Gross believe that the parathyroid hormone keeps calcium phosphate in solution. Paton[3] and others argue that the presence of guanidin bases is responsible for the tetany reacton. They do not work out the mechanism in kinetic terms and Collip, Clark and Scott's more recent work (1924–1925–1926) seems to negative the guanidin hypothesis.

Pathology.—Concerning the histological changes, the present view excludes a specific pathology. The insufficiency of the parathyroids, be it relative or complete, may be brought about by a great variety of lesions. These in reality offer evidence in favor of the parathyroid

[1] Jour. Exp. Med., 1909, **11**, 118, Jour. Am. Med. Assn., 1912, **59**, 319, 327; Proc. Exp. Biol., 1911, **9**, 23; American Journal of Insanity, 1909; MacCallum and Vogel, Jour. Exp. Med., 1913, **18**, 618, 646.

[2] Jelliffe: Transactions, Am. Neur. Soc., 1919.

[3] Quart. Jour. Exper. Physiol., 1916–1917, **10**, 203, et. seq. Greenwald and Gross: Jour. Biol. Chem., 1925, **66**, 217.

insufficiency hypothesis, but go no further. In the minor grades of tetany in children, particularly in so-called spasmophiles which Frankl-Hochwart regards as tetany, the findings of Yanase, of Escherisch's clinic, are illuminating. Here hemorrhages in the parathyroid seemed fairly constant findings, and offer an explanation of the galvanic hyperexcitability. At the other extreme one finds the absolute insufficiency tetanies in experimental parathyreopriva. In acute epidemic forms thyroid (and probably parathyroid) involvements are known. Tumors, tuberculosis and a host of other changes in the thyroids have been described. It will probably be found that in most of these the parathyroids are likewise implicated. Thus, in exophthalmic goiter a combination of thyroid and parathyroid symptoms is often present. In many tetanies pure thyroid symptoms appear. Tetany with healthy (somatically) glands has been observed, but it must be recalled that defective calcium metabolism while preëminently arising from parathyroid insufficiency, may arise from other causes.

Symptoms.—Considerable variation is to be found, but in general four types of symptoms are observable in the fully developed attack. These are the muscular spasms, which may go on to an exhaustion paralysis, or paresis; the Trousseau phenomenon; increased electrical excitability, or the Erb symptom; and mechanical hyperexcitability of the muscles—Chvostek's sign. In some patients one or more of these may be missing. Incomplete forms, so-called, may present even fewer signs. On the other hand, a richer combination of symptoms apparently closely related to the general disorder, may be encountered. Sensory disturbances, anomalies of circulation with edema, of respiraston with cyanosis, and of temperature are sometimes found. True psychoses, perhaps indistinguishable from the hysterical confusions, are found. Lost tendon reflexes in severe cases, trophic disorders of the skin, hair (alopecia areata), and nails occur. Epileptic seizures occur rarely (hyperventilation).

Course.—Clinicians have recognized arbitrarily three groups of cases in adults, and most modern authors are inclined to follow Trousseau in his classical description. In the *benign* form the sensory phenomena, such as formication or a simple sensation of heat, may precede the spasms. These are confined for the most part to the hands or occasionally to the feet. The contractions may be fleeting, persisting from five to fifteen minutes, or they may persist for an hour or more. Often the attack terminates by a recurrence of the sensory symptoms. A period of repose lasting for a quarter of an hour to two or three hours supervenes, and the spasms recur. In some instances two or three attacks a day may persist for several months. Again, more *severe* attacks are observed. These occur more often in young adults, the benign forms having been mostly observed among children. Here the contractions are more violent and more painful. The preceding sensory phenomena are usually more pronounced, and with the increase in the severity of the condition other symptoms may be noted. Headache, malaise, and a rise in temperature of 1° to 3° F. may be noted. The

affected muscles may show signs of congestion, and localized edema of the hands and feet may be observed. Other muscles than those of the extremities may be involved.



FIG. 125.—Tetanic spasm, showing flexion of wrist. (Pool.)

These severe attacks are rarer than the benign ones. Frankl-Hochwart has shown that there is a distinct tendency for the well



FIG. 126.—Tetanic spasm, showing plantar flexion of foot and toes. (Pool.)

marked lighter cases in many instances to become graver, and the good prognosis which most writers have given is seriously doubted by this observer.

In the *grave form* there is no addition of symptoms. The attacks occur with greater and greater frequency, and become more and more intense, and the patients die as a direct result.

Diagnosis.—The diagnosis of a classical case offers few difficulties. In English-speaking countries it is apt to be overlooked, although the more frequent reports of recent years point to the fact that it is being recognized more often, especially in its milder forms. The presence of cramps in the upper extremities, alone or in conjunction with the upper limbs, with the classical obstetrical hand and the additional evidence supplied by the Chvostek, Trousseau, and Erb signs, is usually sufficient to determine a diagnosis.



FIG. 127.—Method of producing tetanic spasm of hand by stretching the brachial plexus by forcible abduction of the arm. Note "obstetrical" hand. (Pool.)

Tetany strumipriva, or, better, *parathyreopriva*, as suggested by Erdheim,[1] offers the most classical manifestations of the disorder, throwing, as well, considerable light upon some of the possible underlying and fundamental features of this peculiar reaction type. Insufficiency of the parathyroids results in convulsive phenomena of the tetany type.

Prognosis.—The point of view here maintained precludes the possibility of the statement of a general prognosis. Very little is known definitely of the prognosis in infants and children. Most authors agree in giving a fairly good prognosis, although Frankl-Hochwart says that healthy children rarely acquire convulsions, and that the prognosis is not good. In many of these children only one tetany-like spasm has been noted. In others the spasms may persist for weeks and even months. In simple cases the prognosis is much better than in those complicated especially with gastric or intestinal affections.

[1] Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1900, vol. **16.**

Bronchitis, pneumonia, and occasionally an ascaris infection also determined a less favorable prognosis. Dangerous signs appear with glossal cramps, which may cause death. Recurrences are frequent in those who recover.

Tetany coming on during pregnancy and childbirth usually has a good prognosis. The hyperexcitability of the nervous system may persist for weeks after delivery. In succeeding pregnancies the recurrence of the phenomenon may be looked for. .

In the cases apparently due to disturbances of the stomach surgical interference has brought about distinct amelioration. Sudden death may occur, and apart from surgical intervention the prognosis is admittedly bad (70 to 80 per cent.). The cases are comparatively rare, however. In severe cases associated with marked gastric dilatation, operation, if only exploratory, is advisable. The mortality after operation in some dozen or more cases now reported is as low as 30 per cent. The subsequent history of these patients remains to be reported.

Tetany following infectious diseases and acute or chronic poisoning seems to present a favorable prognosis, perhaps the best of the various forms. Latent tetanies may be provoked by hyperventilation.

The prognosis of tetany thyreopriva depends upon the amount of thyroid gland removed and whether the parathyroids are included. Total extirpation of the entire thyroid and parathyroid tissue is recognized to have a fatal outcome. Tetany appears after total removal of the parathyroids, not only in man, but in lower animals.

Treatment.—From the standpoint here outlined it may be readily deduced that the treatment must be carefully worked out for each individual case. The parathyroidectomized individual would not be benefited by a gastric operation.

With a positive diagnosis established, the organ involved should come into review. Inasmuch as parathyroid insufficiency is the most general cause, it is rational to treat these cases, many in children, the whole group of so-called idiopathic tetanies, many tetanies of pregnancy and of the thyroid disease with the thyroid and parathyroid preparations. Collip's hormone seems to fulfil most of the conditions, yet occasionally the combined thyroid and parathyroid involvement renders the giving of the combined products of more service. Collip has emphasized that his substance must be used with great care.

The use of foodstuffs rich in calcium and of calcium salts follows as a natural corollary from the studies enumerated. Such medication may entirely replace the use of the glandular substances themselves. In experimental tetanies the success of the calcium salts has been very striking, and in tetanies in children calcium therapy has given almost uniformly good results.[1] Curare, opium, hyoscyamus, the

[1] See Howland and Marriott: Quart. Jour. Med., July, 1918, for good discussion. The dynamic concept of the organism as a transmitter of energy is not utilized in this otherwise excellent study. Also consult Jelliffe: Trans. Am. Neur. Soc., 1919. Osler and McCrae: Mod. Med., 1928, vol. **6**. Greenwald and Gross: Jour. Biol. Chem., 1925, **66**, 217. Adlersberg and Porges: l. c., Förster. Deut. Gesell., 16th Congress. Report, Rossett.

bromides, chloral, chloroform, galvanism, sweat baths, etc., go into the discard. Up to the present time therapeutic experience is not sufficient to definitely prove the durability of calcium medication in the cases in which it seems to be indicated. Light therapy has been of service in some cases. It has some effect upon calcium release.

The surgical expedient of transplanting parathyroid tissue has proved successful in animal work;[1] its successful application in persistent chronic tetanies in man is not yet on a firm basis.

DISEASES OF THE HYPOPHYSIS.

The terms pituitary and hypophysis have been employed synonymously and the present usage is still conflicting. The term pituitary by some designates the collective structure made up of two distinct parts with very different functions, yet functionating as a whole.[2] An anterior part (pars anterior), the hypophysis, which is epithelial and derived from the gastro-enteron, and a posterior part (pars nervosa), which is nervous in origin and termed the infundibular process. A pars intermedia separates the two. By others the hypophysis signifies the whole complex. It is epithelial in origin, and invests the pars nervosa, the neck and the stalk of the pituitary. It is derived from the oral cavity and contributes its secretion to the cerebrospinal fluid.[3] The whole structure is innervated by the vegetative nervous system.[4] By reason of its special relations to the optic chiasm, third, fourth and sixth nerves and to the infundibulum alterations in the gland produce not only syndromes correlated with the internal secretions—tethelin (anterior lobe) and pituitrin (posterior lobe), but also may give rise to profound neurological disturbances of these contiguous structures. Accessory pituitary structures are known: Parahypophysis, hypophysis pharyngea.

The (1922) discussion by Biedl may be given in résumé.

The pituitary consists anatomically of the anterior lobe (the prehypophysis), the intermediate lobe (the pars intermedia), the posterior lobe (the neurohypophysis) and the pars tuberalis; furthermore, there are the accessory parahypophyses; above all, the pharyngeal hypophysis and the remnant of the embryonic hypophysial cavity.

In their embryonic development, the pars intermedia and the pars tuberalis are derived from the ectoderm, and the anterior lobe and the accessory formations from the entoderm; while the neurohypophysis is a part of the brain floor.

From the anterior lobe, a lipoid substance called tethelin can be obtained; the intermediate lobe and the posterior lobe contain, aside from one or more substances of specific pharmacodynamic effect,

[1] Leischner: Arch. f. klin. Chir., 1907, **84**, 1, 208.

[2] Bell, Blair: The Pituitary, London and New York, 1919. Bailey, Percival: Ergeb. d. Physiol., 1922, **20**, 162. Aschner in Hirsch's Handbuch, 1927, vol. **2**. Cushing: The Pituitary and its Disorders, 1910. Retrospect: Jour. Am. Med. Assn., vol. **76**. Josefson, in Hirsch's Handbuch, 1927, **3**, 203.

[3] Berkley: Brain, 1894, **17**, 515.

[4] Foix and Nicolesco, Dresel, and see chapter on Vegetative Nervous System.

histamin and histamin-like substances, which are decomposition products of protein bodies. The substance of the anterior lobe possesses a specific activity as a stimulator of growth; while the peculiar, more closely analyzed effects of extracts of the intermediate and posterior lobes find an extensive therapeutic application. The relationship between the pituitary and the other endosecretory organs may be recognized by the structural changes which take place, during the various stages of sexual life, in the pathological variations of functions, and the changes of function experimentally produced, in the several endosecretory organs (genital gland, thyroid, suprarenals, pancreas), on the one hand, and, on the other hand, in the structural changes of the latter associated with changes in the activity of the hypophysis.

The functional performances of the various portions of the pituitary can be deduced from the large number of existing experimental and clinical observations, taken in conjunction with the anatomico-pathological findings; and the following principles may be enunciated:

1. The pituitary is an organ necessary to life, as evidence of which hypophysial cachexia and cachexia hypophysiopriva, in both man and animals are produced.

2. The anterior lobe of the hypophysis contains gland elements which affect growth through the action of a morphogenetic hormone. As is likewise true of the pharyngeal hypophysis, its functional importance pertains chiefly to the period of growth; in the adult, it becomes important only in case of hyperactivity. As evidence of this, one finds dwarfism and infantilism as the expression of arrested development following the experimental removal of the anterior lobe in animals; nanosomia pituitaria, hypophysial infantilism, ateliosis and senilism as the results of hypofunctioning associated with underdevelopment, or following destructive processes in the anterior lobe in the presence of tumors which arise in the lobe itself or in the hypophysial canal; also, the fact that hypophysial infantilism is affected by anterior lobe medication.

As the opposite of arrested growth, one finds gigantism, the result of hyperplasia and tumors of the anterior lobe, which arise before the close of the period of physiological growth; acromegaly associated with hyperplasias and especially with eosinophilic adenomas of the anterior lobe; acromegaloid manifestations associated with hyperplasias of other varieties of cells, pointing possibly to several substances, affecting growth in various ways. The nature of the disordered functioning is not as yet understood; nor is there any very tangible evidence for the assumption that certain adenomas of the anterior lobe yield secretions which influence metabolism. The disturbances of metabolism associated with acromegaly are due to the effects of a tumor on the functioning of the intermediate lobe. Further evidence is furnished through the application of roentgen irradiation to hypophysial tumors and by their operability and the results of operations that have been hitherto performed.

3. The function of the pars tuberalis has not as yet been clearly ascertained.

4. The intermediate lobe is a gland affecting metabolism and possessing a correlative functional action. The secretion of the intermediate lobe passes through the posterior lobe and the stem of the hypophysis, serving to establish a connection with important synapses and tracts located in the midbrain. The evidence for this conception lies in the obesity, together with genital dystrophy, that follows experimental lesions and destruction of the pars intermedia, following obstruction or division of the hypophysial stem in animals, manifestations which occur at all ages but which are more marked in young animals, owing to the arrest of growth caused by the removal of the anterior lobe. Adiposo-genital dystrophy in man, as described by Fröhlich, is caused by arrested development and destruction of the intermediate lobe, by pressure of tumors on this tissue, or by factors which prevent the flow of secretion through the hypophysial stem, such as hydrocephalus or growing tumors. A careful analysis of the effect of the secretion of the pars intermedia, either direct or through the mediation of the nervous metabolism center, lends further support to this theory. A newly established syndrome is: congenital malformations (atresia ani, polydactylism, retinitis pigmentosa); deformities of the skull with retarded mental development; marked obesity, with or without genital hypoplasia, and peculiar disturbances of digestion (occurrence in two members of a family, in whom there were no changes in the hypophysis and no signs of a brain tumor or of pathological cerebral pressure, but diabetes insipidus was a concomitant symptom).

5. The physiological significance of the topical relationship of the anterior lobe affecting growth and the intermediate lobe influencing metabolism, and the reason for their union in the hypophysial apparatus are not fully understood as yet, but it should be noted that analogous conditions in other endosecretory organs are known. From the pathological standpoint, they constitute the basis of manifold combinations of affections that relate to the hypophysis. The position of the hypophysis in the endocrine system is determined by the correlative relations of its parts, evidence for which is found in the anomalies of growth already mentioned as commonly associated with disturbances of secretion.

A more complete discussion of the physiology of the gland cannot be given here; the student is referred to the works noted in the introduction to the endocrinopathies: the monographs of Cushing, Hinsdale, Falta, Noël Paton, Schaefer, Lewandowsky, Pende, Bell, Barker, *et al.*, are the most available and reliable. Tilney's important monograph (1911) gives the chief anatomical features. Beck has given an interesting historical résumé of the early observations.[1] Percival

[1] Beck: Endocrinology, 1922, **6**, 40. Bailey: Die Funktion der Hypophysis Cerebri, Ergeb. d. Phys., 1922, **20**, 162. Aschner, B.: Physiologie der Hypophyse, Hirsch's Handbuch, 1927, vol. **2**.

Bailey has given the available physiological knowledge to 1922. Aschner to 1927. As yet the neural associations especially with gonadal structures, are imperfectly worked out. No real knowledge of hypophyseal function can be formulated until this is done.[1]

As yet the complex syndromy of the varying grades of involvement of anterior and posterior lobes is not completely analyzed. Robertson has isolated a substance called tethalein from the anterior lobes which are stated to have marked bony growth functions. It is possible that phosphorus metabolism factors are here partially concentrated.

A large number of substances have been thought to have been isolated from the pituitary proper. Some of these (pitutirin[2]) have a definite action on unstriped muscular fiber, hence, cause uterine contractions, raise blood-pressure, induce peristalsis, cause bladder contracture (polyuria). Others cause increased milk secretion, increase the sugar mobilization and modify metabolism in general; thus diminished action causes increase in weight, in fatty deposits of the lipomatoses types, differing from the fatty states of hypothyroid types. Changes in the cerebrospinal fluid flow and modification of gonadal activities are frequent. An interaction with the pineal seems to underlie hypergenital developments and *vice versa*.

Hyperfunction of the anterior lobe may take place at any time, either primarily or as compensatory to other defect states. The chief impulse in bony development seems to be exerted on the long bones —hence giants, acromegaly, disproportionately long arms or legs, prognathism, stumpy fingers. A closed sella may limit this capacity for growth. This, if the clinoids do not erode as they frequently do, often gives rise to a definite headache syndrome[1] which is relieved as the clinoids erode or the gland can enlarge, permanently or periodically. There may be daily, monthly or yearly functional changes in the size of the hypophysis. These are seen in hibernating animals and at menstrual epochs in women and may be related to uterine contractions, metrorrhagias, etc. Diminished hypophysial development may show as various dwarfings, small hands, micromiria, small close teeth, high palatal arches, faulty hair development, adiposity, with or without mental (social) defects. Such defect states of minimal grade are very frequent and various psychosexual retardations—narcissism, overt and latent homosexual trends, etc.—are among the possibilities.

As may be seen a bewildering array of combinations may be worked out in such a scheme. The more outstanding syndromes will be here spoken of as: (1) *Hyperpituitarism* is associated with gigantism and with acromegaly; (2) *hypopituitarism* with various grades of infantilism, physical and mental, with adiposity and genital dystrophies;

[1] See Aschner, loc. cit.: Berblinger, Göttingen, 1923, Dresel, loc. cit. (Med. Klin., 1923, **19**), Greving, loc. cit. (also Ergeb. d. Anat., **24, 27**), Karplus and Kriedl, loc. cit. Müller and Greving: Med. Klin., 1929, vol. **21**. Schiff, Wien. klin. Wchnschr., 1925, vol. **38**, Spiegel, loc. cit. and Ztschr. f. d. g. Neur. u. Psych., 1920, vol. **22**.

[2] For literature on pituitrin see Aschner's chapter in Hirsch's Handbuch, vol. **2**.

(3) *dyspituitarism* shows many mixed syndromes. Absolute loss of the pituitary occurs very rarely.

Percival Bailey (loc. cit.) has suggested a tentative summary of the various syndromies as follows:

I. Anterior Lobe.	
A. Hypoactivity	
1. Preadolescent	Lorain-Levi types.
(*a*) Aneoplastic	
(*b*) Neoplastic	
2. Postadolescent	Amenorrhea, dysmenorrhea, metrorrhagia, which follow treatment of anterior lobes; no sign of disturbance of posterior lobes.
(*a*) Aneoplastic	
(*b*) Neoplastic	
B. Hyperactivity	
1. Preadolescent	Gigantism (no evidence for posterior lobe disturbance).
(*a*) Aneoplastic	
(*b*) Neoplastic	
2. Postadolescent	Acromegaly (no evidence for posterior lobe disturbance).
(*a*) Aneoplastic	
(*b*) Neoplastic	
II. Posterior Lobe.	
A. Hypoactivity	
1. Pars intermedia	Polyuria (Bab) reaction to pituitrin indications of disturbances of anterior lobe and pars nervosa lacking.
2. Pars Nervosa	Hypophyseal adiposity (diminished metabolism, increased sugar tolerance, polyuria and signs of disturbance of anterior lobe of absent).
B. Hyperactibity	Hypophyseal glycosuria (increase metabolism, diminished sugar tolerance, polyuria and signs of anterior lobe disturbance absent).
III. Both Lobes.	
A. Anterior and posterior lobes	
1. Hypoactivity	Froehlich's type with or without polyuria (Biedl).
2. Hyperactivity	Gigantism or acromegaly with increased metabolism and diminished sugar tolerance. Adiposity lacking.
3. Heteroactivity	
(*a*) Anterior lobe hyperactive Posterior lobe hypoactive	Gigantism or acromegaly with polyuria.
(*b*) Anterior lobe hypoactive Posterior lobe hyperactive	Genital aplasia, dwarfism. Amenorrhea, etc., with hypopyseal glycosuria. Increased metabolism and diminished sugar tolerance.

Hyperpituitarism: Acromegaly and Gigantism.—These conditions are closely related, showing overgrowth in the skeleton and particularly in the long bones in gigantism; changes in the toes, fingers and bones of the face, more prominent in the acromegalic tendency. In general gigantism occurs when the disorder begins prior to epiphyseal union, acromegaly when the changes occur after the union of the epiphyses. Prodromata such as fatigue, muscular pains, apathy and sleepiness are frequent.

Acromegaly was first clearly outlined by Pierre Marie in 1886,[1] although earlier cases were described by Brigidi, Verga and others. (See Beck) loc. cit. Marie's pupil Souza-Leite published in 1890 a complete thesis.

It is characterized by the gradual enlargement of the bones of the nose, jaw, hands and feet and a hyperplasia of all of the bony structures due to an overactivity of the vegetative nervous system. This over-excitability of the nervous regulators of metabolism, from excessive glandular secretion, also induces hyperplasia of other endocrinous structures, notably the thyroids, interstitial gonadal cells and the suprarenal cortex. From these contributory factors arise a medley (often contradictory) of thyroid, genital and vascular anomalies, some in the nature of hyperplasias (see Thyroid), some of a degenerative or

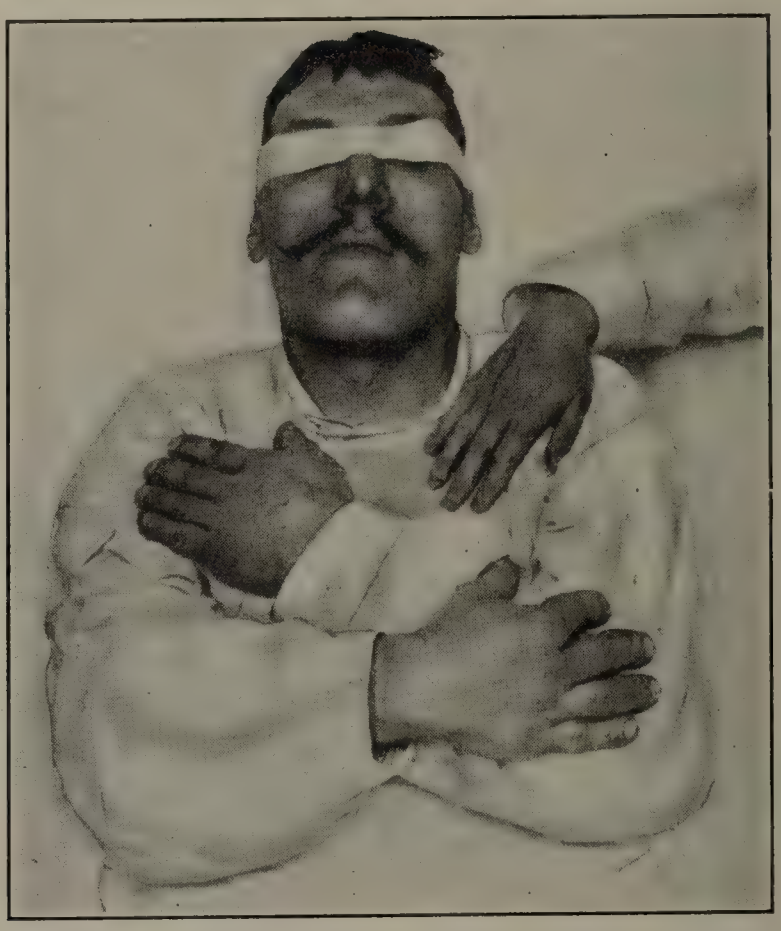


FIG. 128.—Acromegaly. (Josefson, Hygiea, 1904.)

inhibitive character (see Gonads), such as hairy and genital defects. The change in the hypophysis itself is most frequently of an adenomatous or adenosarcomatous type, although this is not essential. In pure adenomata of the hypophysis the symptoms tend to be more clean-cut and classical. As a rule the whole pituitary is implicated which brings the posterior lobe (infundibulin) into increased or diminished activity with contradictory and mixed syndromes, the minute details of which must be looked for in the rich and growing periodical literature.

Acromegaly may arise without appreciable hypophyseal pathology (Petrén). Gigantism is known as a compensation reaction to gonadal insufficiency.

[1] Marie, P.: Revue de méd., 1886.

Symptoms.—The growth in acromegaly is very gradual, usually occurring between the ages of twenty and forty. It includes changes in the skin and hair as well as in the bones. These latter are all hypertrophied, causing striking peculiarities in appearance, particularly of the face. The nose is greatly thickened, as are also the superciliary ridges and the malar bones. The eyebrows are heavy, overhanging and coarse; the lips are thickened and protrude, with marked projection of the often enormously hypertrophied lower jaw. This



FIG. 129.—Acromegaly. (Josefson, Hygiea, 1904.)

hypertrophy causes the spreading of the teeth. The mucous membranes share in the hypertrophy. This marked cranial bone alteration, with the presence of the tumor causes a greatly enlarged sella turcica. The hands and feet are notably widened, the fingers and toes stumpy and thick. The skin and hair throughout, inclusive of the genitals, show the same hypertrophies, as do practically all of the bones of the skeleton. Amenorrhea is frequent in women and loss of potency in men, usually associated with atrophy of the gonads. Glycosuria is frequent. Carbohydrate tolerance may be high, however, and an

increased fondness for sweets is frequent. The muscular tissues have a tendency to atrophy early in the disorder following hypertrophy.

Anomalies dependent upon thyroid alterations are frequent. These consist, for the most part, of increased sweating, tachycardia, diarrheas, exophthalmos at times, Stelwag's symptom, irregular palpebral fissures, variations in pupillary equality, tremor, thermal alterations and marked irritability. Suprarenal cortex alteration is apparently related to the arteriosclerosis frequently seen, diabetes mellitus or glycosuria, and other signs of altered adrenalin (*q. v.*) activity.

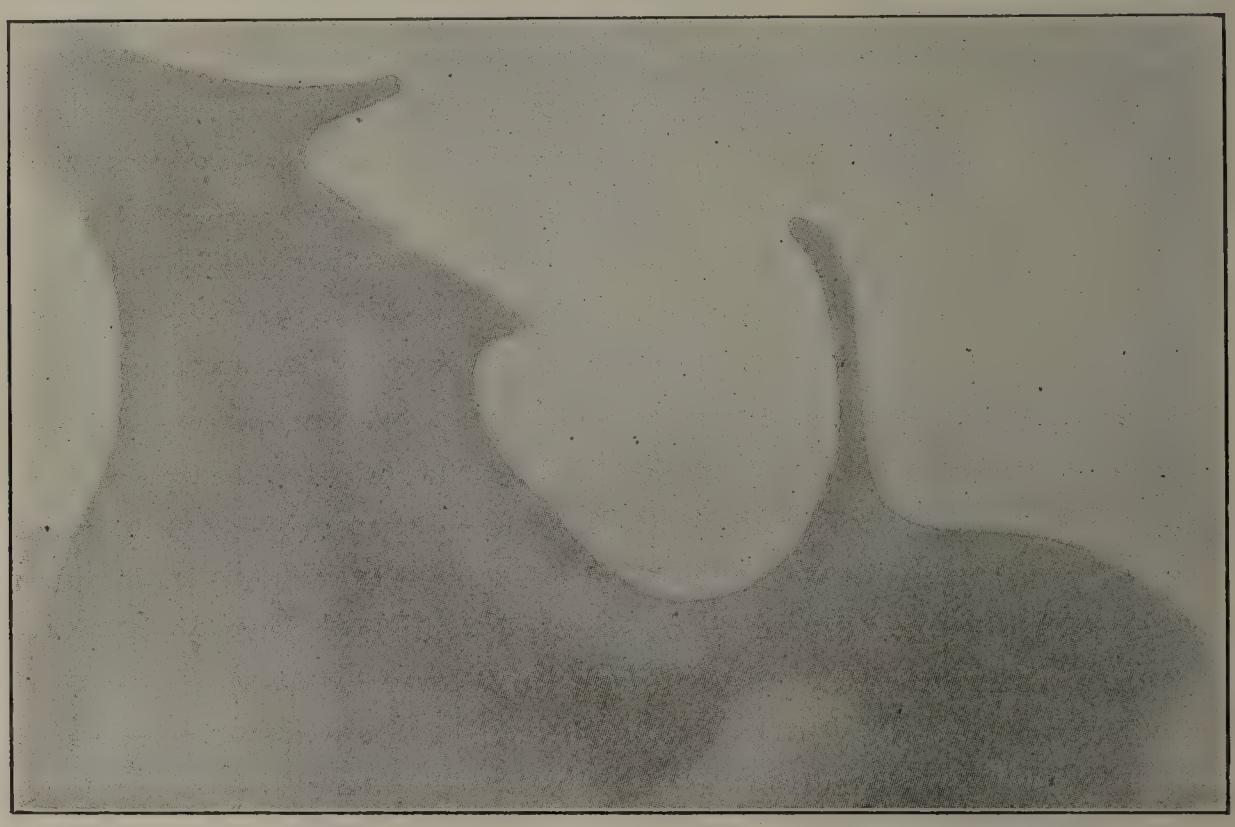


FIG. 130.—Sella turcica in patient with acromegaly (Josefson.)

In addition to the essential metabolic disturbances, symptoms due to the nature of the producing lesions—tumor, hyperplasia, *i. e.*, neighborhood symptoms, are frequently found, but these are not invariable.

Severe bitemporal headaches are frequent. This is an intracranial pressure sign. The sella turcica is usually enlarged from tumor formation, as disclosed by the roentgen-ray examination.

Pressure upon the optic nerves at the chiasm is usual, leading to various types of hemianopsia or even blindness. Distorted fields are the rule.

Mental symptoms ranging from sluggishness to severe deterioration occur, but are not invariable. Epileptic attacks may accompany hypophysial deficiency.

Prognosis.—This is always grave. The disorder is progressive, usually very gradual, five to twenty years, but the advance in symptoms may be arrested spontaneously. As yet no positive mode of influencing slight grades of hyperpituitarism is known. Polyglandular experimentation is widely employed. Thyroid preparations are useful in those cases with accompanying hypothyroidism. Careful analysis

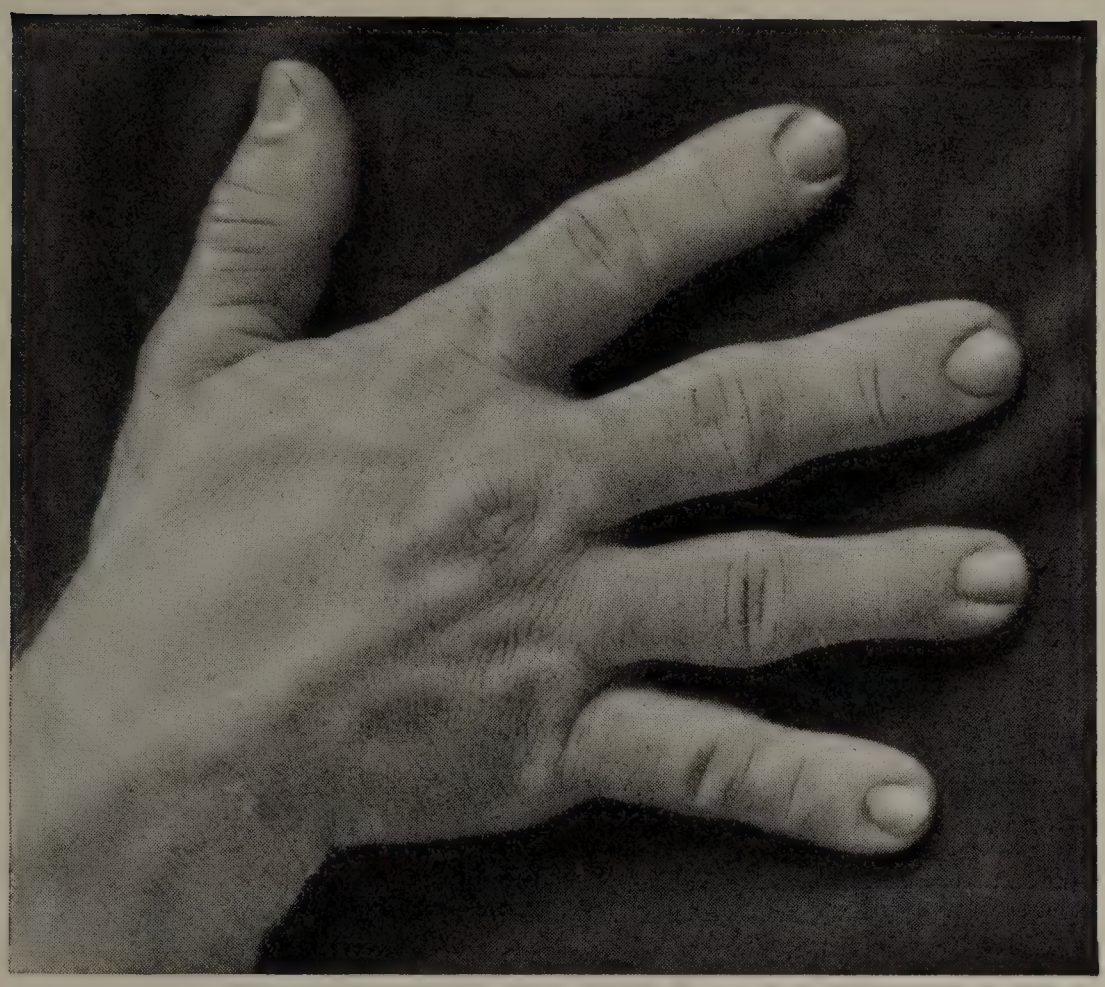


FIG. 131.—Characteristic hand of acromegaly. Note heaping of tissues about nails, "type en large" of Marie. Compare with Fig. 132. (From Cushing's "Pituitary Body.")



FIG. 132.—Typical tapering hand of adolescent hypopituitarism. Compare with Fig. 131. (From Cushing's "Pituitary Body.")

of the symptoms will afford other suggestive clues. Light therapy has been useful in some cases. When pressure symptoms, enlarged sella, visual defects develop, operation is advisable. The results have at times been brilliant.

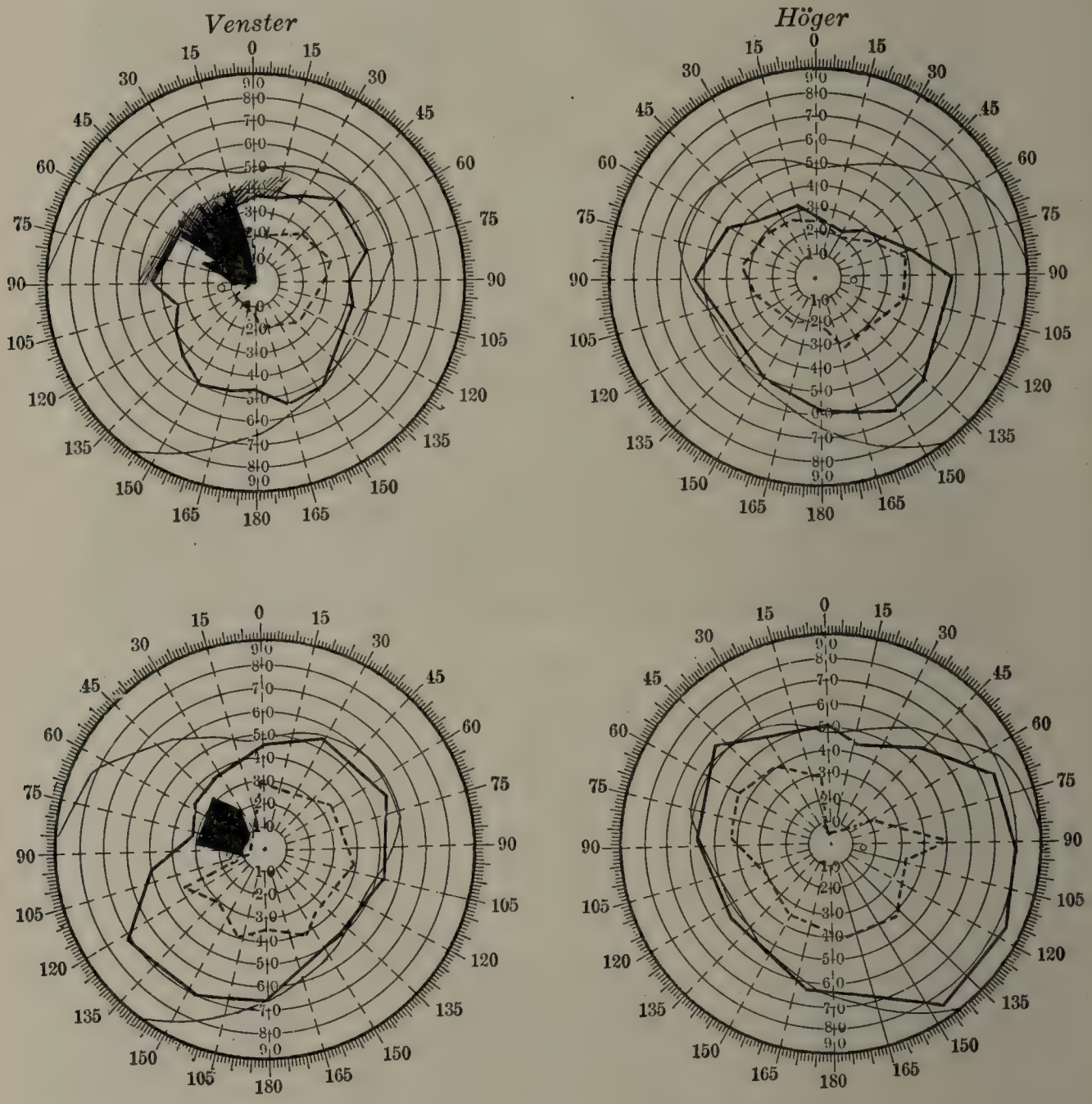


Fig. 133.—Visual fields in case of acromegaly. (Kl. Monatsbl. f. Aughk., 1915, 55. Om akromegalia och hypofystümorer, 1903.) (A. Josefson.)

Hypopituitarism.—Deficiency of the pituitary substance through faulty neural mechanisms, or disease of the body itself, gives rise to a group of syndromes the most classical of which is Fröhlich's *dystrophia adiposogenitalis*[1] a name given by Bartels, probably a dyspituitary whole gland disorder and observed as early as 1840 by B. Mohr (Beck) and possibly by Morgagni in 1766.

Definition.—The clinical picture is characterized by a progressive accumulation of fat, often localized, chiefly about the buttocks and breasts, as is seen in the gonadal disturbances of eunuchs. This is possibly correlated with defective activity of the interstitial glands—from parasympathetic and sympathetic action of the deficient pituitrin, with the consequent failure of development of the secondary sexual

[1] Fröhlich: Wien. klin. Rund., 1901, **15**, 883.

characters, involving the genitals. The psychosexual development even is hindered, showing as various grades of conscious and unconscious homosexuality. Polyuria is frequent and additional pressure signs (tumor) may be found (optic nerve changes).



FIG. 134.—Case of post-traumatic hypopituitarism in a child, with extreme adiposity, high sugar tolerance, and epilepsy. Marked improvement with whole gland feeding (pituitary). (From Cushing's "Pituitary Body.")

Etiology and Pathogenesis.—A not infrequent cause for hypopituitarism is hydrocephalus. This occurring in the young from numerous causes—acute inflammation, hereditary syphilis, tuberculosis, polioencephalomyelitis, etc.—tends to bring about a compression of the pituitary with atrophy. Tumors which in the early stages may cause acromegalic symptoms, may later lead to destruction with defect symptoms. Tumors may also bring about the syndrome without acromegalic features. Frankl-Hochwart[1] has made a collection of a large number of these.

[1] XVIth International Congress, Budapest, 1909.

The pathogenesis is not clear. Loss of pituitary substance itself is a *sine qua non*, but whether this loss acts purely chemically or is mediated by the vegetative nervous system is still not understood. The latter hypothesis is the more favored.

Symptoms.—The disorder is chiefly developed in youth. The obesity is the most striking feature in the youthful cases.[1] The hips, buttocks, mons Veneris and mammary glands are the chief localizations. The lower abdomen is involved in both young and older cases. Fatty cuffs on the malleoli; clavicular collar-like thickening are other local sites for the accumulation. Cases without obesity are known.

The skin is alabaster-like, and in the adult types is cold, hard, and dry and exfoliates readily. At times it is myxedematous. The hairy parts are much smoother or all hair is absent.

The genitals are underdeveloped, penis small, and buried in cushions of fat. The scrotum is small and the testicles may not descend. The labiæ remain infantile, the ovaries small and the breast glands defective. Menstruation is irregular.

The voice may remain thin and child-like and the type of object fixation remain infantile (asexual or homosexual).[2]

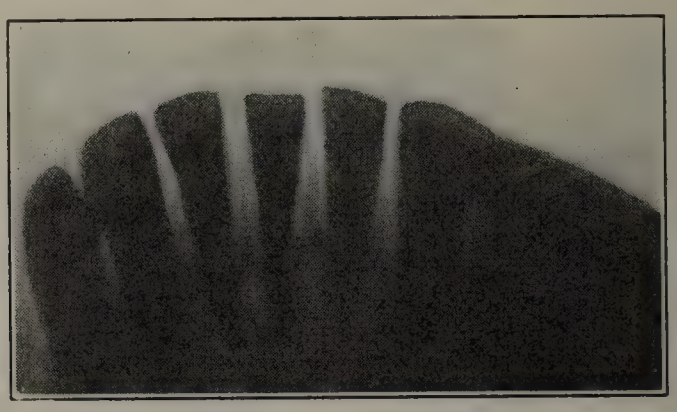


FIG. 135.—Spreading of the teeth in hyperpituitary disorder. (Josefson.)

As a rule there is a fairly persistent though slight subnormal temperature, a marked degree of sugar tolerance, marked reduction in respiratory exchange, slowed pulse and a tendency to sluggishness or even sleepiness. The blood picture tends to show a slightly reduced red cell count, reduction in hemoglobin, the neutrophiles are distinctly reduced, the mononuclears, lymphocytes and eosinophiles increased. Taper fingers are a contrasting picture to the pudgy ones of acromegaly.

The patients often remain childish in their stature (the lower extremities usually being much larger proportionately to the upper in direct contrast with gonadal infantilism), and in their psyche. Lilliputian dwarfism is associated with hypophyseal defect (teratoma).[3]

Neighborhood symptoms may also be observed, as with the acromegalic patients. The optic nerve changes, bitemporal hemianopsia, are among the most important. Other symptoms of a general nature

[1] Consult Falta's discussion, p. 320, Philadelphia.

[2] See an important study by O'Malley, Jour. Nervous and Mental Disease, July, 1918, for various hypophysial defect states, mental syndromes and psychosexual anomalies.

[3] Konne: Deutsch. med. Wchnschr., 1915.

due to pressure, as headache, nausea, vomiting, changes in the sella turcica, etc., often occur, especially from tumors which destroy the hypophysis. Trigeminal neuralgia has been observed.

The syndrome may occur from interruption of hypophyseal pathways in the infundibulum.



FIG. 136.—Hypopituitarism in boy. (A. Josefson.)



FIG. 137.—Hypopituitarism in man. Tumor hypophysis; twenty-nine years. (A. Josefson, Kl. Mon. f. Augenh., 1915.)

Dyspituitarism.—Under this head, a great number of constitutional anomalies due to disturbed pituitary secretions may be gathered. These are incomplete forms of acromegaly and gigantism, cases of adiposity, alone or with genital atrophies, or genital anomalies, showing hyperfunction or hypofunction. Various epilepsies, probably conditioned by hydrocephalic changes occur with dyspituitarism and at times are helped by pituitary therapy.[1] These have been recorded as early as 1705 by Vieussens, by Greding (1781) Wenzel (1810). Variations in mental capacity are frequent, as well as a variety of ano-

[1] Munson, J. F.: Pituitary Gland in Epileptics II, Arch. Int. Med., April, 1918.

malies such as increased sugar tolerance or glycosuria; slightly sub- or supranormal temperatures, polyuria, wakefulness, irritability and a group of character anomalies as well. The skin is usually smooth and soft and free from moisture, and the hair is apt to be thin, fine and scanty. A great variation in cases will be found and, in fact, nearly all the dyspituitary syndromes are polyglandular in their manifestations.[1]

The total absence of pituitary substance brings about conditions of lethargy and narcolepsy, with marked slowing of the pulse and of the respiration. There is insensibility to pain, marked reduction in temperature and in blood-pressure and slow death.

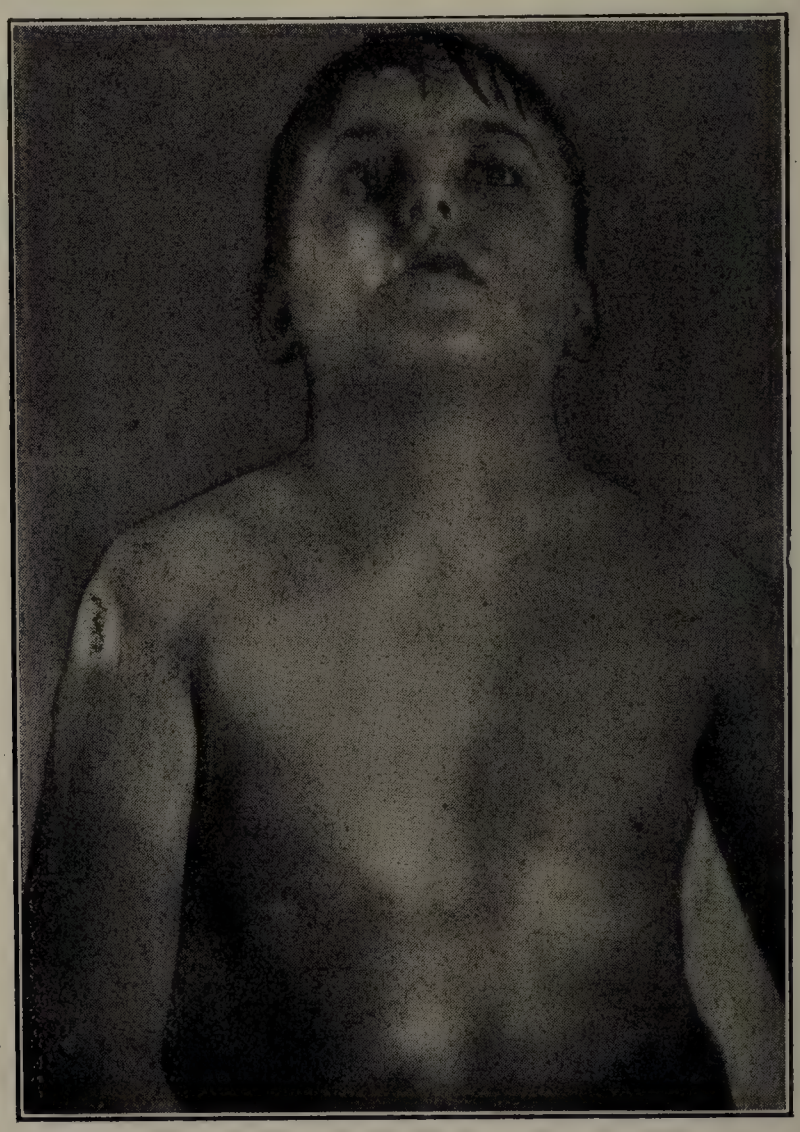


FIG. 138.—Adipose genital dystrophy. Hypopituitarism. Tumor of pineal. (Bailey and Jelliffe.)

Syndrome of Rénon-Delille.[2, 3]—These authors have described a syndrome of hypophysial insufficiency characterized by lowering of the arterial tension, tachycardia, diminution in the amount of urine, insomnia, increase in perspiration, and inability to stand heat. These symptoms, often confused with a so-called functional myocarditis, clear up under hypophysial medication.

[1] Beck: Am. Jour. Med. Sci., November, 1918.
[2] Congress de Méd., Paris, 1907.
[3] Thèse de Paris, Steinheil, 1909.

Treatment.—Acromegalic patients, or those showing pituitary syndromes due to evident tumor of the hypophysial region, need surgical intervention, whether the signs of hypo- or hyperpituitarism be present. Hypopituitary and dyspituitary cases without neighborhood symptoms of tumor may be given pituitary extract (0.1 gm. of combined extracts) sometimes to advantage. In certain refractory cases combined opotherapy, pituitrin and thyroid may be employed to advantage. Certain stationary acromegalic cases are benefited by this treatment and others apparently are rendered stationary.

Radiotherapy is in general inefficient.

The chief surgical modes of relief are: (1) sellar decompression, for hypophysial headaches, or to permit a tumor mass to expand outside of the cranial cavity; (2) partial removal of a hyperplastic and overacting gland; (3) partial removal for the sake of saving eyesight; (4) subtemporal decompression to relieve general brain pressure symptoms; (5) Anton's callosal puncture; (6) combined operations;[1] (7) operations for glandular transplantation.

Infundibular Syndromes.—Little positive concerning a pure syndrome of posterior lobe disease is established. Its exclusive removal seems to be well borne by animals if the anterior lobe is left intact. Its active principle, infundibulin (hypophysin), acts much like adrenalin but apparently through other parts of the vegetative reflex arc than does adrenalin, the chemical structure of which it does not resemble. In animals (rats) feeding experiments have shown that it has a retarding effect on the development of the sex glands, in contrast with a marked stimulation from feeding with the anterior lobe extract.[2]

Polyuria, arrested growth, low temperature and adiposity are often combined with tumor of the infundibular portion of the hypophysis. As the different parts of that structure arises closely related a definite syndromy for purely posterior disorder is still in the making.[3]

DISEASES OF THE PINEAL ORGAN.

Pineal Syndrome.[4]—By Gaskell the pineal gland (epiphysis) is said to appear as a vestigial remnant of the paired median eyes of the paleostracean ancestor of the vertebrate stock. As low down in the animal phylum as Ammocoetes only one of these structures, right, persists and it is rudimentary. Through Meynert's bundle connection is made with the ganglion habenulæ, traces of which still persist in the human brain. Gaskell believes this ganglion habenulæ to be the primitive optic ganglion of the median eye. Researches by Tilney seem to disprove this general assertion and show a separate embryo-

[1] See Cushing: Pituitary Body, Lippincott, 1912.

[2] Weber and Schmidt: Am. Jour. Med. Sci., December, 1916.

[3] Newmark: Infundibular Tumor, Arch. Int. Med., 1917. Ley: Rev. Neur., 1922, **39**, 317. See Bailey, P. loc. cit., Hirsch, lot. cit.

[4] Bailey and Jelliffe: Arch. f. Int. Med., 1912. Jelliffe: Pineal, in Endocrinology and Metabolism, Barker, Hoskens, and Mosenthal. D. Appleton & Co., New York, 1922. Diseases of Pineal.

logical and phyletic origin for the pineal gland and the pineal eye. He also shows fairly definitely the endocrinous nature of the pineal body.[1] That the pineal still retains phototaxic and thermotaxic regulatory functions seems probable (Jelliffe).

The varying syndromes are still only hazily outlined. Timme reports muscular defect syndromes associated with pineal defect. Pineal therapy advocated has not given us results.

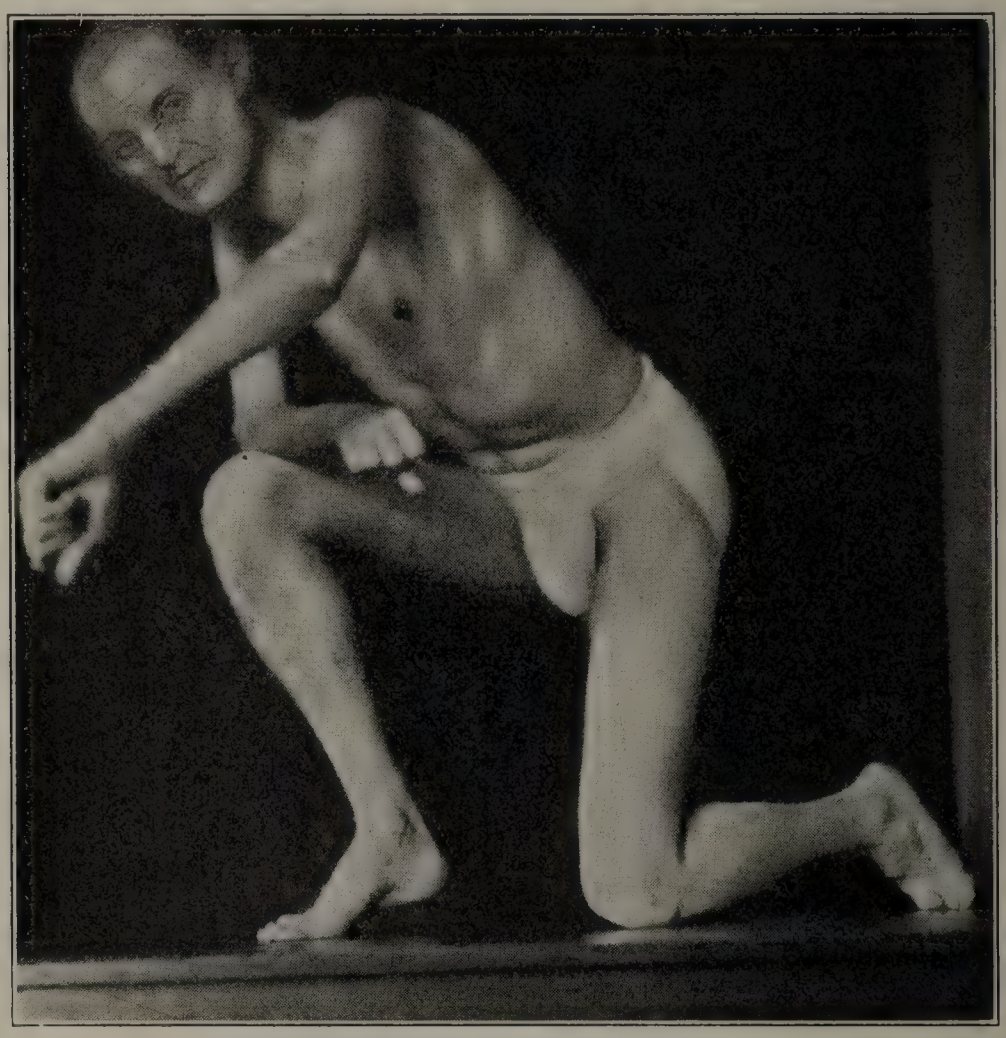


FIG. 139.—Progressive muscular dystrophy supposedly related to pineal gland defect. The roentgen-ray picture of the brain showed a pineal shadow. (Timme.)

Tumors of the pineal, chiefly teratomata, when they become about one-half inch in diameter, cause a striking group of symptoms due chiefly to (1) a hydrocephalus, which causes the adiposogenitalis syndrome of Fröhlich, in part; a sexual precocity, in part, and (2) by pressure upon the quadrigemina, certain ocular palsies. A combination of these is the pineal syndrome. Partial pineal syndromes in which fatty and muscular anomalies are present seem to show a relationship between the hormone action of this structure and muscular dystrophic states. (See Muscular Dystrophies.[2])

Absence of the pineal is known with no certain corrollaries. Precocious sexual development is one of the pineal syndromes.

An interaction between the pituitary and the pineal seems to be established and a giant type, a small genital type and a large genital

[1] Tilney: The Pineal Body, Memoirs, Wistar Institute, 1918.
[2] Timme: Progressive Muscular Dystrophy, Arch. of Int. Med., 1917.

type may result from reciprocal pituitary and pineal balances. The large genital type seems to come on as the pineal involutes and the pituitary enlarges. This is still conjectural.

Operative interference may save a patient with pineal tumor but the technical surgical difficulties are extremely great. Pineal gland therapy is still in an experimental stage.

DISEASES OF THE SUPRARENAL BODY.[1]

Suprarenal Syndromes.—From the standpoint of comparative anatomy[2] the adrenal glands, the sympathetic part of the vegetative nervous system and the chromaffin tissue must be considered as one in relation to the blood vascular system. From the lowest vertebrate forms there is a close relation between the adrenal bodies and the sympathetic system. Gaskell shows that this relation is present as low down as the annelids.[3] The sympathetic nervous system and the adjuvant adrenalin-secreting system are found in their earliest form in the worms. They here consist of cells situated in the central nervous system which are the common ancestors of both, and which are both secretory and nervous in function. These cells are developed in the annelid kingdom parallel with the development of a contractile vascular system, which possesses muscles comparable in physiological actions with the muscle of the vertebrate heart. This vascular muscle is regulated by the processes of the common ancestral cells as well as by their secretory activity. In the primitive form contractile rhythm is an intrinsic property of cardiac muscle; its nerve supply regulates its rhythm, it does not initiate it. The contractile vascular system of annelids is mainly bronchial in function. The vertebrate heart has been derived from it. In Petromyzon two distinct sets of bodies are known. One situated in the walls of the cardinal veins, the renal arteries which project into the lumen of these vessels. They are the homologues of the cortical or interrenal bodies. Another body, the chromaffin series, extends from the region of the second gill slit to the tail, running along the arteries and their branches. These correspond to the medulla. In Reptilia the relationship is closer; in birds, one body occupies the meshes of the other and in mammals the cortex surrounds the medulla. True chromaffin tissue is widely distributed, and is not confined to the adrenals. It is found also in the sympathetic paraganglia of the solar plexus, Zuckerkandl's aortic ganglia, the cardiac paraganglia, the coccygeal and carotid ganglia of Luschka, the tympanic paraganglia and chromaffin cells are found in the

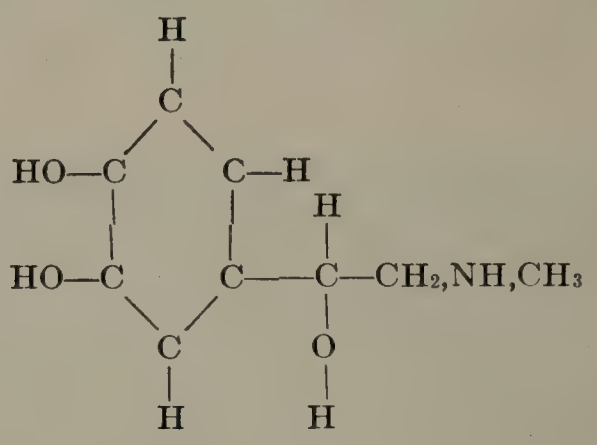
[1] Lucien et Parisot: Glandes surrénales et organes chromaffines, Paris, 1913. Gley: La Physiologie des surrénales et la sécretion d'adrénaline, Rev. de med., April, 1923. Bayer G. Nebennieren, In Hirsch, Hanbuch, 1927, **2**, 467.

[2] Orr and Rows: The Interdependence of the Sympathetic and Central Nervous Systems, Brain, 1918, **41**, 1.

[3] Gaskell, J. F.: Phil. Tr. Roy. Soc., Lond., Series B., 1914, **205**. Jour. Gen. Physiology, 1919, **2**, 73.

central gray substance from the cauda to the brain.[1] The tissues themselves are richly supplied with sympathetic nerve fibers.

All chromaffin tissues yield a true internal secretion, adrenalin, whose chemical composition is known: ortho-dioxy-phenyl-ethanol-methylamine.[2]



Its nearest relative is tyrosin, a well-known product of protein decomposition. Bloch, loc. cit. has shown that the melanin of the skin pigments is an intermediary product in the synthesis of adrenalin, and, it is probable that photic and thermic cosmic stimuli are handled by the skin pigments in the general energic complex of the human body. The chief action of adrenalin is upon the sympathetic nervous fibers increasing their reactive capacity, or sensitizing them as it were. All of the actions of the sympathetic system can be imitated by adrenalin and failure in the adrenalin supply causes failure in the proper action of the sympathetic nerves (Gaskell).

The routine function of the chromaffine tissue is to react to metabolic stimuli largely in response to desire and fear. Their emergency function, as Cannon has termed them, is to provide the necessary over-response to emotional hyperactivity—*i. e.*, to increased or diminished desire and fear—which, as their correlates love and hate, are the ultimate expressions in the symbolic sphere of what are instinctively known as useful or harmful agencies to the organism and to the race. This over- or underresponse brings about, through widespread vegetative nervous system activities, including those upon other endocrinous glands, the approximately necessary metabolic adjustment. This takes place chiefly through the regulation of the blood volume and of the organic and inorganic constituents of its plasma. Adrenalin itself is present in the plasma in proportions of 1 to 20,000,000. Notwithstanding this extreme dilution it acts upon unstriped muscle fiber and on sympathetic receptors. Adrenalin then is a typical product which demonstrates the metabolic regulation mechanisms of the vegetative nervous system. In addition to this broad function of keeping the sympathetic nerve fibers in adjustment it has many specific functions. One of the mechanisms more clearly established, but not entirely worked out, is the interaction with the pancreatic internal secretions, whereby muscle sugar utilization is partly regu-

[1] Bruni: Archivio per le Scienza Medichi, 1917, **40**, 146.

[2] First isolated by Abel H. S., 1899, **28**, 318 as epinephrine then by Takamine as adrenalin, Am. Jour. Pharm., 1901, **73**, 523. The formula previously given has been modified somewhat by Fraenkel, Die Arzneimittel synthese, 1927.

lated. Asthenia is the chief symptom which comes out when this function is disturbed.[1] The adrenals are profoundly under the influence of emotional states. One hardly needs to be reminded of the herculean efforts that the lover can put forth, or the collapse states of fear, despondency and chagrin.[2]

The numerous polyglandular syndromes which have been described cannot be discussed here in detail. Their name is legion and most are still so incompletely thought out that were it not for the many suggestive possibilities they could be dismissed with few words. Only the more pronounced forms can be taken up but the student is reminded that a great many constitutional anomalies, eccentricities and incapacities are related to excess and defective suprarenal activities, in which the medullary and cortical portions must be separately indicated in explaining the symptomatology of the endocrinopathic disturbances.

The general pathological anatomy of the suprarenals comprises congenital aplasia usually associated with varying types of monsters, unilateral absence of the suprarenal has been observed frequently (Winslow, Legg, Bramwell), simple atrophy, inflammatory and toxic atrophy, neurotic atrophy (Laignel-Lavastine) related to disease of the abdominal sympathetic plexuses, simple hypertrophy and hyperplasia, compensatory hypertrophy chiefly of the cortex from disease of one of the glands or from causes as yet undetermined, hyperplastic reaction to chronic sclerosing disorders and differentiated as nodular and miliary hyperplasias adenomata. This hyperplasia may involve both the cortex and the medulla or either alone, the adenomatous hyperplasiæ involving chiefly the cortex. Suprarenal degeneration, parenchymatous, fatty, hyaline, amyloid, calcareous. Among the vascular alterations of the adrenal active and passive congestion, thrombosis and infarcts and hemorrhage must be held in mind, and a group of inflammatory and toxic changes are known. Thus, an acute adrenalitis with a fairly definite pathology has been described, whereas a large group of chronic adrenalitides have been studied, particularly by Delamare, Husnot and Sezary. Syphilitic, tuberculous, mycotic and parasitic lesions are also known. The tumors of the suprarenal make up a final and large group. All kinds of tumors of the adrenals have been found.

Hypoadrenalemia.—The most *acute* form which is present in complete or great loss of the suprarenals is rare. Pende[3] has described six types, to which he gives the names impromptu death of suprarenal origin, pseudoperitoneal type, cholera-like or gastro-intestinal adrenalemia, apoplectiform type, meningo-encephalitic type and myocardial type. In the first form individuals suddenly die without warning,

[1] Mackenzie, G. M.: Suprarenal System and Carbohydrate Metabolism, Arch. Int. Med., 1917.

[2] See Cannon: Loc. cit.

[3] Patologia dell apparato surrenale. Milano, 1909. See Sezary: La domaine de l'insuffisance surrénale aiguë, Rev. de med., 1923, **39**, 246.

without symptoms save perhaps an epileptiform cry, or acute dyspnea or angina. Caseous degeneration of the suprarenals has been observed. The pseudoperitoneal forms resemble an inexplicable attack of acute peritonitis with death. The gastro-intestinal form behaves like an acute poisoning. The apoplectiform resembles a cerebral hemorrhage, but autopsy has shown no cerebral defect but suprarenal hemorrhage.

The cases are extremely difficult of diagnosis and are rarities. Less severe types may be met with, following acute infectious diseases.[1]

Addison's Disease.—This is a more chronic type due to more or less total involvement.[2]

As early as 1855 this disorder was first described by Thomas Addison whose outline practically covered the essential symptomatology. It is a disorder of adult life—thirty to forty years. Its chief features are a gradually developing asthenia, with arterial hypotension. There is morning nausea or vomiting, lumbar pains, an advancing yellowish pigmentation of the skin and mucous membranes, amyatrophy, depression, unwillingness to do anything, with episodic occurrence of myoclonic, tetanoid or epileptiform convulsions, with periodic palsies, confusional states, delirium, chronic paranoid ideas, coma, death. The chief lesion found is tuberculosis of the medulla of the suprarenal glands. The more complete symptom picture may be consulted in works on general medicine.

Partial Hypoadrenalemias.—These have been termed the abortive or latent types of Addison's disease. The melanoderma is absent, but the other symptoms noted are observed. Constitutional hypoadrenalemic states no doubt are very numerous and show themselves as rare and difficult forms of lowered vascular tonus, cardiac instability, muscular asthenia, visceral and ligamentous ptoses.[3] Sergent's white line is a very frequent symptom in many hypoadrenalemias. It is not invariable, however, and severe adrenal defect states may be present without the presence of the Sergent white line. (See Examination.)

Hyperadrenalemia.—A group of genital-adrenal syndromes has been isolated by a number of observers.[4] In these the cortex chiefly of the gland has been involved, either as a simple hypertrophy, an adenomatous hyperplasia, benign and malignant adenomata, or carcinomatous hypernephromata. In nearly all of these cases there has been an associated series of atrophic lesions of the internal genital organs of the female. The uterus, tubes and vagina are apt to be small, the ovaries small, usually cystic, the Graafian follicles regressive or absent, with atresia of the primitive follicles. The male analogues are not yet as sharply delimited.

The chief clinical forms which this interrelationship brings about may be formulated under a few pronounced trends, with a medley

[1] Sergent et Bernard, 1899 gave the first important monograph on these acute insufficiences of the suprarenal.

[2] Bloch, R.: Entwicklungsstörungen d. Nebennieren bei Addison'scher Erkrankung. Beit. z. p. Anat., Bd., **67**.

[3] See Sergent: Études cliniques sur l'insuffisance surrénal. Maloine.

[4] Gallais: Le syndrome génito-surrénal, 1913.

of intermediaries. The chief trends are (1) a pseudohermaphroditism, (2) suprarenal virilism, (3) menstrual and (4) obstetrical forms.

1. **A Suprarenal Pseudohermaphroditism.**—These are feminine hermaphroditic forms externally with virile secondary male sexual characters. A very early case was reported by Crecchio, in 1865, of a woman, aged fifty-two years, taken to be a man. She had a large penis-like clitoris with hypospadias, no scrotum nor testicles, a uterus with two tubes, two ovaries without a trace of corpora lutei, and an enlarged and voluminous suprarenal. She had been markedly asthenic, dying in a syncopal attack with vomiting and persistent diarrhea. This is a type case from which many variations in the individual primary and secondary sex character developments may be seen in other patients with this syndrome. The usual glandular defect is a simple cortical hypertrophy of the adrenal and the anomaly is usually embryonic and the symptoms develop slowly throughout childhood and adolescence.

2. **Suprarenal Virilism.**—In this trend the suprarenal tumor develops[1] without congenital anomalies of the genital organs. Here the secondary sex character changes are predominant. The primary sex modifications are more functional, sterility, menstrual irregularities, than microscopically anatomical. The vegetative mechanism of the ovary is nevertheless interfered with. Females are chiefly involved. The phases of development vary, but there is usually early menstrual irregularity or amenorrhea. Anorexia, nausea and vomiting are not infrequent. The adolescent girls are usually very fat, and there is much muscular hypertension and restlessness. The girls are thought to be fine athletic specimens, although irritable, nervous and at times flighty. They tend to be aggressive, domineering and often play the man role with their schoolmates, or "crushes." Vasomotor crises are frequent. A moustache is apt to develop and pigmentation of the Addisonian type may show itself. This stage gradually changes over after several years to a distinct thinning of the body and marked muscular weakness. Thoracic pains then develop and in the more marked cases an abdominal tumor. The patients are apt to be depressed, and at times even suicidal. The disease may stop at any stage before a terminal cachexia with asphyxia and convulsions. In young girls the tumor is apt to develop to a pronounced degree by eleven or fourteen years of age.

The usual tumor is a benign hypernephroma, sometimes malignant.

Another trend consists of the "infant hercules" anomalies, who at the ages of from four to eleven years develop genital hair, beards, general hypertrichosis and markedly older skeletons. Sometimes the intelligence is precocious, again they are imbeciles.

3. A *menstrual form* is characterized in older women, twenty to fifty years, by persistent and inveterate dysmenorrhea. Adiposity is apt to be marked, and often persistent, sometimes the patients become thin. Pigmentation may develop, nævi are frequent and

[1] See Sellheim: Arch. f. Frauenk., vol. **12**, No. 5, illustrations.

hypertrichosis generalized. These women often pull out the hair or peroxide their moustaches. Hyperthyroid-like symptoms are frequent, probably a true compensatory effort to make good the adrenal loss. Tumor is less frequent, but cortical hyperplasia usual. Psychogenic components play a large role in this type.

4. The *obstetrical form* is a minor variant of the former trend. The chief anomaly here is the false pregnancy syndrome which sometimes develops, or an ectopic pregnancy with its grave complications.

The chief differential diagnostic syndromes are hypophysial giantism, Fröhlich's syndrome, pineal genital anomalies, thymic hypertrophies, myxedema, hereditary trophedema, adiposis dolorosa and genito-dystrophic gerodermia.

Therapy.—The therapeutic indications in adrenal disorders are still much in the dark. Many types of diversion, of interest-provoking stimuli, of pain-reducing and worry-removing agents, physical, pharmacodynamic or psychogenic, are all more or less empirically employed for the transitory asthenias or collapse states of hypo-adrenalemias of mild grade chiefly induced by emotional disturbances.

Chronic asthenic states in unconscious phobic cases may be reached by thoroughgoing psychotherapy along psychoanalytic lines.

The opotherapeutic indications are also crystallizing out. Adrenalin itself, seems as yet to be a disappointing substance. Far better results seem to be obtained by the use of pituitary substance, by thyroid and other sympathetic stimuli than by the use of adrenalin in the asthenic. The hypertensive states are rendered worse by pituitary and thyroid. Ovarian therapy is indicated in certain of the surrenal-genital syndromes, especially in those with pronounced menstrual disturbances. Thymic hyperplasiæ and adrenal defect often seem to be related and suggest that thymic sudden death is rather a suprarenal defect and certain surgical shock deaths have a related pathology.

Certain chronic Addisonian-like types are much benefited by suprarenal in small doses, or by adrenalin in minute doses (0.004 to 0.005 gm.). In the chronic capsular cases the fresh gland itself seems to work better than adrenalin. In the present state of opotherapy each patient must be worked out separately starting with minute doses and noting the effects chiefly on the pulse-rate, blood-pressure, Sergent's white line and the asthenia. Ephedrin is coming into prominence as an adjuvant or substitute for adrenalin.

In certain hypertensive states with high blood-pressure, due to unconscious factors of pathological extroversion (impatient, busy people, always trying to do twice as much as is possible—chronic unconscious sadism) psychoanalysis is of inestimable service. In the exhaustive states of acute septic processes, acute toxemias (gas gangrene of War 1914–1918)[1] the cortex undergoes marked changes, the

[1] Goormaghitigh: Suprarenal Capsule and Infectious States, Archives de méd. exp., 1919, **28**, 277

medulla less so. The whole gland extract should be used as a stimulant in such prostrations from infections with other signs of adrenal insufficiency.

DISEASE OF THE GONADAL SYSTEMS.

Genital Syndromes.—Agenitalism, Hypergenitalism and Hypogenitalism.[1]—Genital syndromes arise from disorders in (1) the gonads and (2) the interrenal tissues of the adrenal cortex.

Of all the glands of internal secretion the gonads have best and earliest been known to possess definite control of metabolism. The ancient practice of castration called attention to this intimate relationship from the earliest times.

In the male the testis is formed of (1) true gametic cells, which develop spermatozoa, and are not known to possess any hormone activity, (2) the interstitial cells of Leydig which are probably the true cells of internal secretion.

In the female one finds (1) the Graafian follicle containing the ovum and (2) interstitial cells, between the follicles, which closely resemble those of the testis. Both interstitial cell types appear to be modified gamete cells and both are the hormone producers.

This interstitial hormone acts as a connecting link between the soma and the gonads and through this specific action, particularly marked in the male, exercises a direct and specific stimulus upon the somatic structure of the body, thus increasing growth activity, causing definite lines of development, varying in the sexes, and so affecting the whole muscle and nerve metabolism as to produce profound and far-reaching alterations.

The gonads are supplied both by parasympathetic and sympathetic fibers. The chief genital or gonadal syndromes result from aplasias or malformations, giving rise to various *hermaphroditic* syndromes; from agenitalism or hypogenitalism with *eunuchs, eunuchoids,* and their many transitional forms and, from hypergenitalism with the syndromes of *dyshyperovarianism, dyshyperdiastematy* and *chlorosis*(?). The genital like the other syndromes are usually polyglandular.

Hermaphroditism.—True hermaphrodites are probably non-existent so far as the male sex is concerned. True cases of ovotestis are extremely rare. Pseudohermaphroditism, while unusual, is nevertheless not infrequently observed. A great variety of findings are recorded. Females with enlarged clitoris, vaginal cul-de-sac, ovaries, uterus and bilateral testes. In certain patients primary and secondary sexual characters correspond, in others there are male gonads with female hair distribution, pelvis formation, fatty deposits, high voice and enlarged mammæ. It is highly probable that the adrenal interstitial cells play an important role in these various admixtures of hermaphroditic primary and secondary traits.

[1] Consult Harms: Innere Sekretion der Keimdrusen, Fischer, 1914. Tandler u. Grosz: Biologische Grundlagen d. sekundären Geschlechtscharaktere, Berlin, 1913, and the monographs mentioned, particularly Biedl, Hirsch, Handbuch. Lipschutz.

Agenitalism: Eunuchs.—The symptoms vary considerably, depending upon the age of the individual when the (usually surgical) loss of the gonads takes place. In early loss in the male (castration before puberty, destructive orchitides) the penis, prostate and seminal vesicles remain small, erotic desire fails to show itself and potency is lost. In the female a similar failure of development takes place. The girl is apt to grow tall, boyish in type, with infantile secondary characters.

Later loss, after puberty, tends to increase the size of the skeleton—a tall, thin type and a short, fat, dumpy type with broad hips, female fat distribution on the breasts, buttocks and iliac crests. The lower extremities develop disproportionately more than the upper, or *vice versa*. The head is flattened behind, the sella turcica widened, the superciliary ridge is apt to be prominent. The skin is usually smooth cool, marble-like, poor in pigment and color; the hair of the head usually thick while that of the face is absent or only downy, that of the pubes follows the female type of distribution—horizontal. Small thyroids, thymus, larynx and wide pelvis are the rule.

The average castrate is apathetic, with shambling gait, bent in his posture and a sleepy or indolent mental attitude. The face is usually fat with puffy eyelids. The voice is high and thin. Erotic desires are not always absent nor is intercourse impossible, even though the penis is apt to be small.

The female—artificial menopause—tends to grow stout and irritable, autonomic tonus is lowered, ptoses are frequent, vasomotor instability, with hot and cold flashes, with darting, jumping pains, anxiety, nervousness and flightiness develop. Alcohol, bromides and other drug habits not infrequently develop as attempts at relief of the annoying symptoms. The ovariectomy craze of the 1880–1890 periods having spent it force as a cure-all fewer of these cases are seen.

Eunuchoids.—These result from less marked disturbance in the development of the gonads. A great variation also exists here following various accidents to the testes and ovaries, inflammations, tumors, infections (tuberculosis, parotitis, gonorrhea), etc. Two trends are prominent, the tall and the fat types. The changes are those found in eunuchs, although for the most part less pronounced or monosymptomatic in their appearance. Every gradiant may be encountered, hence the difficulty in describing a very variable mosaic. Cryptorchism is a frequent complication.

The skin changes are present in both forms. It is usually thin, pale, anemic, and apt to be slightly waxy; fine lines or wrinkles develop readily, giving an appearance of old age with youth. The hair anomalies are as already discussed. Atrichia, irregularities in development, lanugo substitutions, alopecias are not infrequent. The eyelids and eye-brows are sparse. Single long hairs develop on the chin, the moustache is scanty and wiry. Sterility is the usual result in both cases. Many of these patients are superficially potent, some even hyperexcitable, but the reverse is the rule. Meritorious psychical achievements are frequently possible as seen in many writers, artists,

interior decorators, milliners, dressmakers, etc. The whole subject is so large as to defy text-book compression.

A late type of eunuchoidism following disturbances of the testes (syphilis, trauma, roentgen-ray, alcohol, gonorrhea, exposure, tuberculosis, etc.) develops somewhat similar signs.

Infantilism.—Various types have been described which have been keenly discussed. More than in any other group perhaps does the polyglandular hypothesis seem necessary to comprehend the many major and minor variations. Lasègue gave the name to the group. Lorain then described his types as: (1) Those small, graceful and finely built, (2) those that remained more or less infantile and (3) a feminine type with broad hips, small genitals, scanty facial hair, long and thin hair of the head, large breasts and prolonged primary dentition—failure of second teeth, etc. Brissaud then showed that certain of Lorain's types were myxedematous and Hertoghe took them out of the gonadal group and classed them as thyreopathies. Later students pointed out hypophysial anomalies, others pancreatic, still others implicated the spleen. Hypotheses ran riot throughout this entire field but sound relationships are slowly crystallizing out.



FIG. 140. — Eunuchoid. Hyporchismus in boy. (A. Josefson.)

Dysgenitalism. — Chlorosis. — Without committing oneself to the definite position that chlorosis is an anomaly due to defective genital hormone activity yet the evidence now seems to point in that direction. Its occurrence in girls at the time of puberty is one of the factors accentuating this relationship. Von Noorden was among the first to ascribe chlorosis to a defective secretion of the ovarian interstitial cells. In this syndrome other endocrinous glands, notably the thyroid, show alterations.

Symptoms.—The symptoms are not recorded here, as they are better found in works on general medicine and the vegetative paths involved in control of the blood-making organs and of the tonus tensions of the bloodvessels and the blood itself have not been sufficiently worked out to be stated definitely in this place. The chief factor to be emphasized here is that chlorosis is largely conditioned by an excessive plasma plethora in the bloodvessels. The bone marrow is overstimulated to create an increase of red cells, which because of the great dilution from the surplus of plasma volume are correspondingly poor in hemoglobin.

It may be assumed provisionally that this plasma retention is made possible by diminished transudability of the vascular walls (sympathicotonic) and is an opposing picture to that which is seen in the disordered transudability in the various types of angioneurotic edema which have been discussed (vagotonic). (See Blood Syndromes, p. 262.)



FIG. 141.—Hypoövarismus. (Josefson, Deut. Ztschr. f. Nervenh., 1910.)

Dysgonadal Syndromes.—*Hyperovarian Signs.*—Habit of precocious sexuality. Precocious intelligence active, bright, gay and jolly. Precocious puberty. Menorrhagia, metrorrhagias, amenorrhea of local lesion. Congestive dysmenorrheas, nervous dysmenorrheas. Harmony of form, good constitution. Terminal anemic paleness. Thyroid normal, with inconstancy. Marked fecundity, menopause retarded. Nervous type. Constipation not marked.

Hypoövarian Signs.—Retarded habitus, generalized or localized. Various types of infantilism, morons. Late menstruation. Habitual amenorrhea, metrorrhagia. Nervous dysmenorrhea from flexion or other malformation. Pale, puffy, pseudomyxedematous, adiposity, acrocyanosis, cold extremities. Thyroid enlarged, mild exophthalmic signs, infecundity more often, early menopause. Nervous; constipation marked.

Gonadal Inferiority Syndromes.—Only a word can be said in passing concerning the many physical and mental variations related to constitutional or acquired gonadal inferiorities. The chief somatic varieties have been touched upon. The psychical varieties found in enormous numbers would require a special volume. Man's chief obsession is his sexual potency. Nature's great aim is to develop adult psychosexual individuals. Practically all mankind is struggling

along this pathway and halting at various levels of psychosexual evolution. The chief criteria to determine the stage of this development are found in the unconscious of the individual. The psychoanalytic technic alone can determine this. All conscious criteria are usually camouflaged substitution products.

Jelliffe, loc. cit., has endeavored to show that in the study of unconscious processes one may determine, much in the same manner as the paleontologist determines a geological horizon, the stage of psychosexual evolution which the individual in question has reached. His dynamic strivings bear a direct relationship to this stage of development, and his diseases, speaking in general, develop in definite relationships to his dynamic strivings.

Thus he would construct a dynamic pathology in accordance with this chief aim of Nature, *i. e.*, the evolution of the phylum. Man's chief unconscious endeavor is to remain in the herd. If defective, according to this concept, he either flees from the herd, *i. e.*, develops what is crudely conceived of as a psychosis, or remaining more or less faithful to herd concepts, sacrifices, through bodily disease, that functional group, *i. e.*, organ activity, which most nearly symbolizes his chief unconscious inferiority. Thus he would say, *à propos* of Mott's findings of defective gonadal structures in the disorders known as dementia precox, that the disease is not due to the defective gonadal structures, but that the faulty psychosexual evolution in the individual causes him to withdraw interest from the world of reality and that the gonadal degenerative changes are an index of this failure to participate in the universal goal of mankind, namely the capacity for productive, creative, activity, *i. e.*, maintenance of phyletically valuable functions. The behavior symptoms of the disease are the various substitution products teleologically evolved to maintain his false sense of potency.[1]

Treatment.—Various types of testicular and ovarian therapy have been used. In certain hypo-ovarian cases moderate doses of dried ovary (0.10–0.20 gm.) twice a day, over a fairly prolonged period have been reported to be of some service. The glycerin extract hypodermically has been abandoned. Corpus luteum preparations are now being extensively employed with results as yet not readily interpreted. Combinations with thyroid, pituitary, and suprarenal extracts are recommended. Thus in simple ovarian insufficiency ovarian cachets may be employed solely as substitutive. For the headaches and the hot flashes pituitary or adrenalin preparations may be used as regulatory or as hormostimulatory. Suprarenal therapy is contraindicated in the hypertensive states, whereas in myasthenic, constipated, and wakeful nervous patients with lumbar pains, headaches, digestive disturbances and other dysovarian signs it has proved serviceable.

Opotherapy for gonadal syndromes is still in an experimental period of development. Personal experiences point to it as largely sug-

[1] Kempf: Psychopathology has much material supporting this attitude, to which his work also in general subscribes, St. Louis, 1920.

gestive, but in certain carefully analyzed cases excellent results have seemed to depend directly upon the opotherapy. The indiscriminate use of testicular extracts (spermin) has not added any permanent gain in therapy. In obstetrical and gynecological work the action of pituitrin upon the unstriped fibers of the uterus and bladder is very marked and can be made serviceable.



FIG. 142.—Hyporchismus. Man. Eunuchoid. (A. Josefson.)

Ovarian and thyroid extracts may be added with advantage to iron and arsenic in the treatment of chlorosis.

There is a frequent type of compensatory hyperadrenalema following the menopause which is often very advantageously handled by moderate doses of ovarian substance. This hyperadrenalemia is often a producer of marked arteriosclerotic states, atheroma, headaches, angina and even cerebral hemorrhage. Its unconscious psychic accompaniments are greatly in need of careful investigations.[1]

Status Thymolymphaticus.—The thymus has very close relationships to the gonads, and it has been thought that the thymus and testes are reciprocally acting organs. This does not seem to be true for the thymus and the ovaries.[2]

Certain individuals have excess of thymus lymph tissue throughout

[1] Maranon: The Critical Age, Madrid, 1919.
[2] Thomas: Physiologie der Thymus. In Hirsch Handbuch, 1927, 2, 423.

the body. In recent years the researches of Bartel,[1] Wiesel and A. Paltauf have shown that this condition is very frequent. Indeed it would seem that persistent thymic states are the rule rather than the exception. Notwithstanding the fact that a pathological diagnosis postmortem is easily arrived at, the clinical diagnosis during life presents many difficulties. This latter is largely due to the circumstance that the diseased organs are difficult to examine and, furthermore, the signs of defective development which result from the condition are often very slight.



FIG. 143.—Pubertas precox (hyperorchismus) eleven-year-old boy. (A. Josefson.)

The recognition of status thymolymphaticus often requires exhaustive chemical, physical, *x*-ray, and other forms of examination. Such recognition is highly important, however, since these individuals, if they may be grouped, are prone to react very markedly to anesthetics, bodily shocks, infectious diseases, and to drugs, particularly salvarsan, sera, cocaine and mercury. A large medley of conditions accompanying and partly due to status thymolymphaticus, have been described. These may be briefly summarized as follows:

1. *General Signs.*—As a rule, the length of the body exceeds the stretch between extended finger-tips. The length of the upper part of the body and of the extremities is above the average. The arrangement of fatty tissue tends to make the male resemble the female type, and *vice versa*. The mental status is inclined toward the infantile. Alcohol resistance is very slight.

2. *Face.*—The under jaw and the mastoid processes are underdeveloped and the former results in faulty bite, anomalies of dentition by displacement and by crowding. The palatal arch is high; the

[1] Status thymicolymphaticus u. Status Hypoplasticus, Deuticke, 1912. Symmers, Am. Jour. Med. Sci., 1918, vol. **156**. Emerson, Tr. Int. Cong. Med., 1914. Ewing: Jour. Am. Med. Assn., November 9, 1918, vol. **71**.

tonsils and tongue papillæ are increased in size. The epiglottis is inclined to be infantile in type. Epicanthus, eccentric pupils, irregularly pigmented irides, adherent ear lobules and narrow external auditory meatus may be present. "Peaches and cream" complexion.

3. *Neck.*—The thyroid, cervical, and other glands are enlarged.

4. *Skeleton.*—The thorax is long and narrow. Cervical floating ribs are present. Compensatory lordosis of the spine is lacking. The scapulæ are wing-shaped. The pelvis develops heterosexually; the sacrum is small, the pubis high. Hyperdactyly, flat-foot, and hyperextension of the elbows may be looked for.

5. *Hair.*—Axillary and pubic hair are diminished; the extremities may be hairy.

6 The *thymus* is enlarged, the breasts resemble those of the opposite sex; polymastia may be observed. The aorta is narrow, the heart small, the blood-pressure low. Palpitation is frequent and there is cardiac dilatation with weakness.

7. In the *abdomen* ptoses are frequent. The jugulo-pubic distance is increased, the abdominal circumference diminished. The spleen is enlarged, the kidneys prolapsed. There is a tendency to orthostatic albuminuria and to alimentary glycosuria.

8. The *blood* picture shows a neutropenia, lymphocytosis, and eosinophilia.

9. The *genital* anomalies are in the nature of cryptorchism, hypoplasia, disturbances of menstruation and secondary sexual characters of the opposite sex.

10. There is a marked disposition to other disease and usually a tendency to an increase in the severity of the disorder. Thus, tuberculosis shows more often in other organs than the lungs; infectious diseases of childhood are severe; there is a tendency to tetany, gliomata, syringoses, hydrocephalus, tabes, paresis, myasthenia. Diabetes, excessive fat and gout occur. Pernicious anemia, leukemia and chlorosis, exophthalmic goiter, Addison's disease, osteomalacia, nephritis, eclampsia, asthma, infantile emphysema, eczema, hemangiomata, appendicitis and tumor formation are among other accompanying disorders.

Observation of many cases of status thymolymphaticus shows that little weight is to be given to the occurrence of isolated symptoms. The diagnosis consists in the accumulation of the anomalies. The differences in body dimensions are of universal importance, whereas the increase in the tongue follicles and the infantile character of the epiglottis and its frequent omega shape, are more characteristic. Genital hypoplasias are frequently associated with eosinophilia and lymphocytosis is to be expected.

From the standpoint of endocrinology the interrelationships between thymus loss, deficiency, persistence and hyperactivity and the influence on other endocrinous organs is of great interest. As yet there are thousands of observations but they tend to lose themselves in a maze of confusion when one would attempt to outline definite syndromes.

Timme has outlined a thymus, adrenal, pituitary combination,[1] which is frequently observed and readily recognized. The main features are fatigability, low blood-pressure, headache and growth anomalies. The syndrome starts off before puberty with thymic persistence with growth of the long bones and many of the characters of the status hypoplasticus of Bartels, here partly enumerated. The second stage begins at puberty. The myasthenia is more pronounced. The sella is usually small and when an attempt at pituitary compensation is made by hypertrophy headaches appear, especially if the sella is circumscribed. This hyperfunction of the pituitary, if possible, provokes further bony growth and giantism or acromegalic features may develop and the headache becomes definite. Erosion of the clinoids now may begin. In from three to ten years the complete constitutional disorder is developed. With large sella turcica, acromegalic features, marked fatigability, headaches gone, but mental defects and a tendency to convulsive seizures. Intercurrent disease usually causes an early death.

There is a marked antagonism between the thymus and the thyroid gland.[2] Feeding thymus causes atrophy of the thyroid and parathyroid[3] and hyperfunction of the thyroid causes atrophy of the thymus. Uhlenhuth has fed thymus to amblystoma larvæ and produced typical tetany cramps, the cause for which he interprets as a specific toxin, but which we believe is due to parathyroid atrophy, from thymic hyperactivity, although these larvæ have no well-defined parathyroids. Dustin is disposed to see in the thymus a regulatory structuralization for nucleoproteid synthesis but the precise mechanism is not certain. In certain vagotonic exophthalmic goiter cases, there is often present a persisted hyperactive thymus, with hypo-adrenalemia.[4] These are presumably those dangerous thyroid cases in which sudden death results from operation.

The whole problem of thymus feeding is yet experimental. Certain cases of myasthenia gravis have been helped, others not.

In fact what is *not* known about the functions of the thymus far outweighs what *is* known. Persistent thymus is a much more frequent situation than has been heretofore recognized.

DISEASES OF THE PANCREAS.

Pancreatic Syndromes.—The chief activity of the pancreas is subserved through an assimilatory hormone, insulin, first solated by Banting, Best and Macleod.[5] In mild grades of pancreatic insufficiency disturbances of carbohydrate metabolism appear only when only when great demands are made upon the glycogenic function of the

[1] Timme, Endocrinology, July–September, 1918.
[2] Dustin et Zunz: Jour. de Phys. et. d. Path., 1918.
[3] Uhlenhuth: Jour. Gen. Physiol., 1918, No. 1, vol. **1**.
[4] Rose: Le thymus et la maladie de Basedow, Sem. Méd., January 21, 1914.
[5] Jour. Lab. Clin. Med. 1922, **7**, 251; Am. Jour. Physiol., 1922, **50**, 479.

liver through excessive alimentary carbohydrate intake. In graver disturbances in addition to the modification of anabolism a high grade of catabolic destruction takes place with a failure to form higher and lower fatty acids (ketonuria).

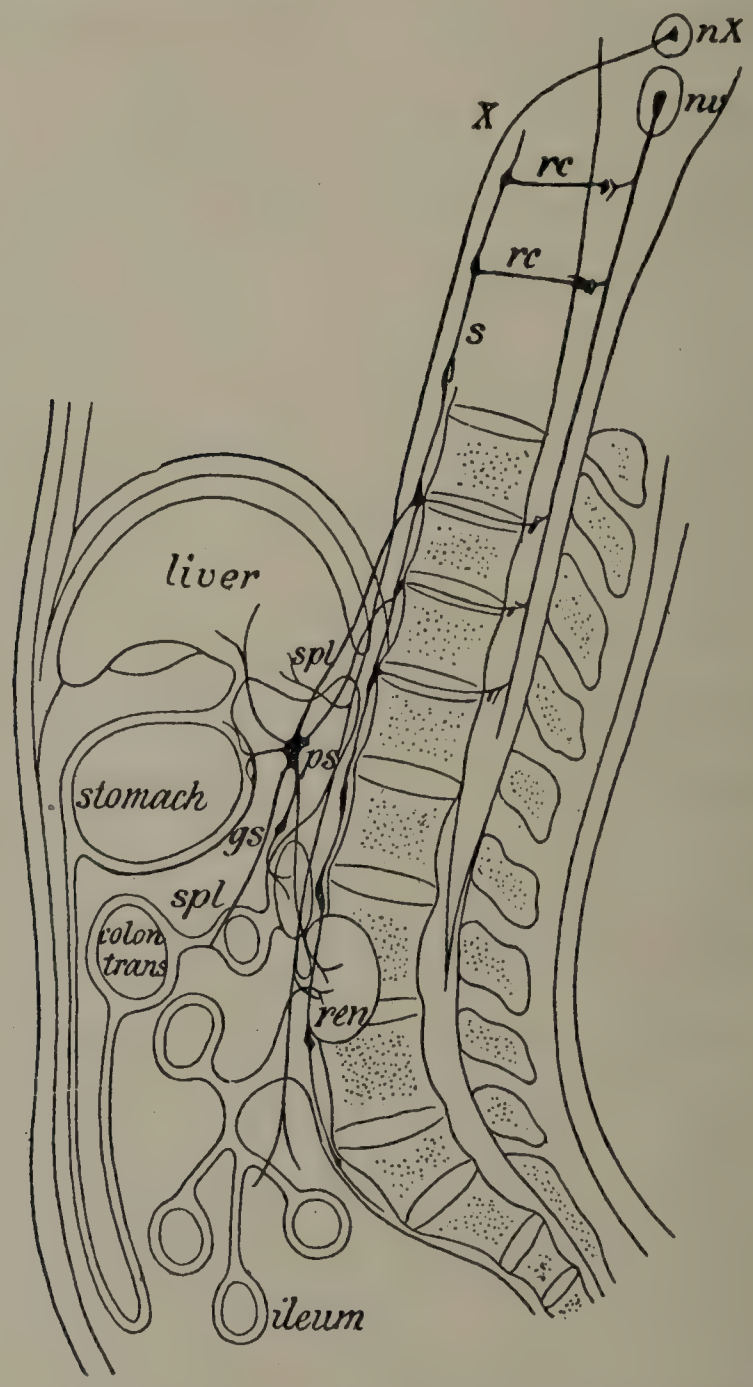


FIG. 144.—Scheme of innervation of the liver, spleen, and kidney. *nX*, nucleus of the vagus; *X*, vagus; *nv*, vasomotor nucleus in medulla; *s*, sympathetic; *rc*, rami communicans; *spl*, splanchnic nerve; *ps*, solar plexus; *gs*, semilunar ganglion; *spl*, spleen. (Bechterew.)

Pancreatic syndromes occur as a result of gross anatomical disorder, acute pancreatic hemorrhage, and chronic pancreatitis, syphilitic pancreatitis, etc.; all of which are discussed fully in works on internal medicine. Those of interest here, however, are diabetes mellitus (true diabetes), and pancreatic infantilism, both closely related to disorder of the glandular tissues of the pancreas—its internal secretory part. Although the pancreas seems primarily a digestive gland it also produces an internal secretion in the islands of Langerhans which exerts a definite control over the mobilization of sugar. There is a highly complicated balanced relation with the thyroid, hypophysial and suprarenal secretions which tend to facilitate the use of sugar as

an energizing material by the muscles. This mobilization may be considered to be made effective by the terminals of the vegetative nervous system in the liver cells; just how is not yet known.[1]

This is not the place to look for dogmatic statements relative to *diabetes mellitus*. From the extensive studies of Allen,[2] Joslin, Petrén and numerous others it may be assumed that for practical purposes a true diabetes means a definite disturbance of pancreatic hormone activity. The islands of Langerhans are involved, and in severe cases these are extensively involved. What causes the involvement is not so clear, nor can the complicated problem be thoroughly discussed here. Only one factor can be stressed and that is that rarely any isolated disease of a bodily organ, especially an endocrine organ, is adequately comprehended in terms of that organ alone. Other organs of the body are usually involved. It may be assumed that a specific lytic toxin may cause a destruction of the islands. If so, what it is, is still unrevealed? Acute inflammation, adenomatous degeneration, or other destructive acute or chronic (pancreatitis) change in the islands requires explanation. Humoral explanations are valid only as partial explanations for certain types of degeneration. But any summary of the problems which leaves out the vegetative nervous system must necessarily be incomplete, since neglect of the chief mechanisms of metabolic integration will result in partial explanations only.

The studies of Brugsch, Dresel and Lewy show that definite neuro-chemical-anatomical pathways exist. Their cortical synapses are not yet known but the neuron chain from the mesencephalon to the pancreas and return has been experimentally demonstrated.[3] Thus the neurogenic control of certain factors in carbohydrate metabolism is brought nearer to a real dynamic comprehension. The Claude Bernard "piquere" is at last cleared up. There is no "diabetic center." There are neural pathways which integrate certain pancreatic functions. While it is not thus assumed that *diabetes mellitus* is *primarily* a neurological problem, the belief here emphasized is that if the neural integrative factors are neglected, a true dynamic understanding of this very important syndrome is impossible for all cases. The intensive studies of epidemic encephalitis following on von Economo's important contributions in 1917 have shown a number of hyperglycemias resulting from involvement of mesencephalic structures. The studies of Lewy, loc. cit., help to bring these into line. Hyperglycemias are also known to occur in certain striatum syndromes (Wilson's disease), paralysis agitans. These findings emphasize the need for wider generalizations which involve the vegetative nervous integrative factors. Furthermore, it may be added that psycho-analytic study of diabetes cases (Jelliffe) shows that specific defects of unconscious psychosexual evolution factors have been found. These

[1] Eiger: Zeit. f. Biologie., vol. **66**, p. 229.

[2] Allen, F. M.: See Important Studies in Journal of Metabolic Research, 1922, **1**, Nos. 1, 2, 3, 4, for complete details as to the *pancreatic* changes.

[3] Brugsch, Dresel, Lewy: See Section on Vegetative Nervous System, Bayer in Hirsch Handbuch, 1927, vol. **2**.

also must be more adequately comprehended before the entire picture of diabetes mellitus can be adequately presented. Thus the hyperglycemias which are frequently seen in many cases of the severe psychoneuroses,[1] manic depressive psychoses, etc., must be correlated with the so-called organic cases. Unconscious homoerotic factors are prevalent.

Pancreatic infantilism shows polyglandular disturbances through arrested bodily growth and arrested sexual development. Vagotonic symptoms such as excessive diarrhea and flatulent distention are also present.

[1] Schwab, S.: Tr. Am. Neur. Assn., 1922.

PART II.

THE SENSORIMOTOR SYSTEMS.

THE NEUROLOGY OF SENSATION AND MOTION.

INTRODUCTION.

The organism as a whole can be conceived as an intricate pattern of tendencies; the partial tendencies of the several parts or organs and the larger tendencies of the whole biological unit. The partial tendencies constitute the field of physiology—the function of the various organs. The field of the tendencies of the whole organism is the domain of psychology. These tendencies, be they partial or total are states of disequilibrium which tend to develop mechanisms for restoring the equilibrium or neutralizing the stimulus of the tendency. Repeated tendencies of the same character, each tending to the production of the same mechanism form channels of energy discharge which become progressively more stable and definite. The reaction becomes fixed, stereotyped. This is the process of the structuralization of function of which the reflex is the type of functional response at the sensorimotor level.[1]

The various correlating functions of the organism which have been developed through the centuries thus come to be laid down in structures and it is of such that the so-called central nervous system and its peripheral ramifications consist. Its reflex types of response are the types at the sensorimotor level as that term is used in this book. Definite nervous pathways, therefore, correspond to functions which have become so thoroughly organized as to be laid down in permanent pathways. The relation between cardiac and respiratory rate has become so thoroughly established as a necessity that nothing is left to chance; a permanent interconnection has been so definitely structuralized that it can be relied upon always, under all conditions, to bring about the required, definite result.

In this process of the structuralization of function there must of necessity remain an unorganized residual which represents the organism's capacity for adaptation to the unusual and the infrequent. This unorganized residual is represented at the psychological level by all those, as yet uncrystallized tendencies, those unrealized hopes and ambitions which mean that the individual has not as yet become stabilized beyond recall in his habits of thought and action. At the

[1] von Uexküll: Theoretical Biology, New York, 1926.

organic (so-called) end these are represented by those physico-chemica mechanisms served by the endocrine glands and their hormones (probably the specific substances of all the organs) and that oldest portion of the nervous system, the vegetative, which have not yet become crystallized into definite forms of response. These reactions are general in character, acquisitive (love), avertive (hate) but which as yet have not acquired specific avenues of discharge which are capable of satisfactorily expressing the higher tendencies of the organism as a whole, that is at the psychological level.

The cerebrospinal pathways represent those pathways of energy discharge which have been laid down in structure and represent those more specific, organic, physiological avenues of integration which meet the demands of a greater specificity. The more generic craving, such as hunger, has to develop a mechanism of response which has as its function those specific activities calculated to acquire food: fear and hate have to develop those specific types of response which are calculated in these higher types of organisms with a cerebrospinal axis to produce those niceties of correlation which will lead to a successful flight or fight, etc., all of which activities have to be organized to produce effective responses in accord with the demands of the organism at its stage in evolution. For example the acquirement of food or the reaction of flight are very different in a hydroid or a spider respectively, and very different in those animals from the like demands upon a mammal or upon man. These elements of structure might be said to be on their way to a more stabilized structuralization but nevertheless a sufficient residual of as yet dynamically fluid mechanisms remains for the purpose of dealing with new problems.

The relations of these two parts of the nervous system, however, cannot be adequately understood if the more recent cerebrospinal system is simply conceived as overlying the phylogenetically older vegetative system. The dynamic factors which have produced the differentiation must, because of their very nature, be still active so that when demands are made upon the organism which are beyond the capacity of its definite structure they may spill over and flood the lowlands of the less definitely organized portion of both the vegetative and the cerebrospinal apparatuses with a fair chance of ultimately channelling their way to a reasonably efficient outlet in action. Structurally this interrelation is evidently shown in the intimate admixture of unmyelinated, probably vegetative nerve fibers, with the myelinated fibers of the cerebrospinal system and the double innervation of the voluntary musculature by both sets of fibers as well as the probable double structure of voluntary muscle itself. The sacroplasmatic substance is clearly analogous to unstriped muscle and there is evidence that it is innervated by the vegetative nerve fibers, while the anisotropic disk system is innervated by the cerebrospinal myelinated fibers. The organism is thus constituted to respond both in the more definitely reflex way of the cerebrospinal structures and in the more diffuse way of the vegetative, physico-chemical structuralizations.

CHAPTER IV.

CRANIAL NERVES.

DISEASES OF THE OLFACTORY SYSTEM.[1]

Olfactory.—In man the olfactory apparatus has seemingly lost much of the dominance it possessed in many lower vertebrates. Other sensory zones, notably those for the eye and ear and language have taken the lead in the program of evolution and have left smell, important though it be, in the vanguard.

In the primitive vertebrate[2] the olfactory areas in the cerebral hemisphere, and the visual and general sensory nuclei in the thalamus and the midbrain, are linked by means of numerous tracts to the hypothalamus, which at first is a relatively large part of the brain. In the pursuit of food, as well as in many another activity of the animal, this large hypothalamic apparatus is thrown into action as soon as the sense of smell excites a response. By means of numerous descending tracts the hypothalamus exerts a widespread influence upon a variety of visceral and vasomotor functions, some of which stir into activity the glands and other structures directly concerned with the processes of swallowing and digestion, as well as other mechanisms which enable the creature to devote the whole energy of its body to the immediate and vital purpose of the pursuit. And when the consummation of the reaction is attained, and the buccal mucous membrane is itself stimulated, ascending tracts carry to the hypothalamus the effects of this excitation of the taste organs, and through it stir up even more potent effects in the digestive and other visceral systems.

As Elliott Smith says, it is not "a mere coincidence that the pituitary body is developed from a combination of buccal epithelium and hypothalamus, and is an organ which excites (no doubt by very different means) profound effects in the visceral systems, in some respects analogous to that obtained by the neural bucco-hypothalamic connection.

Thus the hypothalamus represents a mechanism whereby the instruments concerned with the *phenomena of consciousness* can influence the visceral functions of the body, and express those manifold emotional effects which play so obtrusive a part in many experiences that are strongly charged with affective tone.

The cerebral hemisphere was derived from and built upon what

[1] Winkler, C.: Manuel de Neurologie, Chap. 1. Le Système du Nerf Olfactif., 1918, p. 10.

[2] Smith, G. Elliott: Significance of the Cerebral Cortex. Croonian Lectures II, III: British Med. Journ., June 28, 1919.

in the primitive vertebrate was chiefly the receptive instrument for smell impressions, and the olfactory tracts, unlike all the other sensory paths, lead directly to the cerebral cortex without the intermediation of the thalamus (See later discussion.) The primitive hemisphere, therefore, must have been the instrument whereby the affective aspect of smell was appreciated—that is, it performed the functions with which the thalamus is concerned for the other senses.

"Guided by the sense of smell, the behavior of the primitive vertebrate is brought under the influence of other 'distance-receptors,' which secondarily acquire an affective significance or meaning by being brought into intimate relationship with the dominant sense. Specializations of structure and function then develop by the cultivation of these other senses, often at the expense of the sense of smell, and animals like the teleostean fishes are evolved which are guided mainly by vision.

"The new mode of life on land demanded not only the development of entirely new modes of locomotion, but also a transformation of the distance receptors of the hind-brain, as well as the organs of smell." Progressive telencephalization is the term given by Tilney to this phyletic process. "This determines a high development of the midbrain and a retrogression of the cerebral hemisphere. Incidentally such creatures lose the possibility of further advancement in the direction of the higher vertebrates, not merely because they have become specialists, but also because the adaptability of the cerebral hemisphere upon which real progress depends has been sacrificed in the acquisition of visual efficiency. For the organ that was evolved to detect slight movements and sound in the water became the organ of hearing, and the olfactory mucosa, which was specialized to respond to very delicate forms of chemical stimulation, now became much more highly sensitive and adapted to appreciate the more subtle effects of minute air-borne particles of odoriferous substances.

"The heightened powers of smell, the new powers of hearing, and the necessity for acquiring new methods of locomotion, provided a powerful stimulus to the cerebral hemisphere; and there are reasons for supposing that the earliest Tetrapoda were provided with a brain with a well-developed cerebral cortex. But their surviving amphibian descendants only in part emancipated themselves from the aquatic mode of life, and were satisfied with slow-going, clumsy movements of a simple character. As the result their cerebral cortex underwent a progress of retrogression, and the amphibia dropped out of the race for vertebrate supremacy.

"But one group of proto-amphibians was more progressive. They acquired the power of more rapid movement and of more varied response to the influence of their environment. The sensitivity of touch, smell, vision, and hearing became heightened and the influence of these senses so intimately integrated by evolutionary changes in the cerebral hemisphere as to make possible a much more complex and intelligent behavior. The increasing significance of vision, hearing,

and touch, found expression in the sudden growth of the thalamus and the development of larger tracts of fibers ascending from the thalamic nuclei into the lateral edge of the pallium, which then suddenly expanded and bulged into the ventricle. The great mass of low-grade pallium, which may be called 'hypopallium,' thus formed represents the first relatively independent representation in the cortex of the senses of vision, hearing, and touch, at first chiefly of the trigeminal nerve. Then for the first time the cerebral cortex became an instrument whereby these senses played a combined part in controlling behavior and added their quota to the discriminative side of the animal's consciousness. The reptile thus came to acquire the power of learning by experience from other senses than smell and of correlating the information acquired by touch, vision, and hearing, to perform acts of discrimination in which these several senses took a definite part. But this hypopallium was an imperfect instrument; and when it became superseded by the development of the higher mechanism of the neopallium, it (the hypopallium) became converted into [the greater part of] the nucleus caudatus, the putamen and the claustrum."



FIG. 145.—Extent of true olfactory receptors on the mucous membrane. (v. Brunn.)

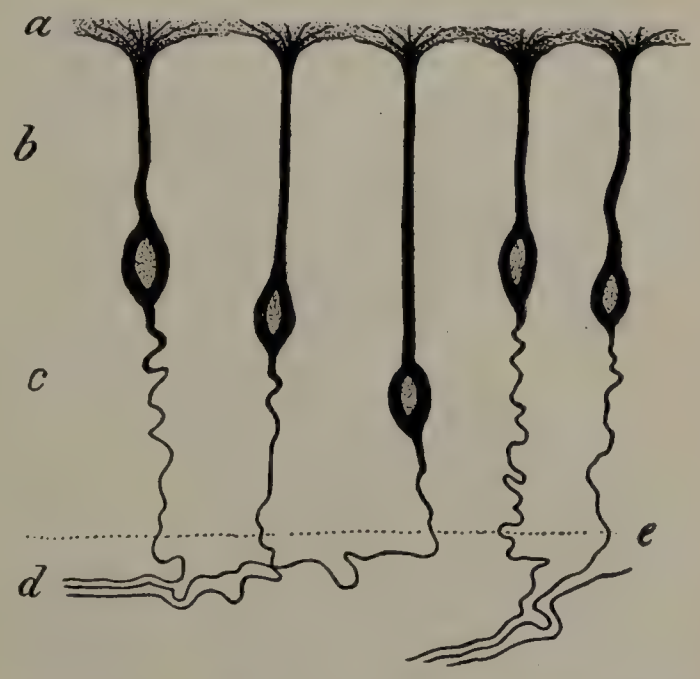


FIG. 146.—Scheme of bipolar receptors of the olfactory. (After R. Cajal.)

The receptors for smell are located in a limited portion of the Schneiderian mucous membranes. They react to a vast variety of minute stimuli, being for some substances from 1 to 20,000 per cent. more sensitive than the receptors for taste. One part in 8,000,000 of musk is capable of being consciously detected. There is marked variability in individual threshold capacity as determined by Zwaardemaker's olfactometer.[1] Moreover there are undoubtedly many smell systems with receptors histologically alike but capturing different

[1] Zwaardemaker: Neur. Ztbl., 1893; Parker: Organs of Taste and Smell, 1922. See Henning, Hans: Der Geruch., 1924, 2d Edit., for a most fundamental study of the sense of smell in all of its aspects, also recent Handbuch der Physiologie der niederen Sinne. Skramlik, 1926, and Handbuch d. norm. u. path. Physiologie, Bethe, et al Receptionsorgane, 1926. Fröse, Die rhinogenen Aktionsströme im vegetativen Nervensystem, Berlin, 1927.

olfactory energy modalities.[1] Thus many smell reactions are practically non-apprehensible to consciousness, yet minimal unconscious stimuli nevertheless may produce widespread reactions. Horse and cat asthma, hay fever, etc., may thus have unknown etiological factors from unconscious odor associations, provoked by unknown chemical substances of unconscious cellular memory activities.



FIG. 147.—Illustration of first and second neurons of the olfactorius. The first synapse takes place in the glomeruli. (Edinger.)

Changes in that portion of the membrane, such as occur in any acute inflammatory disease, coryza, influenza, diphtheria, etc., cause diminution or loss of ability to smell.[2] Albinism is often associated with smell anomalies. Chronic inflammatory processes, often accompanied by fetid odors, polyps, frontal or maxillary sinusitis, lead poisoning, usually bring about unilateral or bilateral loss of smell. Most of the causes for this mostly peripheral loss of smell may be estimated by direct inspection. Certain drugs acting locally, cocaine, etc., influence smell.

From the receptors, unmyelinated fibers traverse the cribiform plate and enter the olfactory glomeruli where they form the primary olfactory ganglia. From here axones pass to the mitral cells where a second synapse is made, from which emerges the olfactory tract, the secondary projection system of the olfactory system. The tertiary connections of the olfactory tract are chiefly as follows: (*a*) With the tuberculum olfactorium and trigonum olfactorium (anterior olfactory lobe); (*b*) by way of the striae lateralis to the gyrus semilunaris and gyrus ambiens (posterior olfactory lobe); (*c*) anterior pyriform lobe. These make up the first olfactory cortical areas which in man is much reduced.[3]

Lesions in and about the cribriform plate from fractures, meningitis, syphilis, pressure of frontal tumor, may determine a diminution or loss of smell; possibly lesions in this portion of the olfactory pathway may cause hallucinatory odors, but this is still debatable. Certain tumors lying upon the orbital plate of the sphenoid and compressing the lobus

[1] Etable, C.: Smell Systems and Histologically Differentiated Plural Olfactory Energy Systems. Travaux de Lab. Ramon y Cajal, 1924, **22**, 329.

[2] Collet: Rapport sur l'anosmie, Arch. Int. de Laryng., 1899, **12.**

[3] For more precise details see Winkler, loc. cit. and study by Etable, loc. cit.

olfactorius have seemed to give rise to unilateral and bilateral hallucinations of smell. (See Fig. 147).

Disease of, or pressure upon, the olfactory tract in its peripheral, hypothalamic, or cortical portions results in either diminution (hyposmia) or loss (anosmia) of smell; hallucinations, illusions (parosmia, ismosmia or cacosmia), or hyperesthesiæ, causing excessive sneezing. Odor influences taste directly and by association involves the entire

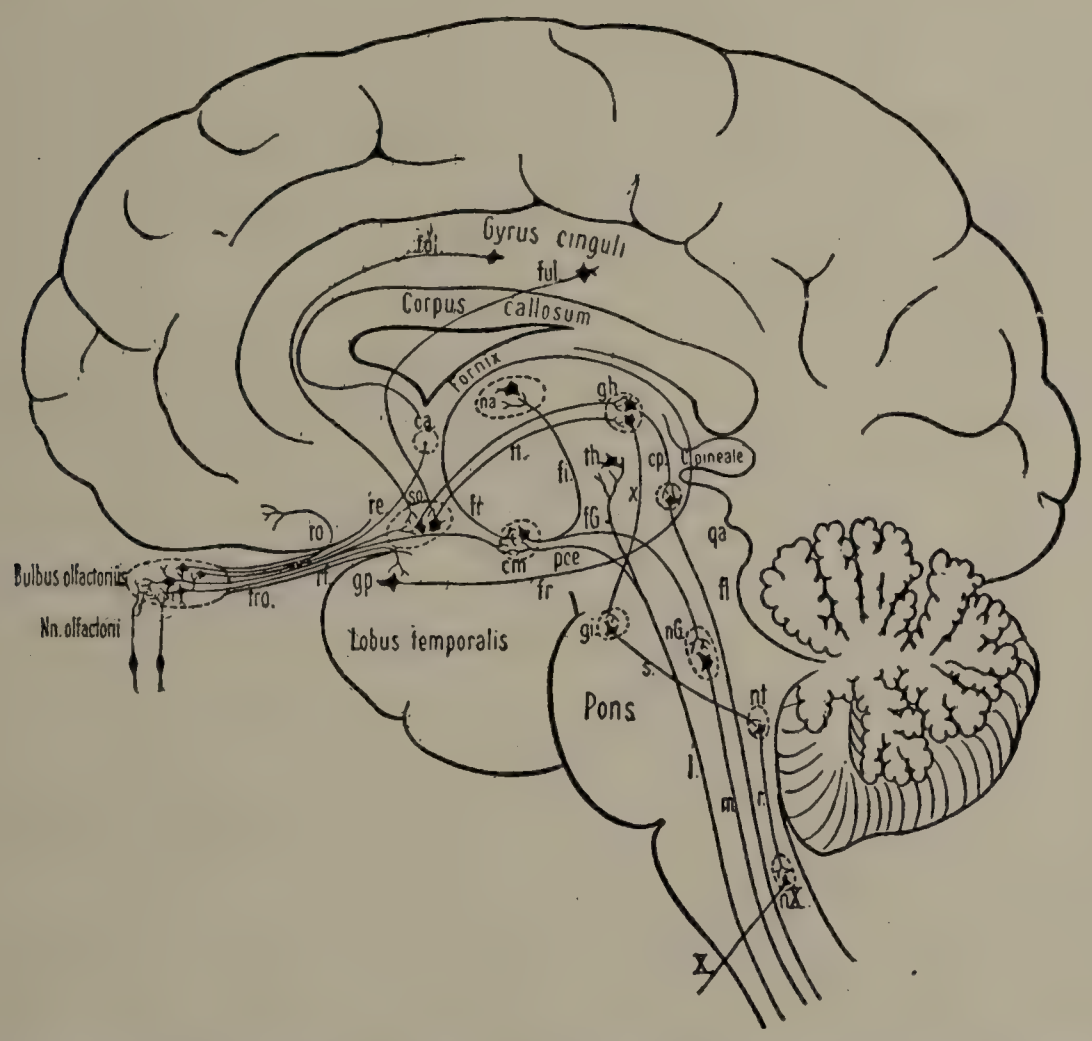


FIG. 148.—Scheme of olfactory paths. *X*, vagus root fibers; *ca*, anterior commissure; *cm*, mammillary body; *cp*, fibers from nucleus habenulæ to posterior commissure; *fG*, tract from mammillary body to Gudden's nucleus; *fi*, fasciculus mammillo-thalamicus; *fl*, fasciculus long. medianus; *fr*, fornix; *ful*, fibers of fornix; *gli*, nucleus habenulæ; *gi*, interpeduncular ganglion; *gp*, gyrus pyriformis; *l*, median lemniscus; *m*, fibers from Gudden's nucleus to substantia reticularis; *na*, anterior thalamic nucleus; *nG*, Gudden's nucleus; *nt*, tegmental nucleus; *nX*, vagus motor nucleus; *peE*, ped. corp. mammillaris from fillet; *qa*, quadrigemina; *r*, fibers from n-tegmenti to cranial nerve nuclei; *re*, radix lateralis tractus olfactorii; *rf*, fibers of olfactory tract to trigonum olfactorii; *ro*, median olfactory tract root; *s*, fibers from interpeduncular ganglion to tegmental nuclei; *so*, olfactory trigone; *th*, optic thalamus; *tro*, olfactory tract; *tt*, tenia thalami; *x*, fasciculus retroflex. (Bechterew.) (Compare this diagram with that of Winkler, Manual de Neurologie, vol. i, p. 29, Fig. 15.) and the studies of Etable, loc. cit.

vegetative nervous system. The different parts of the olfactory pathways need to be taken into consideration. The study of olfactory hallucinations, particularly in certain psychoses, and in certain tumors in or about the frontal lobes, and the uncinate gyrus, renders this of value.

Association and commissural connections of the primary olfactory cortex are made through the anterior commissure. Ventral and medial

olfactory radiations also arise from this cortex. Association fibers pass to a secondary olfactory cortex in the limbic lobes. The medial radiations are numerous. They pass to the lamina terminalis, to the frontal Broca convolutions, to the nucleus amygdalus and to the cornu ammonis by way of the fornix, the largest of the bundles. Thus this secondary cortical association system comprises the tuber cinereum, the infundibular portion of the third ventricle, the lamina terminalis, the subcallosal gyrus, the zone of Broca, the indusium, the fascia dentata, the cornu ammonis and the amygdalus. (See Fig. 148 and Winkler.)

Clinical correlations with disorder of these portions of the olfactory pathways are still to be made. Certain over affective reactions to odors, disgusts, nausea, even vomitings from odors need to be more carefully sifted in this connection, especially in relation to brain tumor localizations. One personally observed patient with chordoma of the base complained of the smell of "burning material;" another with a frontal fibroma was anosmic to test and yet had a constant hallucinatory projection of smelling disagreeable things, feces, etc. Such phenomena should not be viewed as whims or fancies of hysterical patients.

Lesions here also may result in peculiar olfactory auras, as seen in certain hippocampal epilepsies (uncinate fits of Hughlings Jackson). Such fits occur from temporo-sphenoidal tumors also. Olfactory agnosias also result from lesions in this general region. Some of congenital origin, with agenesis of the cornu ammonis have been described. Anosmias or olfactory agnosias are frequent in general paresis, and in abscess and tumor. Complete absence of the rhinencephalon has been observed.[1]

The most common motor reflex observable is that of sniffing, with dilatation of the nostrils. This is occasionally seen as the result of a central somatic lesion (paresis, encephalitis), or as a symbolic automatic or unconscious act (compulsion neurosis, hysteria, schizophrenia).

Projection pathways—efferent—pass to the (small cells) mammillary bodies and to the habenulæ, in the former by ventral fibers passing to the tuber cinereum and infundibulum and by way of the fornix to the cornu ammonis (association efferent fibers). The true efferent projection system according to Winkler passes from the large cells of the mammillary bodies to the dorsal tegmental region and tuber cinerium on the side of the aqueduct. The posterior longitudinal fasciculus and fasciculus dorsalis tegmenti permits an association with the vagus nucleus. The tractus rhinencephalo-habenularis passes to the habenulæ. A further projection system unites the mammillary bodies with the diencephalon and neopallium. This is comprised chiefly in the fasciculus of Vicq d' Azyr, which arises chiefly from the median ganglion of the mammillary bodies. It terminates chiefly in the anterior nucleus of the optic thalamus. Winkler believes that a tertiary neuron system thus integrates the old primitive rhinencephalon with the

[1] Riese, W.: Deut. Ztschr. f. Neur., 1925, vol. **89**; Ztschr. f. d. g. Neur. u. Psych., vol. **69**.

neoencephalon. The clinical correlations here are still more obscure. Only a few can be discussed and these in a very haphazard manner, especially in a text-book.

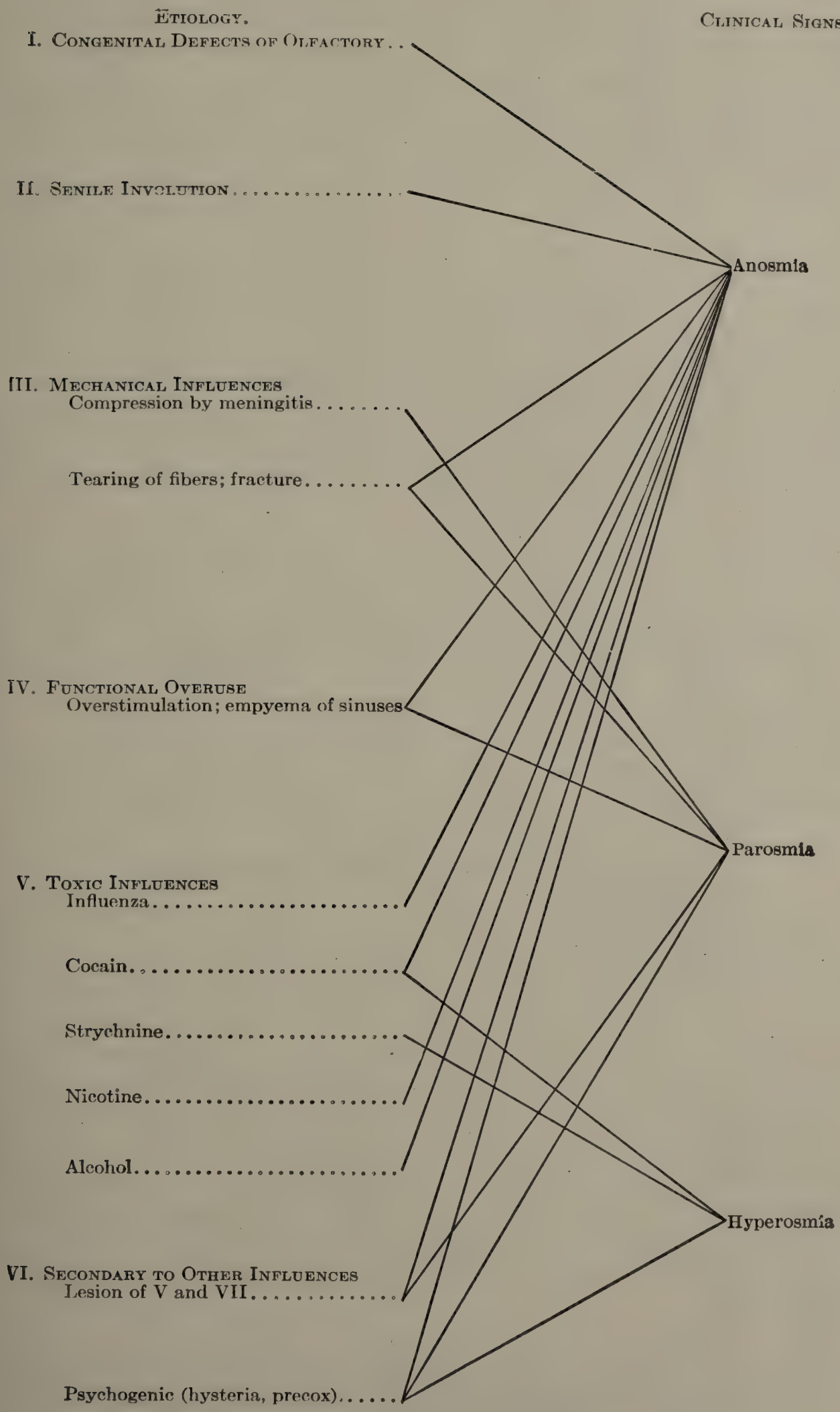


Fig. 149.—General summary of olfactory disturbances. (Veraguth.)

The relation between odors and the vomiting reflex is to be borne in mind in hysterical vomiting, (vagus connections) furthermore the very primitive associations between odor and sexual complexes (pallium symbolic connections). Hysterical anosmia may be an isolated phenomenon and is usually classical of the mechanism of conversion in a narrow Freudian sense. The psychology of smell and its complicated relationships to infantile phantasies of disgust, to childbirth, from intestinal canal, to the identification of feces and money, make the study of nasal disorders of great import in the psychoneuroses and psychoses. Hay fever, rose colds, etc., in many instances, are psychogenic conversion phenomena, others may be chemical reactions (pollen, horse odor, etc.). Many of the so-called anaphylactic reactions probably rest upon a psychogenic basis. In certain psychogenic epilepsies the olfactory symbolisms are highly developed. What relations these have, either as cause or result to the cornu ammonis lesions found in these epilepsies (Alzheimer) [1] [2] or to similar changes found in other convulsive states (paresis, etc.) is a highly interesting problem bearing on the relations of this deep-seated disorder to early vertebrate cortical function. Alikhan has reported an interesting epileptic family with hereditary anosmia.[3]

Treatment.—The underlying cause of the changed olfactory state needs treatment, not the state. Local applications of cocaine, adrenalin, etc., may be useful for acute situations. Many unnecessary mutilating operations are done upon the nose for psychogenic olfactory disorders which should be treated by psychotherapy. Marked psychogenic factors are nearly always present in nasal syndromes of the hay fever, rose cold, horse asthma types. Every nose receives the stimuli; why do certain ones react and not others? The terms protein sensitization idiosyncrasy or anaphylaxis still need to be explained.

DISEASE OF THE VISUAL APPARATUS.

The course of the light pathways and the topographical arrangement of its synapses are extremely complex, since sight has become so important a tool for advance in the evolution of vertebrate mental power. The earliest light tropisms in animals began with the protozoa, presumably in such an organ as the red spot. (Euglena and others.) From protozoa to man the evolution of the receptors which have been elaborated to capture light energies has been most complex and almost unanalyzable. Science only knows the merest rudiments. Different light modalities probably traverse different optic pathways. Of these little is known. Photic stimuli act upon other tissues than those of the retina, especially and importantly the chromatophores of the skin. Instance the great advances in light therapy in different pathological cellular states.

[1] Bailey, P.: Flaubert's Epilepsy, Proceedings of Charaka Society, New York, vol. 3.
[2] Clark, L. P.: The Epilepsy of Dostoiewsky, Medical Record, New York, 1915.
[3] Alikhan: L'épilepsie et l'anosmie héréditaire, Schw. med. Wchnschr., 1922, No. 11, p. 211.

The range of light response of the human retinal receptors is very great, yet it does not include the entire group of vibratory phenomena. What transforms the light energy into nerve energy—electrical—is not positively known. Mechanical and photochemical (through the visual purple of the rods) hypotheses are at present in the ascendant.[1] The human eye resolves points separated by 0.002 mm. at the fovea but this visual acuity rapidly fades off toward the periphery. It varies greatly for different colors and in different eyes. The stimulus threshold varies also and is very distinct in different individuals in whom enormous variations in color values exist. The best-known classical

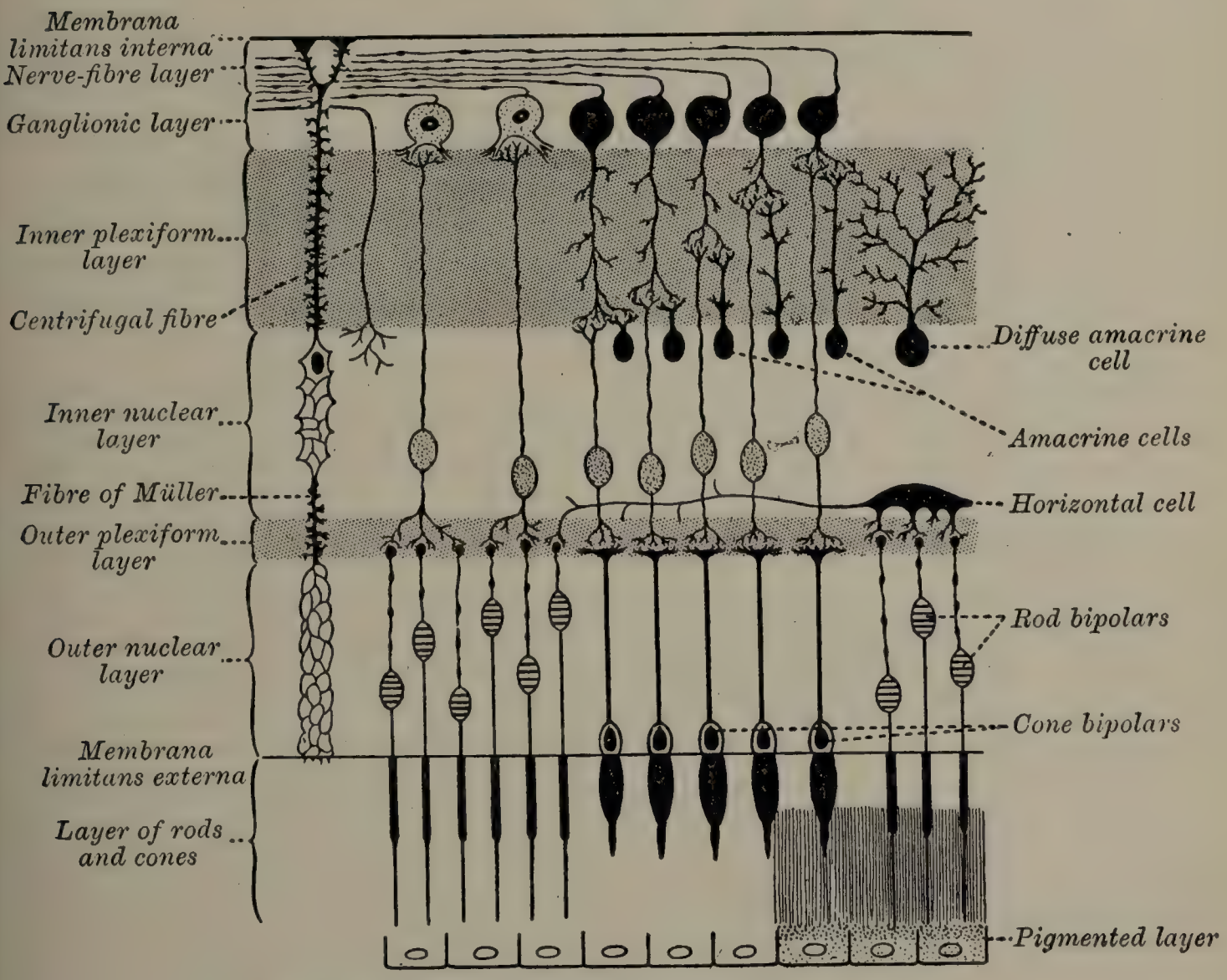


FIG. 150.—Plan of retinal neurones. (After Cajal.)

form of this difference is color-blindness. The human eye is sensitive only to vibrations approximately 0.0008 to 0.0004 mm. in length vibrating at a rate of 400,000,000,000,000 to 800,000,000,000,000, per second. This makes up about one-tenth of the entire range from the slowest tactile (heat) stimuli to the highest vibrations of the roentgen rays. No human sense organ is yet known that responds to the Hertzian electrical waves, the ultra-violet or the Milliken rays. The solar spectrum con-

[1] Hecht: Photochemistry of Visual Purple, Jour. Gen. Phys., 1920, **3**, 1. Bard: Du role des batonnets et des pigments retiniens, Jour. de Phys. u. d. Path. Gen., 1919, p. 276. Laurens, H.: Visibility of Radiant Energy, Am. Jour. Physiol., 1924, **67**, 348. Also for receptor organs, see Skramlik and Bethe, loc. cit. Olfactory.

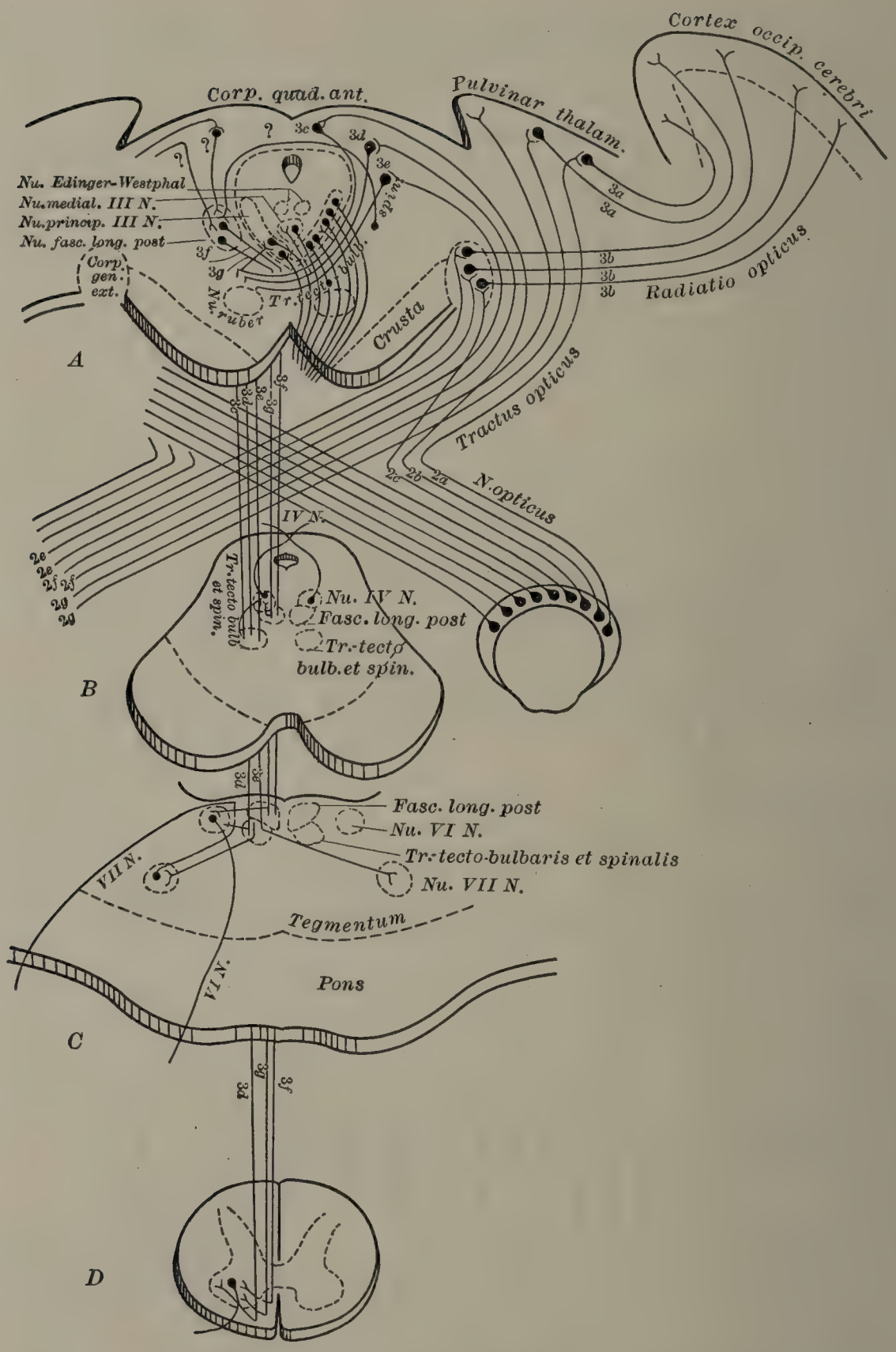


FIG. 151.—Diagrammatic scheme of optic paths and chief connections at four levels. *A*, level of II and III nerves; *B*, level of IV nerve; *C*, level of VI and VII nerves, tegmentum of pons; *D*, spinal cord. *Neuron* 1. Receptors in rods and cones of retina are not indicated in the diagram. *Neuron* 2. 2*a*, axones passing to pulvinar of same side; 2*b*, axones passing to corpus quadrigeminum of same side; 2*c*, axones passing to external geniculate of same side, all from temporal side of retina; from nasal side 2*a*, axones crossing in chiasm going to opposite external geniculate; 2*f*, axones crossing in chiasm to go to opposite anterior corpus quadrigeminum; 2*g*, axones crossing in chiasm to opposite pulvinar. Papillomacular bundle fibers crossed, partly uncrossed (see Fig. 153). *Neuron* 3. Pulvinar axones to occipital cortex; 3*b*, external geniculate axones to occipital lobes; 3*c*, *d*, *e*, corpora quadrigemina fibers, middle layer decussating (Meynert) to median longitudinal fasciculus and forming tractus tectobulbaris et spinalis to go to medulla and anterior columns, forming synapses with third, fourth sixth, and seventh nerves and motor nuclei of spinal nerves (space orientation); 3*f*, *g*, fibers from interstitial nucleus (Cajal) of fasciculus longitudinalis posticus forming part of longitudinal fasciculus passing to anterior columns, forming synapses with III, IV, VI cranial nerves and motor spinal nerves. *Neuron* 4. Axones from oculomotor, facial, and spinal nuclei. (Strong.)

tains about 10 octaves of this series. Many vibrations unperceived by the human eye seem to be responded to by the eyes of other animals. Von Kries estimated that the human eye could distinguish between 150 and 250 pure spectral tints, thus making a functional range of between 500,000 to 600,000 possible distinctions. Enormous variability exists which is at the bottom of much of the variation in perception of the painter or color artist. Evolution to wider and more useful adaptation is undoubtedly going on constantly. This is chiefly brought about through the psychical stimulus.[1]

The (1) retina, the (2) mostly decussating optic nerves ending in the external geniculate, the pulvinar of the thalamus and the anterior corpora quadrigemina, constituting primary centers, and finally (3) the optic radiations terminating in the calcarine region of the cortex make up the primary, secondary, and tertiary incoming neurons of the optic pathway the gross anatomy of which is shown in Fig. 151.

Color-blindness.—This exists in varying degrees and should be studied in special works since it offers many complex problems. Red-green difficulties are among the most important socially. Total congenital color-blindness consists in an inability to recognize any tints but black, white and gray; and although such cases are rare, and there is considerable difficulty in demonstrating a total inability to recognize colors, there are a few instances on record which appear to prove that the condition does exist. The problems connected with this form of color-blindness have reference to the relation it bears to certain symptoms that usually accompany it, more especially amblyopia, photophobia, and alterations in the visible spectrum. Total color-blindness may be associated with perfectly good vision, although it is exceptional for the visual acuity to be higher than one-third of the normal. Comparison with the changes met with in albinism are instructive. Albinism exhibits a syndrome closely resembling that of total color-blindness, except that color-vision itself is retained. In this condition the fovea is absent or imperfectly developed. Julia Bell[2] suggests that if the amblyopia and other symptoms common to the two diseases are due to this defect, one must look for some further defect in achromatism to account for the absence of color-vision; and that possibly the presence of central scotomata and the alterations in the visible spectrum observed in achromatism have a bearing on this point. There seems to be no doubt that the spectrum seen by the totally color-blind is identical with that seen by the normal eye under the condition of dark adaptation—that is to say, the red end of the spectrum is shortened, the brightest region is displaced from the yellow toward the green, and the stimulative action of the short-waved rays is much increased. It would appear that in this condition the macular region is not functioning, and that vision is carried on in

[1] Phillips: Questions Relating to Eye Training. Parsons: Color Blindness, 1917. Parsons: Studies in Perception, 1926.

[2] See Nettleship Memorial Vol. in Karl Pearsons' Treasury of Human Inheritance. Part II, Color Blindness, Julia Bell, vol. **23**, Cambridge, 1926.

the peripheral areas; such would seem to be the case, for instance, in retinitis pigmentosa. The eye is in an irritable state, the pigment is withdrawn from the percipient elements, and the retina is sensitive to slight stimulation. It is possible, therefore, that total color-blindness is due to the fact that stimuli from any wave length produce vibrations affecting too wide an area, and thus tend to bring about a sensation of all colors, and so of white and gray.

With regard to the anatomical changes associated with total color-blindness, the observations are meager. Two cases only are on record in which a microscopic examination was carried out; in one the eye and optic nerve were normal, and the color-blindness was attributed to some central lesion; in the other the cones of the foveal region were found to be imperfectly developed. Hereditary influences play a very limited part in the causation of congenital total color-blindness, for about 20 per cent. of all recorded cases appear to have no similarly affected relatives and show no other hereditary defects. On the other hand, a very high proportion of cases are the offspring of consanguineous parents, and this factor is almost certainly of great significance.

The more common form of color-blindness—dichromatism or congenital color-blindness—contrasts sharply with the total form, in that it is strongly inherited, belonging to that group of inheritable diseases which is mainly manifested in males and transmitted by their unaffected daughters, but not by themselves directly to their sons. Dichromic vision is usually considered to be characterized by a defect in the recognition of both red and green, the defect being more marked sometimes as regards the red, sometimes as regards the green, and in either case with varying intensity. Much controversy has centered around the question whether the mental picture normally stimulated by yellow and blue does in fact correspond to the two colors seen by dichromates. According to the theory elaborated by Edridge-Green it does not. This observer appears to consider that in the evolution of color-vision the first two wave lengths to be differentiated as colors are those at the extreme ends of the spectrum, and that the power to react specifically to stimuli from these regions is what is retained by the dichromic eye. Perhaps the most forcible argument against the Edridge-Green theory is afforded by cases of unilateral color-blindness, since in them the affected eye can be tested against the normal one. Careful examination of what appear to be genuine congenital cases seems to leave little doubt that the two retained fundamental tones are yellow and blue. Other characteristics of dichromic vision are its persistence, its frequent association with perfectly normal vision for form, and an enhanced sensibility for gradations of light and shade; the color sense also appears to be definitely changed in character under artificial light. With regard to inheritance, although rare in women, 91 cases of its occurrence in the female are on record.[1] Further, it has been asserted that for color-blindness to become manifest

[1] Bell, loc. cit.

in women the father must be color-blind, the mother must be a carrier, and the sons of the color-blind woman must be color-blind. Bell denies this assertion, in so far that the record of one sibship contained 3 color-blind females, whose father had normal color-vision; and in several cases color-blind women have been demonstrated to have sons with normal color-vision.

Retinitis.—Involvement of the first neuron is termed retinitis. It may result from the extension of an inflammation or may be due to toxic or to hidden constitutional factors. The chief indications are ocular discomfort or photophobia, diminution of the visual acuity, appearance of scotomata, general contraction of the visual fields, micropsia, megalopsia or metamorphopsia.

Different grades of retinitis are distinguished opthalmoscopically. The chief types are simple, albuminuric, syphilitic, diabetic, hemorrhagic and anemic retinitis.

In *simple retinitis* there is clouding particularly of the superficial layers, in patches or in larger portions at the posterior pole. The veins are dull and dark and full, and seem imbedded in the swollen or hazy retina. Sight is dim and worse in spots (scotomata). The disorder usually involves first one eye and then the other.

Albuminuric retinitis is frequent in nephritis (25 to 40 per cent.). Headache and loss of vision in a middle-aged to older person are the usual signs. There are characteristic changes in the retina and albumin and casts in the urine. Cirrhotic kidney is the most frequently accompanying somatic lesion. The chief change is an arteriosclerosis of the retinal vessels. They are unduly tortuous and show contractions and widenings, often being beaded. There is also a translucency in the retina, white stripes accompany the vessels. The veins are likewise tortuous, and disturbances of circulation show particularly at venous-arterial crossing. Retinal edema with grayish opacity shows. Hemorrhage is frequent. The margins of the disk become obscured, the nerve expanding into the retina without sharp lines of demarcation. The disk may be much swollen, woolly in appearance, and much extravasated. Fatty degenerations with "snow bank" appearances occur.

Blindness, scotomata, dimness of vision appear as in simple neuritis, but chronic cases of albuminuric retinitis may be present with little loss of visual acuity in the early stages. Permanent impairment of vision is the rule. Albuminuric retinitis accompanied by hemorrhages, and fatty degeneration of the retina, in a patient over thirty-five to forty years usually portends serious consequences within comparatively few years.

Syphilitic retinitis is probably much more frequent than has been supposed. It may result from hereditary or acquired syphilis, in the latter case appearing soon after infection. Clinically there is contraction of visual fields, dimness of vision, maybe night-blindness, or marked dimness of vision with poor illumination. Shimmering lights which are persistent and annoying occur, with micropsia and at times

metamorphopsia. Central, partial or complete scotomata are fairly constant.

The ophthalmoscope shows hyperemia with serous exudation much resembling the albuminuric variety but in milder degree. Hemorrhages are much rarer, and the "snow bank" glistenings much less pronounced. Opacity about the disk is a variant feature, with inflammation of the uveal tract.

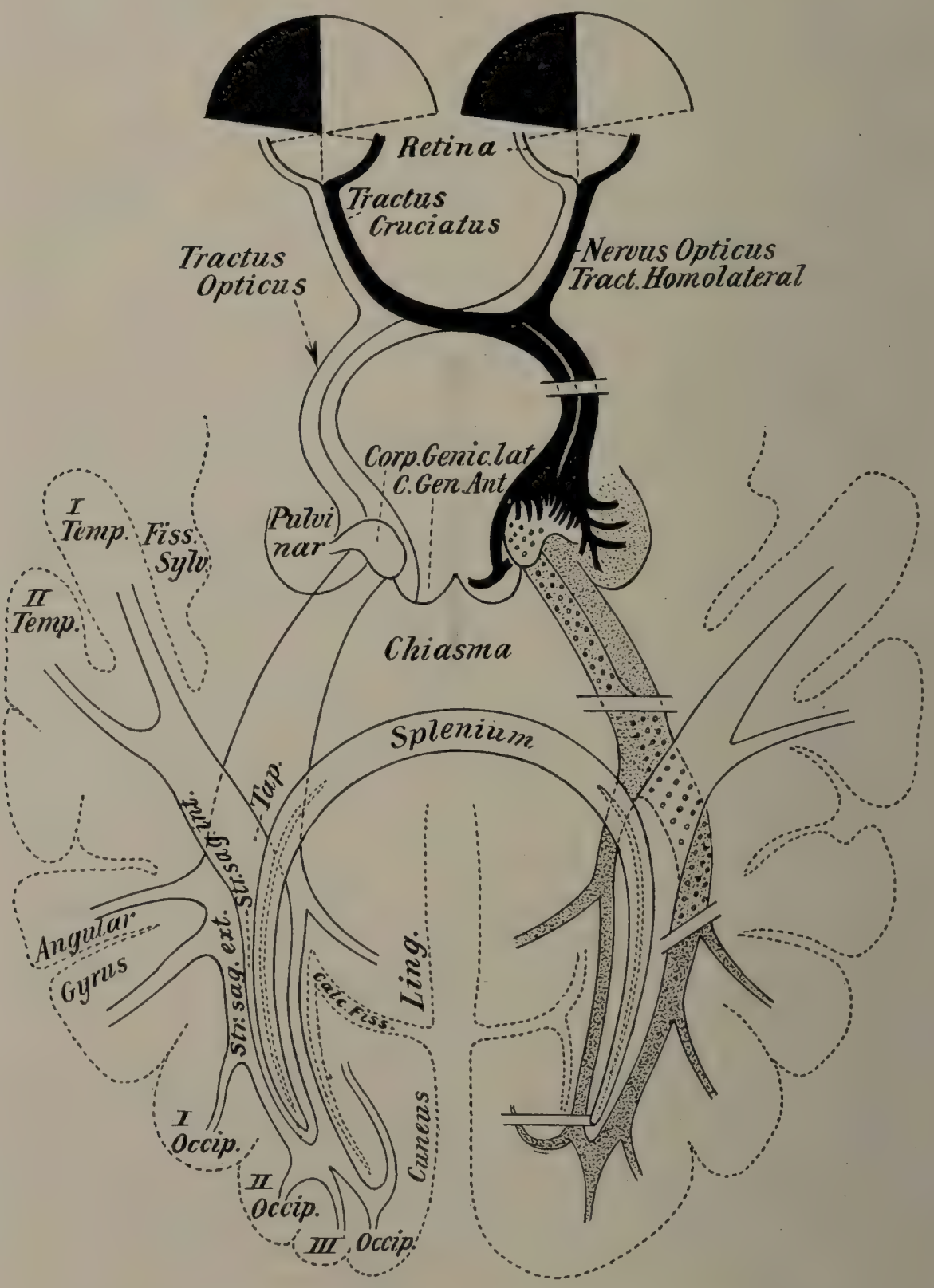
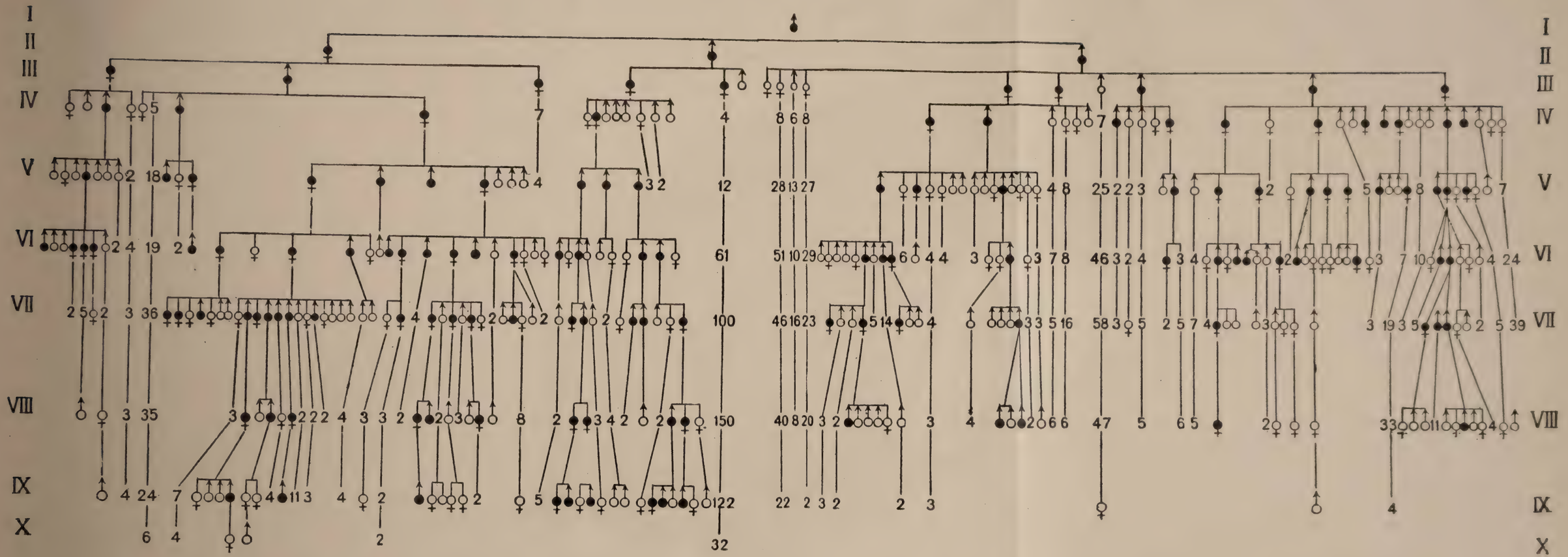


FIG. 152.—Course of Optic Pathways. (v. Monakow, Winkler.)

Hemorrhagic retinitis is of importance, but the student must be referred to works on ophthalmology with the other types.

Retinitis pigmentosa is a hereditary variety of primary retinal degeneration showing night-blindness with striking frequency. Nettle-

PLATE VIII



Descent of a Form of Stationary Night-blindness. (Condensed from the Chart Published by Nettleship, Based on Cunier's Records with Later Additions.) Only those Families which Contain Affected Members are Here Set Out in Detail. The Affected Man (DR) Who Married the Affected Woman (DR) is Represented by the Sixteenth Black Symbol from the Left in Generation VII.

Black symbols show the night-blind individuals. The descent is always through the affected, showing that the condition is due to a dominant factor. According to the records there is a great excess of normals over the affected. It cannot be said that the responsible factor is a simple allelomorph. Nevertheless, the table gives a remarkable illustration of the permanence and mode of descent of a dominant variety. (Bateson.)

ship's famous study of a French family showed this to be a striking instance of Mendelian dominance. (Plate VIII.)

Optic Nerve.—The disorders affecting the second optic neurons fall into two groups: those affecting (A) the optic nerve, morphologically a true brain tract, (B) its terminations in the midbrain structures.

A. **Diseases of the Optic Nerve.**—Here three situations need to be distinguished: (1) whether the affection lies anterior to the chiasm, (2) whether it involves the chiasm, or (3) lies behind the chiasm in the path of the optic neurons of the third order.

(1) Diseases of the Optic Nerve before reaching the chiasm: *Optic Neuritis,* in general sense. Three types are distinguishable with pronounced symptomatology. They are: (*a*) Axial Neuritis, (*b*) Interstitial Peripheral Neuritis, and (*c*) Diffuse Neuritis.

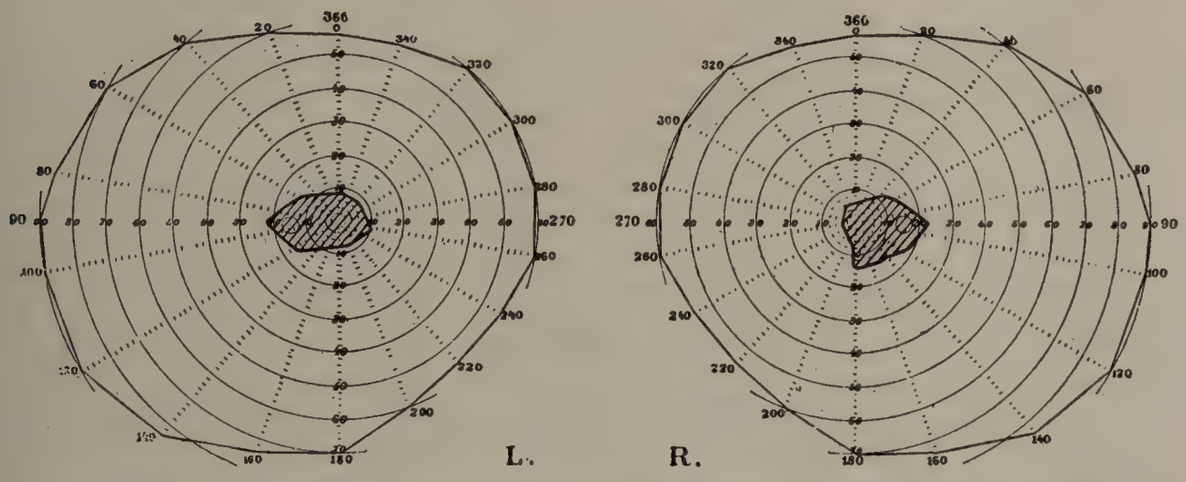


FIG. 153.—Central scotomata in acute axial neuritis. (Wilbrand and Sänger.)

(*a*) **Axial Neuritis.**—This is a system disease of the papillo-macular bundle, involving the nerve in front of the chiasm. It may be acute or chronic.

Acute Axial Neuritis occurs usually in young adults, twelve to twenty-four years, more particularly women. There appears sudden clouding or dimness of vision, and occasional photopsias. A frontal or temporal headache, or deep pain in the orbit, made worse by pressure or movement of the eyeballs, is present. The loss of sight is rapid, reaching a maximum usually in five days, and often is so severe that the patient can just count fingers at 15 feet, or is blind. With the loss of sight the headache lets up. There may be no retinal changes. The pupil of the affected eye is larger, and is usually sluggish to direct light stimulation, but shows no consensual light reflex loss.

After the acute stage is over there is gradual recovery of the sight at the periphery with various degrees of persistent central scotomata. The loss may be unilateral or bilateral and absolute, or unilateral or bilateral to color only, or various gradations of these paracentral scotomata, etc. The scotomata gradually diminish and after six to eight weeks, with proper therapy, may entirely disappear (Fig. 153).

The fundus picture may remain normal throughout or show a papillitis. This will depend upon how far back of the optic disk the lesion, which is usually a vascular one, occurred. When there is a papillitis it shows slight paling of the temporal half or halves of the fundi (Fig. 153).

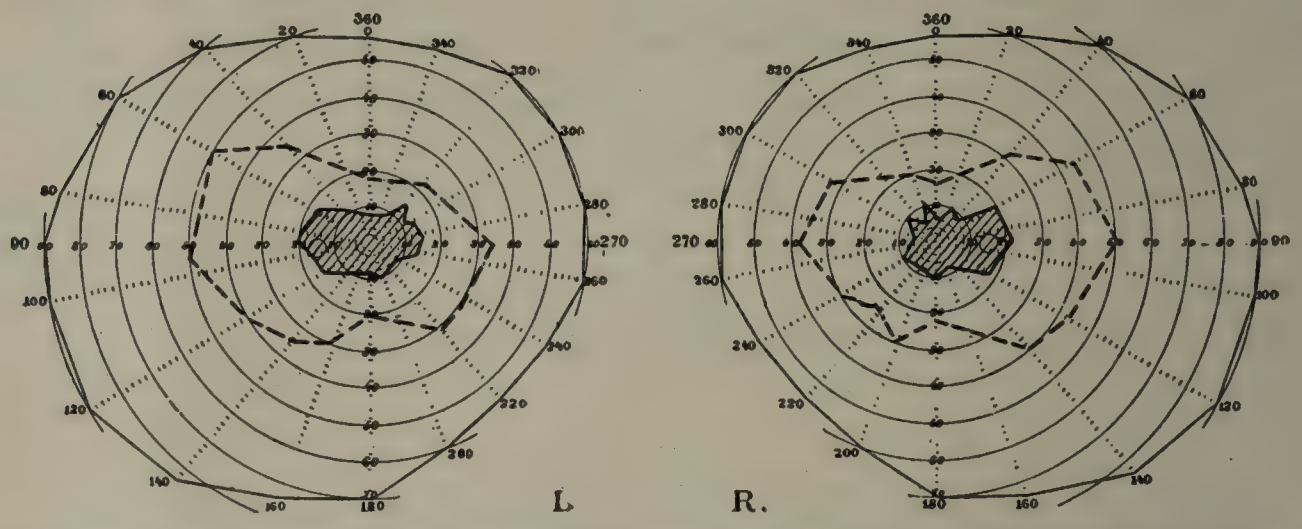


FIG. 154.—Scotoma for red and green in tobacco axial neuritis. (Wilbrand and Sänger.)

Etiology.—The most frequently ascribed cause is exposure to cold. This is probably only an incident to other real causes such as infectious disease—syphilis, tuberculosis, typhoid, erysipelas, sinusitis, influenza,[1] mumps, pneumonia, tonsillitis, cerebrospinal meningitis, malaria, beriberi, etc., or toxemias, such as those of pregnancy, nephritis, burns, CO_2 poisoning, methyl alcohol, quinine, felix mas, morphin, etc.

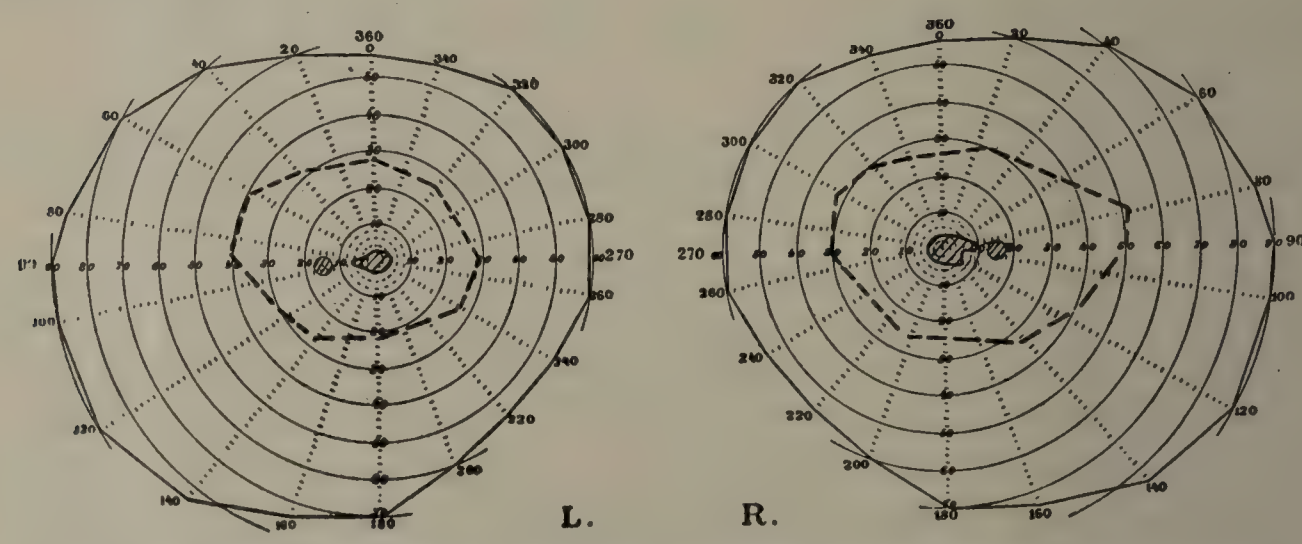


FIG. 155.—Beginning scotoma for red at the onset of a tobacco or alcoholic axial neuritis. (Wilbrand and Sänger.)

Treatment.—Hot baths, and treatment of cause, as antisyphilitic in syphilis, etc.

The *Chronic Form* is much more frequent. It is the classical situation in chronic alcohol or nicotine poisoning, and affects males more often. Here the course is a chronic one, spreading over several

[1] Jelliffe: Nervous and Mental Involvements of Influenza, New York Med. Jour., November, 1918.

months or years. The blindness appears slowly, and at first consists of a central scotoma for colors, or of a hemeralopia, the patient seeing better in the dusk than in the bright light (fatigue). The scotomata become more marked if the poisoning continues (Fig. 154).

The type of scotoma varies widely. Bilateral, fairly symmetrical, oval scotomata for red and green, lying between the blind spot and the fixation point is the early picture. It usually starts as a defect for red, stretching toward the blind spot (Fig. 155). The chief defect usually lies about 2° to 8° from the fixation point. Absolute central scotomata are rare.

The acuity of vision is usually diminished, and more on one side than the other. In monocular reading the type to the right of the fixation point is not clear for the right eye, while for the left eye the defect lies to the left. The defect in vision bears little direct relation to the size of the scotomata. Pupillary anomalies, diminution of both light and accommodation reflexes and pseudotabetic pictures are to be found.

The fundus picture may be normal with gross defect in vision and large scotomata, or there may be hyperemia, a mild neuritis, with some temporal pallor. If marked pallor is present it speaks in general for a more severe process.

The course is chronic. Removal of the cause may save the sight, but slight scotomata may persist. Atrophy is the end in the severe cases.

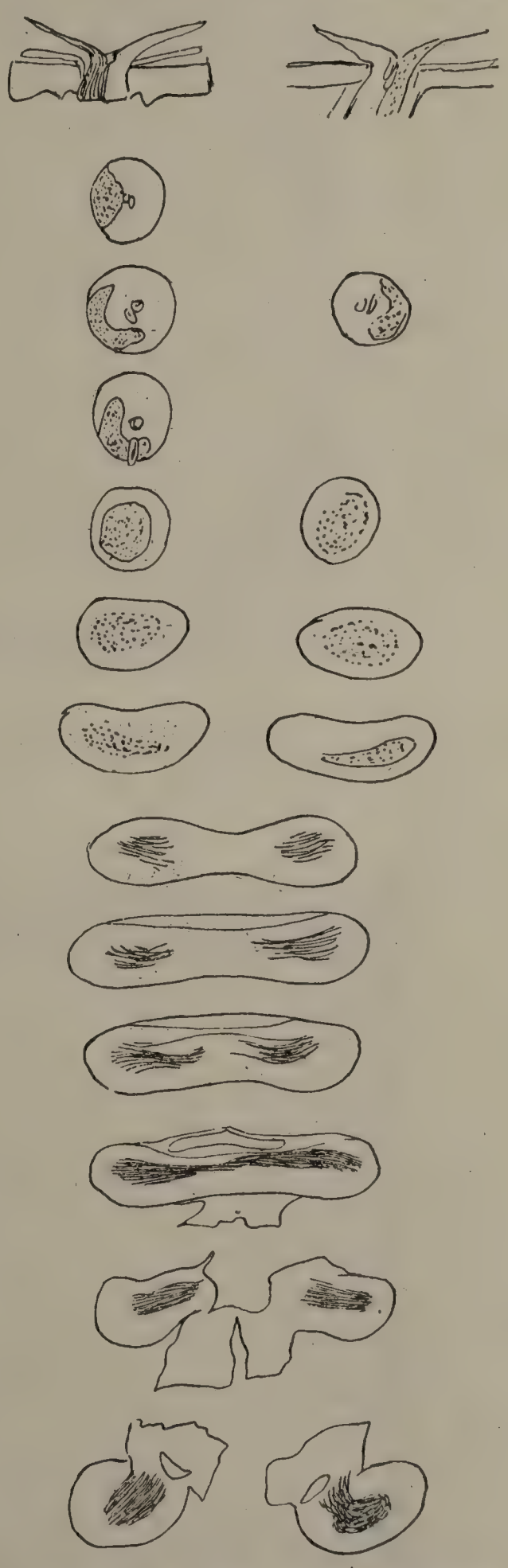


FIG. 156.—Course of the papillo-macular bundle at different points in the optic tract. (Wilbrand and Sänger.)

Pathology.—The changes in both acute and chronic types occur in the papillo-macular bundle. They are degenerative rather than inflammatory changes, with greatest severity in the optic canal, because of the richer vascular supply of the nerve trunk at this point.

Etiology.—The chief causes are alcohol and tobacco. Other less frequent poisonings are by carbon bisulphide—in the rubber industries—arsenic, lead, sulphuric acid, anemia (anchylostoma), diabetes, aniline, gasoline.

Hereditary Forms.—These occupy intermediary positions. One type affects usually the males, passing through unaffected females (Knight's move in heredity). These develop acute or subacute headaches about the age of twenty, with gradually diminishing vision. Unequal bilateral scotomata appear and gradually increasing central blindness. There is usually a subacute and progressive stage for about six months, one eye lagging behind the other in its deterioration.

Gradually increasing central scotomata show. Nyctalopia is frequent. The marginal vision is retained, save for colors. Total blindness develops in about two-thirds of the cases. One-third remain more or less stationary or even improve. In mild cases central color scotomata alone appear, or there is diffuse loss of color sense without scotomata.

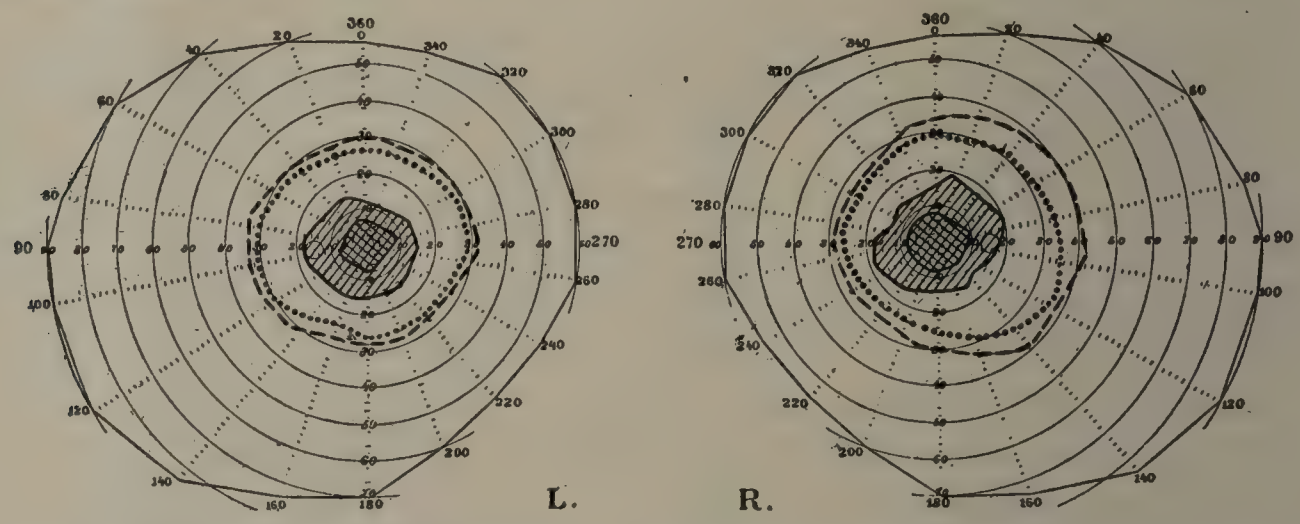


FIG. 157.—Visual fields in a patient with hereditary axial neuritis. Fields for white normal, for blue – – – –, and for red, concentrically contracted. Absolute central scotoma with larger bordering scotoma for blue and red. (Wilbrand and Sänger.)

The fundus picture is variable, but the presence of atrophic changes, usually temporal, stands in marked contrast with any other changes in the nervous system.

Hereditary syphilis plays a role in some cases. In others there is an anomaly in the germ plasm.

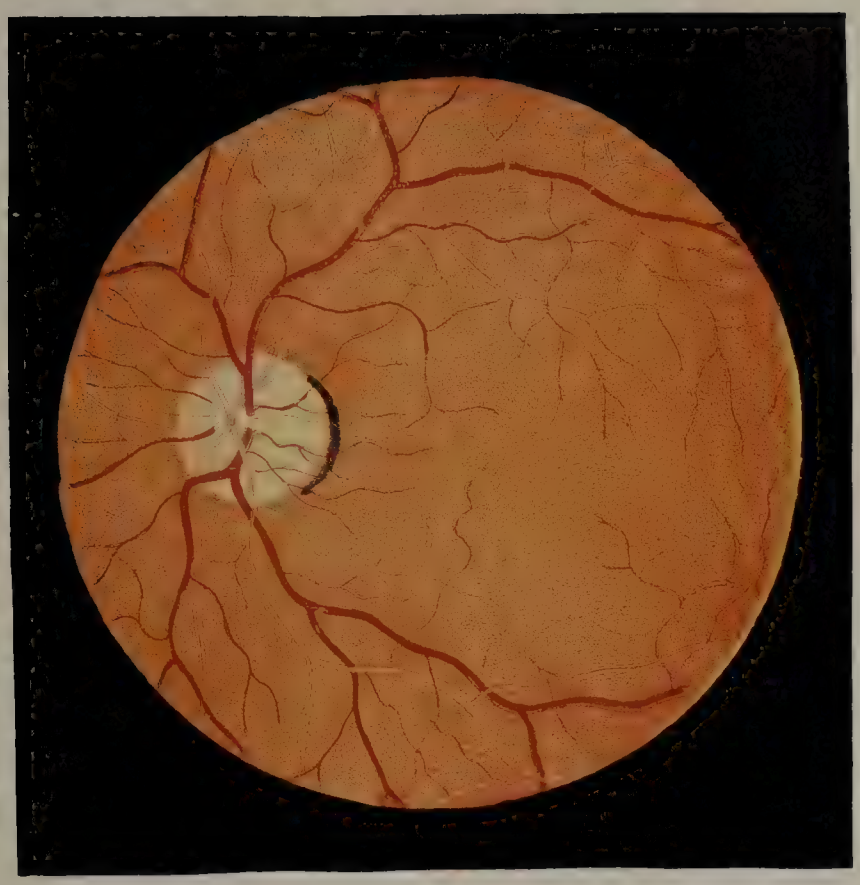
(*b*) **Interstitial Peripheral Neuritis.**—Here the disease involves the periphery of the optic nerve rather than the central or eccentrically lying papillo-macular bundle. Concentric limitation of the field of vision for white and colors is the chief finding rather than central scotomata. Here there is a peripheral inflammation of the nerve trunk, starting in the pia and proceeding inward in the septa.

The concentric limitation of vision is rarely observed in the beginning. As it slowly advances the patients become uncertain of space localization and need to turn the eyes frequently to get clear pictures of the surroundings. Central vision is usually sharp even for color.

The fundi show simple or neuritic atrophy, occasionally choked disks. Very variable fields are observed (Figs. 149, 151, 152 and 153).

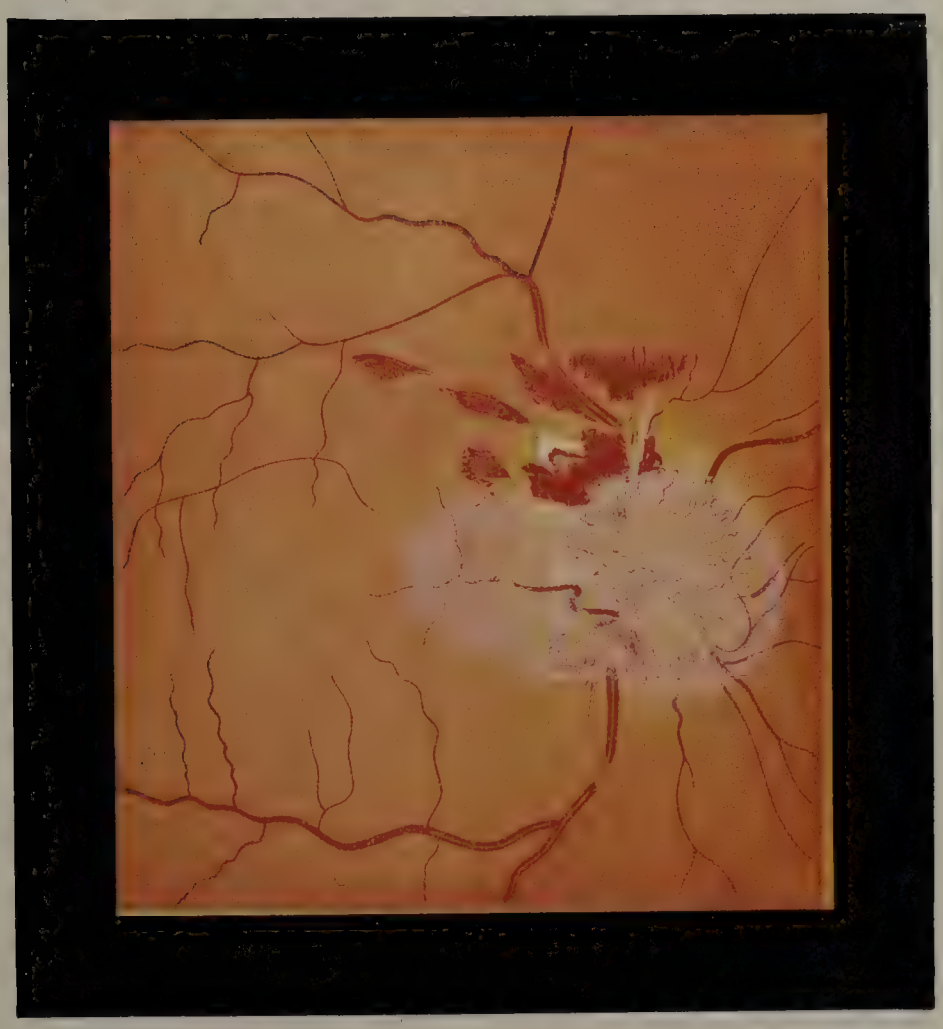
PLATE IX

FIG. 1



Normal Eye-ground (average tint). (Norris and Oliver.)

FIG. 2



Ophthalmoscopic Appearances in Early Stage of Optic Neuritis.
(Norris and Oliver.)

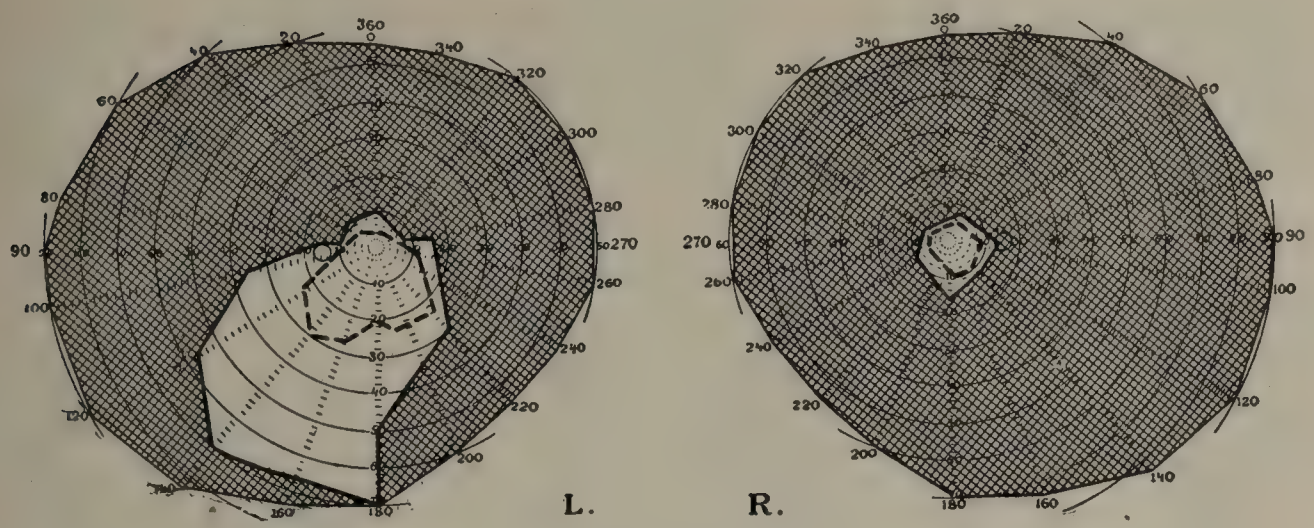


FIG. 158

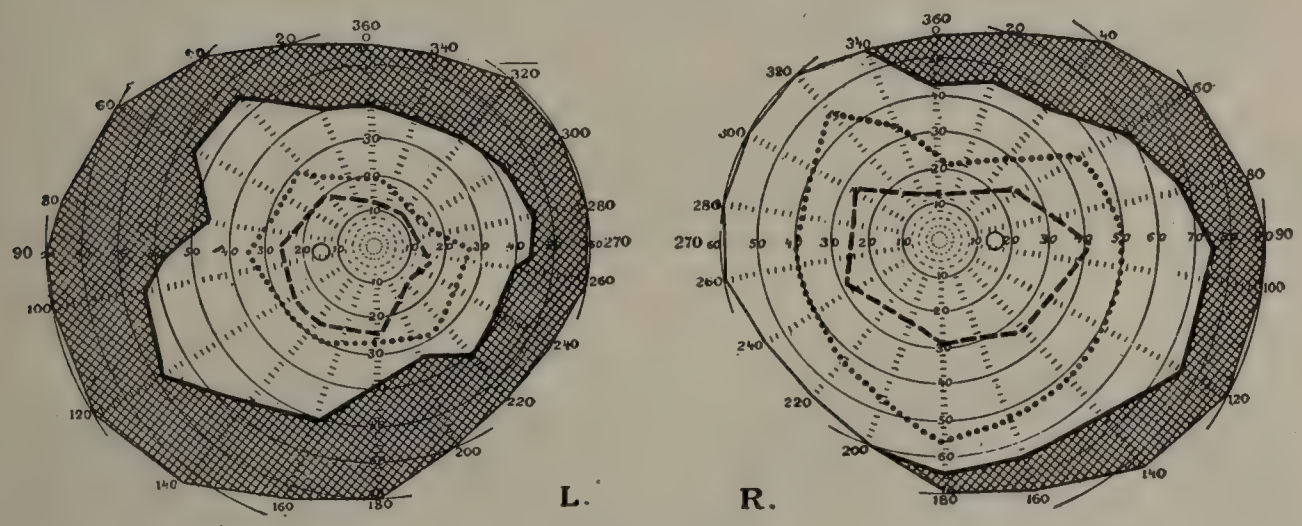


FIG. 159

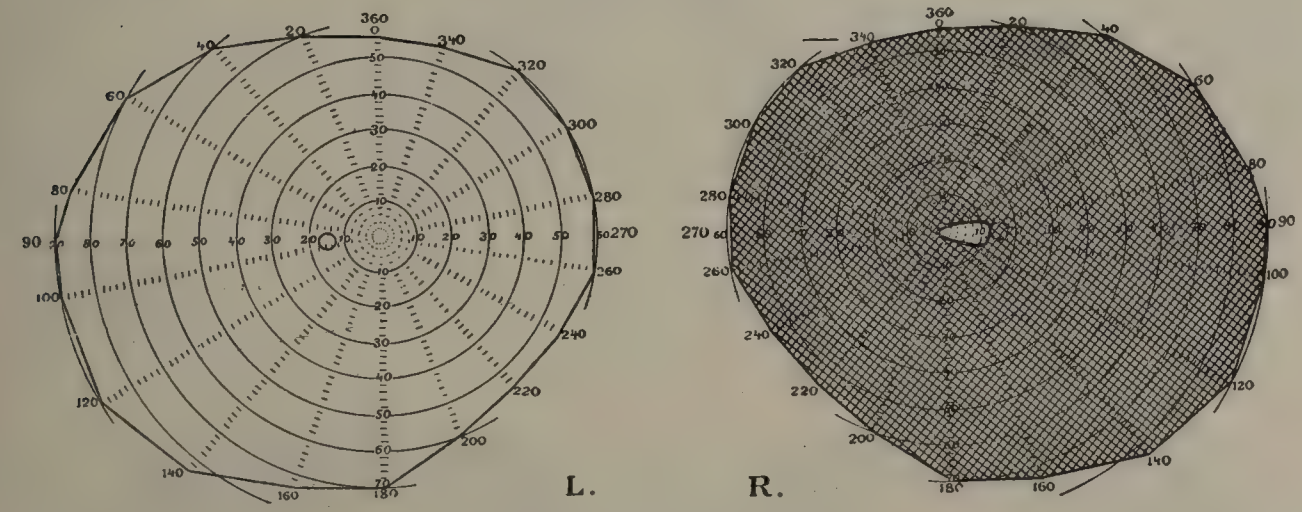


FIG. 160

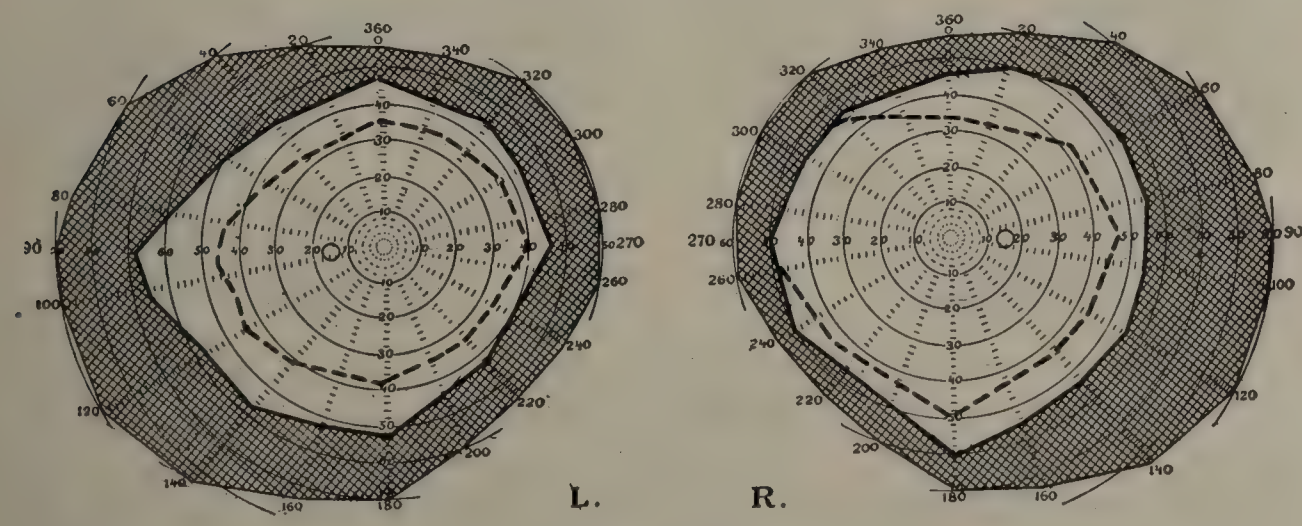


FIG. 161

FIGS. 158, 159, 160 and 161.—Visual fields in cases of interstitial peripheral optic neuritis from syphilis. (Wilbrand and Sänger.)

Hysterical limitation of the field is to be considered here. Normal fundi are consistent with a true interstitial neuritis, since lesions lying far back in the nerve trunk may cause little or no disk changes.

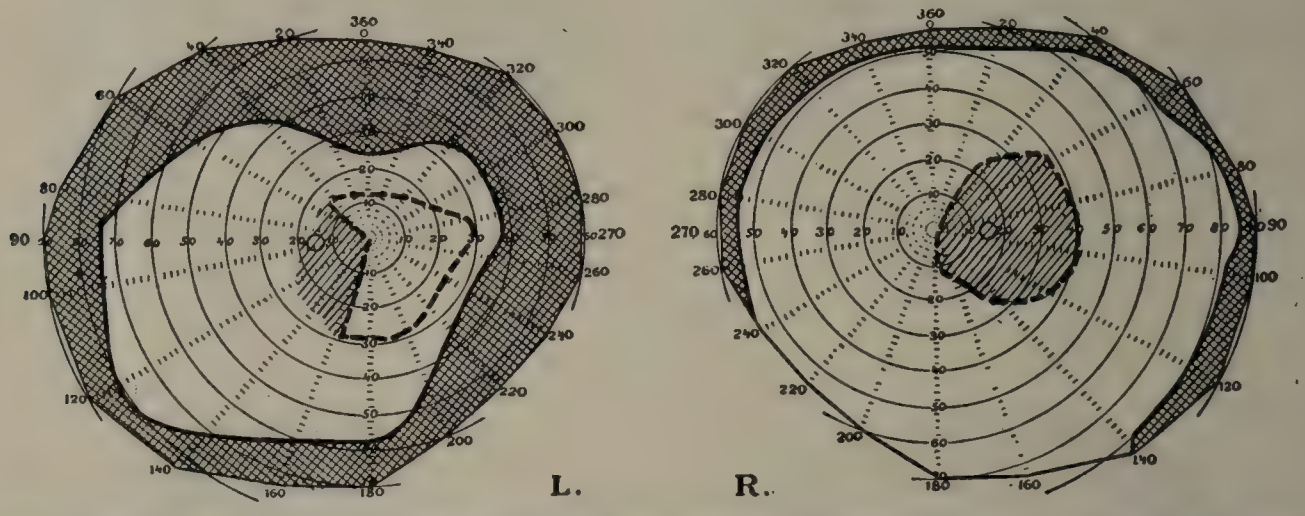


FIG. 162.—Visual field with diffuse neuritis due to multiple sclerosis. On right side a temporal hemianopic color scotoma, on the left intermittently appearing hemianopic uncertainty scotoma. (Wilbrand and Sänger.)

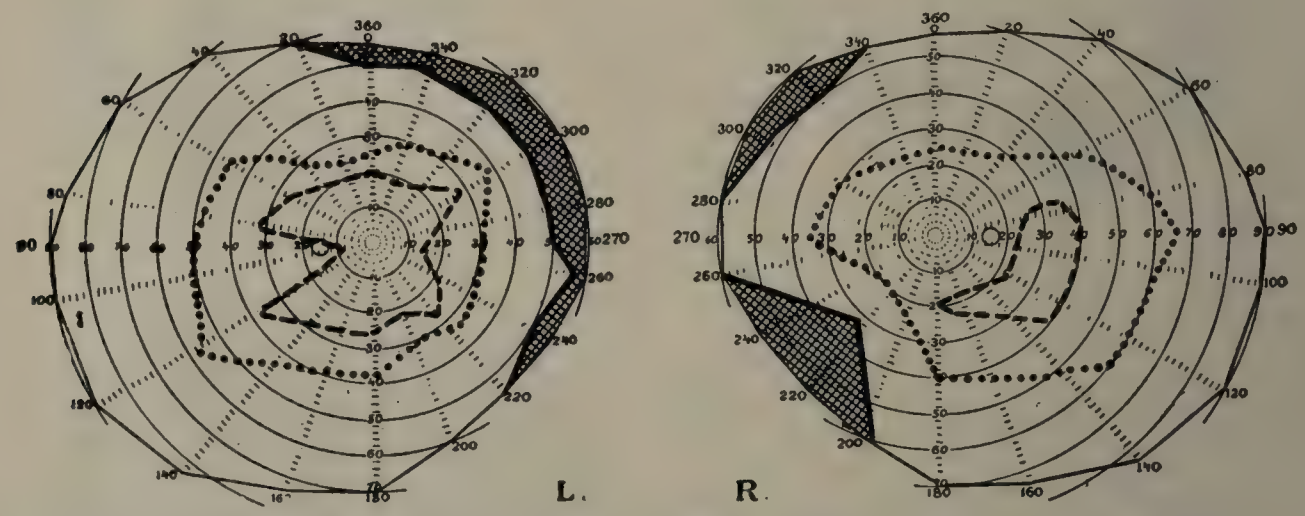


FIG. 163.—Visual field showing atrophy in tabes. Field for blue, for red ----
(Wilbrand and Sänger.)

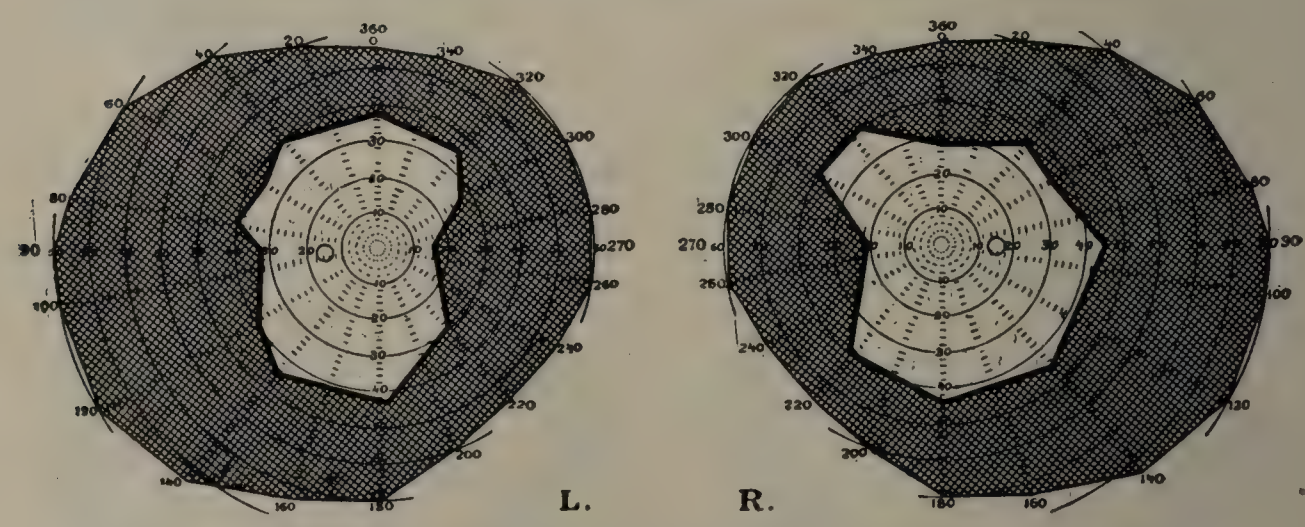


FIG. 164.—Optic atrophy in tabes with loss of color discrimination.
(Wilbrand and Sänger.)

A psychoanalytic anamnesis usually will clear up the diagnosis of a possible hysteria. Tabes with neuritis may begin as an interstitial neuritis. The cytobiological findings will establish the diagnosis.

Etiology.—Syphilitic meningitis of the base is the most frequent cause. A negative Wassermann in this group of patients is not a just criterion to deny specific medication. Other etiological factors are measles, diphtheria, influenza, myelitis, gonorrhea, sinusitis, typhoid, lead, arsenic, diabetes, leptomeningitis, cerebrospinal and tuberculous meningitis, and local pressure in optic canal.

The *therapy* is causal, usually specific. Arsenic or salvarsan or similar synthetic is less to be feared than an active syphilis. Many so-called neurorecidives causing blindness are due more to the syphilis than to the arsenic content of the drug. It must be remembered, however, that arsenic is capable of producing an interstitial optic neuritis.

(*c*) **Diffuse Neuritis.**—Here the inflammatory process involves the entire nerve tract, resulting in marked amblyopia or blindness. It may be implicated locally or throughout its entire course from the retina to the chiasm. In acute myelitis at times this severe inflammation occurs as a complication. Infectious diseases are important. Influenza here plays almost as large a role as syphilis.[1]

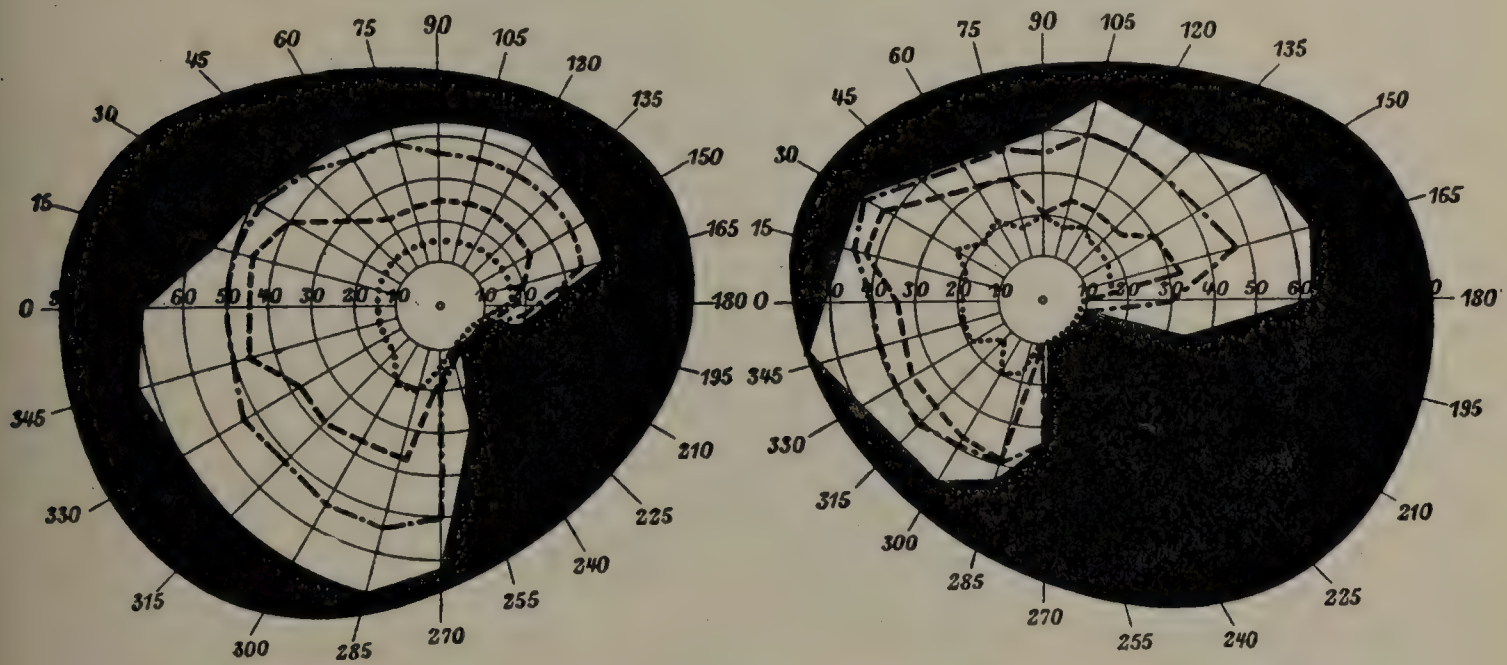


FIG. 165.—Quadrant hemianopsia of lower right segment due to hemorrhagic destruction within the external geniculate. (See following figures.)

The infections and toxemias mentioned in the preceding paragraphs may also induce a total optic neuritis. Malaria, scarlet fever, yellow fever, erysipelas may be added to the causes. Orbital sinus disease is important, and also multiple sclerosis.

Other atrophic states, double, one-sided, total or partial, occur, either in the papillæ, from pressure of a glaucoma, or may be descending atrophies from higher lying causes such as brain tumor, hydrocephalus. Primary progressive atrophy, arising by itself, probably rarely occurs. The most suggestive cause of an isolated, bilateral, progressive optic atrophy without other tangible neurological signs is tabes. Cyto-biological tests will complete the diagnosis (Figs. 163 and 164.)

[1] White, L.: Optic Canal in Optic Atrophy, Boston Med. and Surg. Jour., 1925, **193**, 771.

B. **Disease at or about the Chiasm.**—The anatomical peculiarities, due to the crossing of the fibers at the chiasm, introduces certain definite signs which are of value. Scotomata and concentric limitation are replaced by hemianopsias of varying type.

In lesions in front of the chiasm bitemporal hemianopsia will be present. This is rare. A lesion behind the chiasm, usually in the sella turcica, and not infrequent, as in pituitary disease, causes a binasal hemianopsia, partial or complete. Lesions to the right or left of the chiasm will cause incomplete homonymous hemianopsias—whereas lesions in the tract back of the chiasm—*i. e.*, in the midbrain or optic radiations or occipital lobes will cause a usually more complete homonymous hemianopsia.

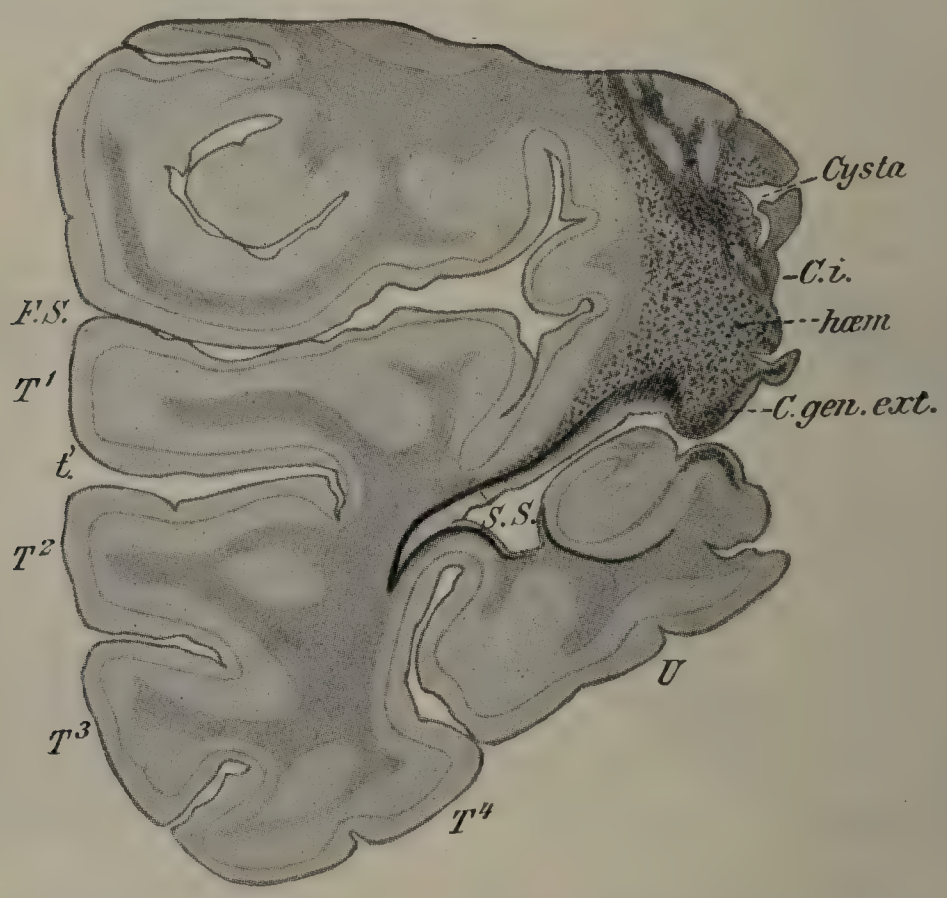


FIG. 166.—Site of lesion in external geniculate giving rise to quadrant hemianopsia seen in Fig. 165 and the secondary degenerations in Fig. 167. *C. gen. ext.*, external geniculate; *hæm*, hemorrhage; *C.i.*, internal capsule; *F.S.*, fissure of Sylvius; *S.S.*, optic radiations. (Henschen.)

Horizontal hemianopsias, either superior or inferior, occur in chiasm lesions from pressure above or below. They are readily explained from the position of the crossing fibers in the chiasm. Such hemianopsias may rarely occur from retinal causes.

A common cause for chiasm changes is pituitary tumor. Syphilis, however, is specially frequent in just this situation, most basal syphilitic meningitic processes beginning here. The process spreading forward to the optic stem produces a multiplicity of field changes. Thus one may have partial scotomata, monocular temporal hemianopsia, bitemporal hemianopsia (the most frequent), temporal hemianopsia with blindness of one eye, blindness in one eye and nasal hemianopsias of the other, blindness in both eyes. This very great irregularity

and changeability, advancing or receding under treatment, is of much importance in excluding a pituitary tumor. A loss of the hemiopic pupillary reaction is of importance in making a definite localizing diagnosis.

The pupillary changes are variable. Other signs of basal syphilitic meningitis are discussed in the chapter on Cerebral Syphilis (*q. v.*).

Affections of the chiasm are more rarely encountered as a result of trauma, brain tumor with general pressure, cavernous sinus disease, cerebrospinal and tuberculous meningitis, bone disease, aneurism and arteriosclerosis of the carotids.

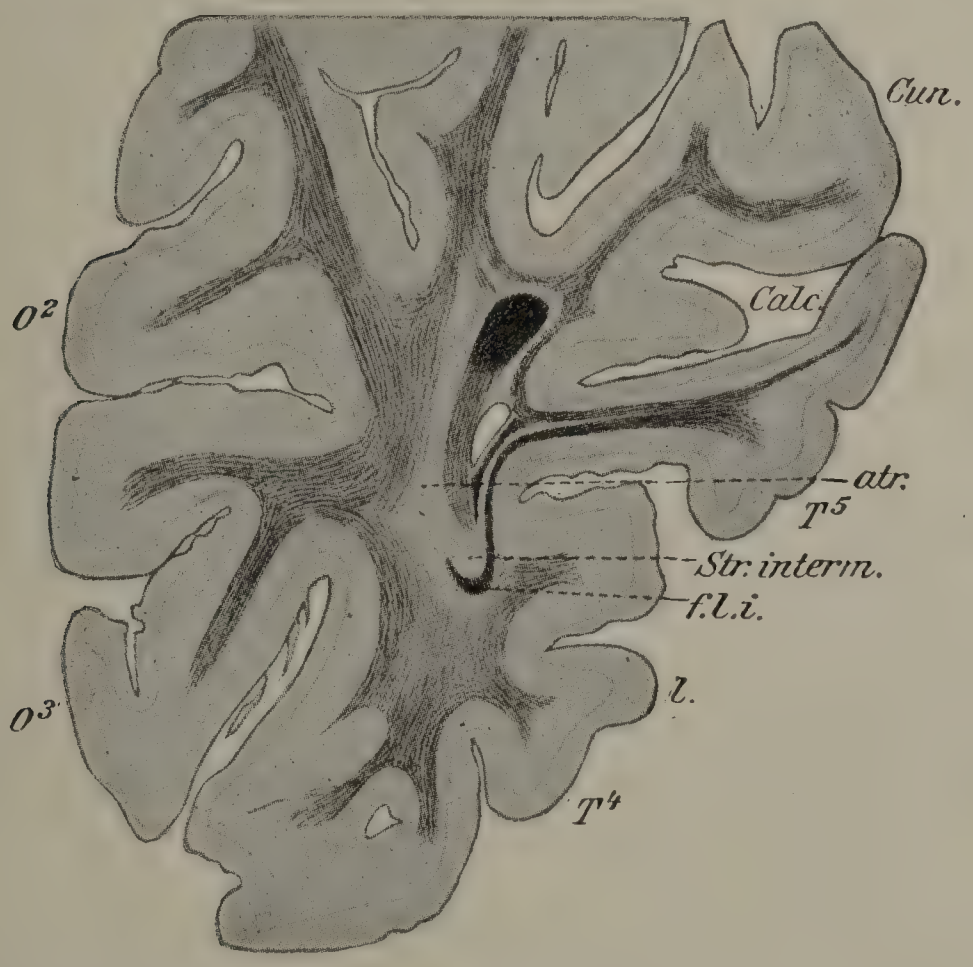


FIG. 167.—Showing atrophic degenerations in optic radiations (*atr.*) from hemorrhage in external geniculate (Fig. 166,) giving rise to quadrant hemianopsia of Fig. 165. *Cun.*, cuneus; *Calc.*, calcarine fissure; *f.l.i.*, inferior longitudinal fasciculus; *l*, left hemisphere; *atr.*, atrophy. (Henschen.)

C. **Quadrigeminal and Pulvinar Disease.**—In lesions of the optic tract posterior to the pulvinar, *i. e.*, in the optic neuron of the fourth order, pupillary disturbances are absent. Thus Wilbrand and Wernicke (three years later) have shown that by careful illumination of the blind side of the eye one can distinguish between hemianopsia in the optic neuron of the third order (by loss of pupillary light reflex) and a hemianopsia of the optic neuron of the fourth order (intact hemiopic pupillary reflex). As a matter of fact this test is extremely difficult to perform, but fortunately lesions in the midbrain—end-station of optic neuron of the third order—are almost invariably accompanied by other sensory signs (usually a complete or incomplete thalamic syndrome, *q. v.*), thus aiding in the diagnosis. The distributions of the optic neurons of the third order are multiform. Some fibers end in the corpora quadrigemina. Lesions here cause pupillary changes;

others end in the thalamus (pulvinar), and their involvement causes no disturbances of vision. The majority of the fibers form their synapses in the external geniculate bodies. These form rich collateral associations with the auditory tract, the sensory tracts, the third, fourth and sixth nuclei by means of which eye movements are associated with light stimuli, and through the median longitudinal fasciculus with the synapses for the lower cranial neuro-nuclei and spinal muscle nuclei, thus associating the projicient muscular system with the eye stimuli. (See Plate X).

Lesions of the optic tract in the geniculate region cause not only homonymous hemianopsias often only quadrant, but they are also liable to be complicated by the involvement of these other near-lying structures. Isolated quadrant hemianopsias may result from small hemorrhages, thrombi, emboli, tumors, or encephalitis (poliomyelitis—rare) in the external geniculate as well as from lesion farther back in the tractus (Figs. 165, 166 and 167). They may also break up a huge number of associations of the projicient mechanism. Geniculate vision is known for lower animals (Brouwer), and probably causes unconscious adaptations in man.

D. **Cortex Diseases.**—Lesions of the end stations of the optic tract or its associated areas in the occipital lobe may cause mind-blindness, *i. e.*, optic agnosia. Here the patient may have no disturbance of sight, or he may have partial hemianopsia, but is unable to recognize words or objects previously known, speech being intact.

Visual hallucinations are present in disorder of the optic end-stations in the occipital lobe. When they show definite projections in space one can make an approximate localization of the portion of the lobe involved. This may be of value in determining the site of a tumor or abscess formation.

The chief arterial supply of the posterior neurons is drawn from the calcarine branch of the posterior cerebral. The most occipital portion is supplied by the median cerebral. The anterior cerebral sends branches which innervate the optic radiations just posterior to the corpus callosum, but lesions of this artery at this place cause no definitely recognizable lesions. The geniculo-calcarine radiations pass in close relation to the temporal lobes, and may be involved in temporal lobe disease. (Flechsig, Henschen, Archambault, Meyer, A., Cushing.[1]) Careful perimetric charting shows such involvement as partial homonymous field defects, visual hallucinations may be present as well.

Recent experiences in war surgery are permitting a more comprehensive mapping of the cortical representation of the different parts of the retina. That of the macula in particular has been specially studied by Henschen, Bolton, Inouye, Brouwer and Lenz. Lister and Holmes[2]

[1] Meyer, A.: Tr. Am. Phys., 1907. Cushing: Brain, 1921, **44**, 341.

[2] See Lister and Holmes: Disturbances in Vision from Cerebral Lesions, Proc. Roy. Soc., Sect. on Ophthalmology, March 22, 1916, one of the many illuminating papers on this topic. (See also Brain, 1916, vol. **39**, parts 1, 2. Also Villaret et Beaulieu, Ann. de Méd., 1918, v. Brouwer: Jour. of Neurol. and Psychopath., 1925, **6**, 1. Kleist: Die einzeläugigen Gesichtsfelder, etc., Klin. Wchnschr., 1926, vol. **5**.

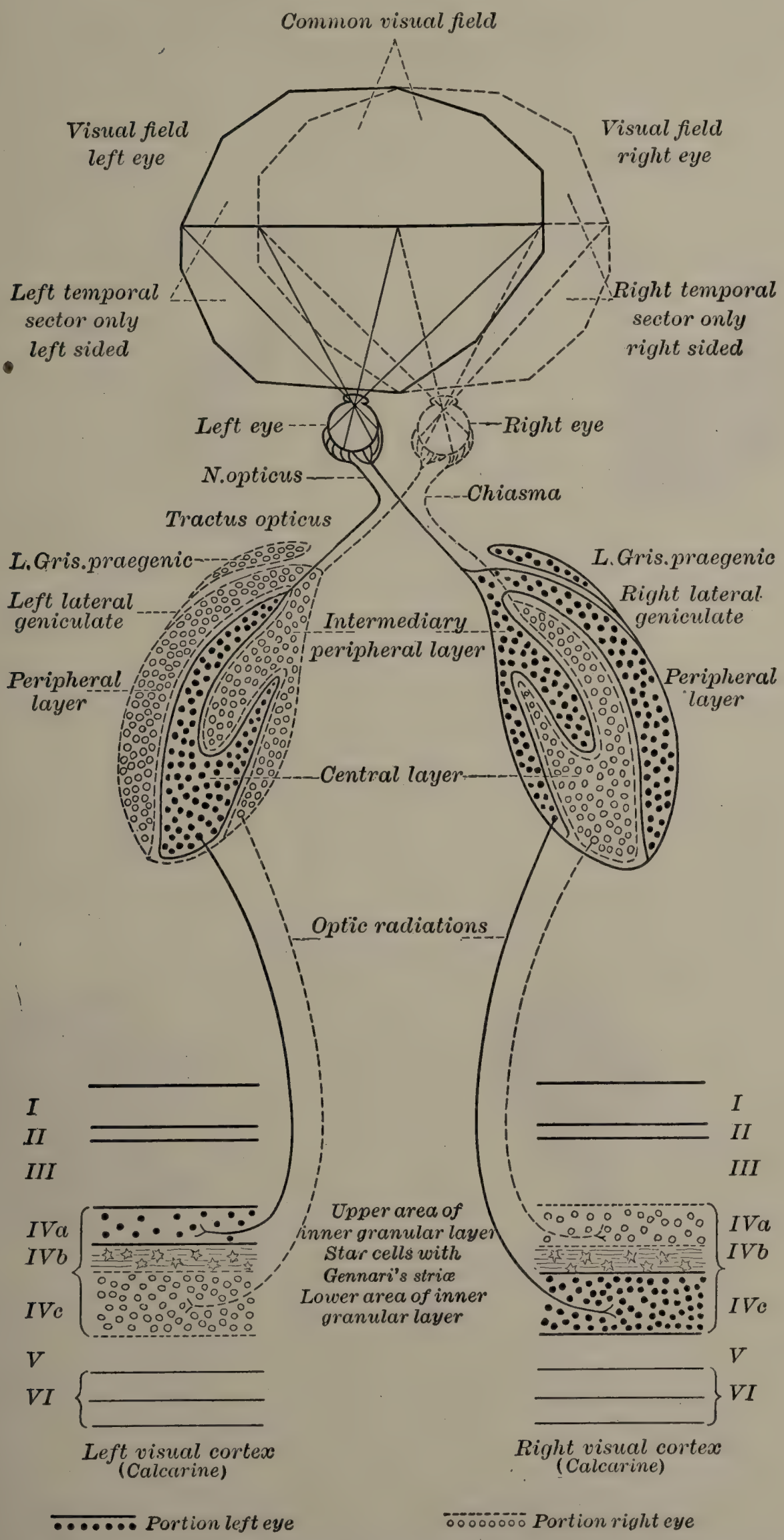


FIG. 168.—Scheme of projection of visual fields, showing geniculate and cortical distributions. (Adapted from Minkowski (Schw. Arch. Neurol. u. Psychiat., 1920, **7**, 288) and Kleist (Klin. Wchnschr., 1926, vol. **5**.)

support Henschen's and Inouye's view that in lower quadrant hemianopsias of cortical origin from gunshot wounds the upper halves of the retinæ are represented in the upper lips of the cal-

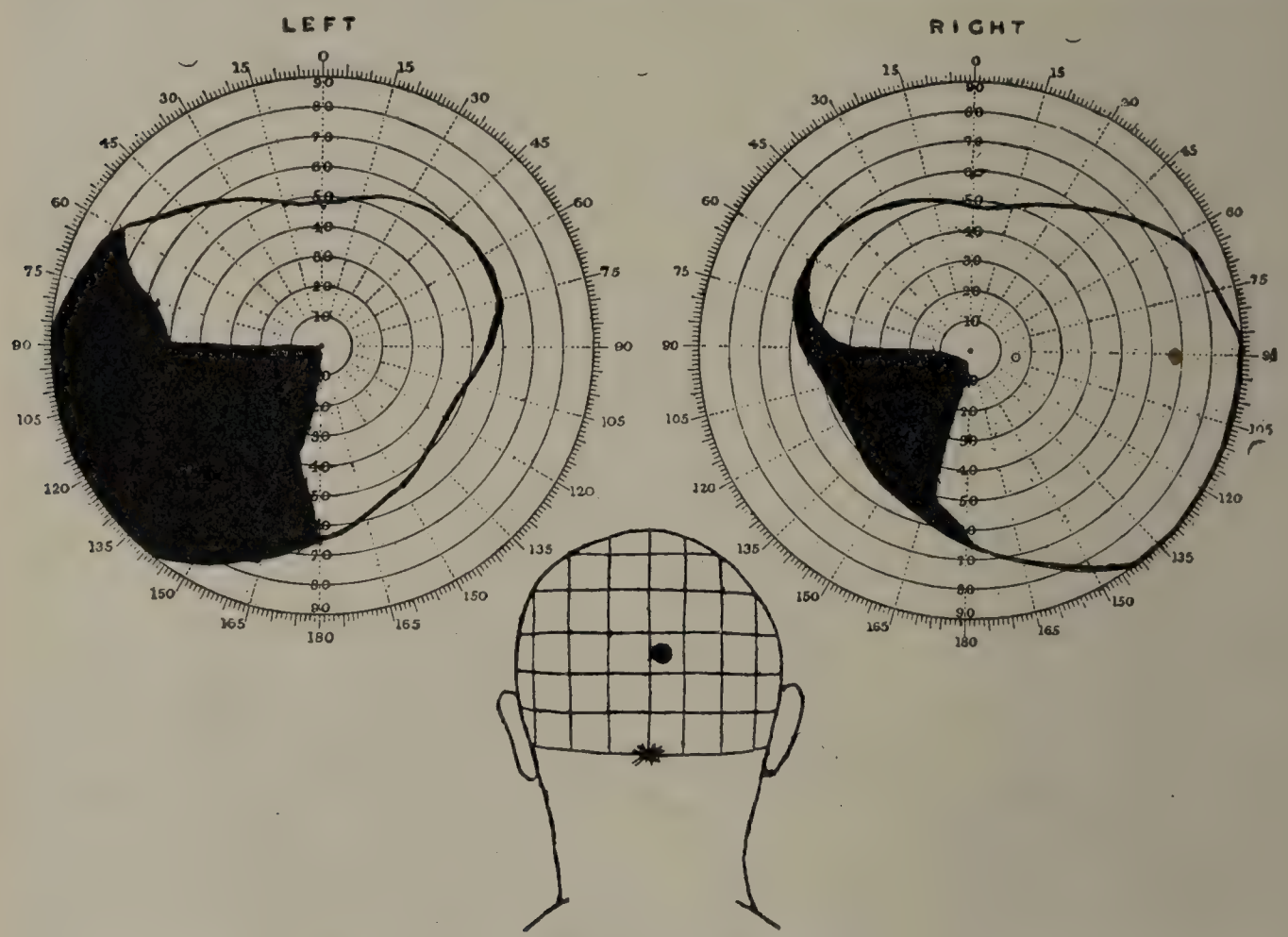


FIG. 169.—Showing loss of vision in lower left quadrant from bullet wound just above level of right calcarine fissure. (Lister and Holmes.)

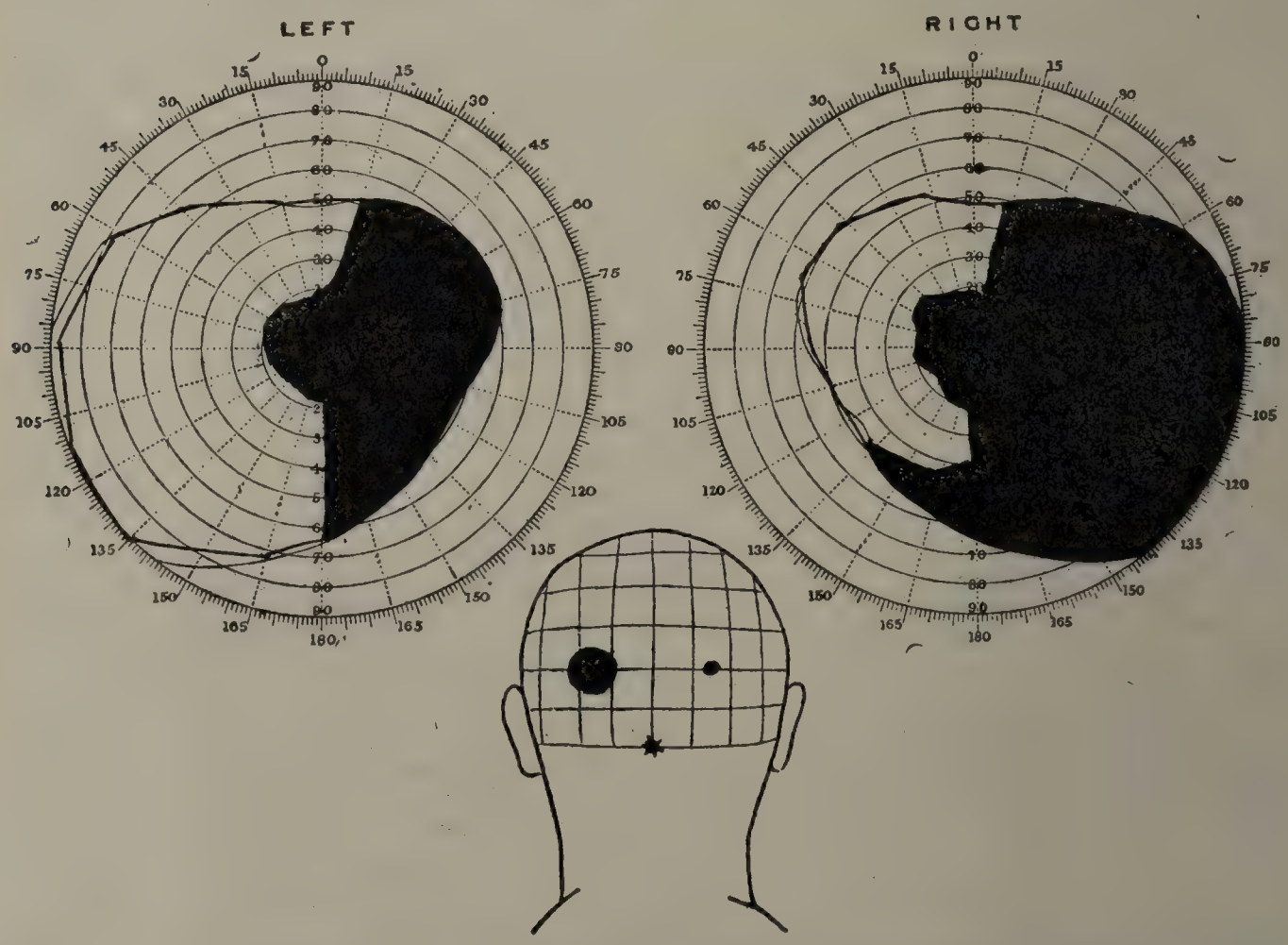


FIG. 170.—Homonymous hemianopsia with central scotoma from lesion of left optic radiations (occipital lobe) of one side and tip of occipital pole of other side. (Lister and Holmes.)

carine fissure (Fig. 169). In cases of homonymous hemianopsia with central scotomata the penetrating lesions caused injury to

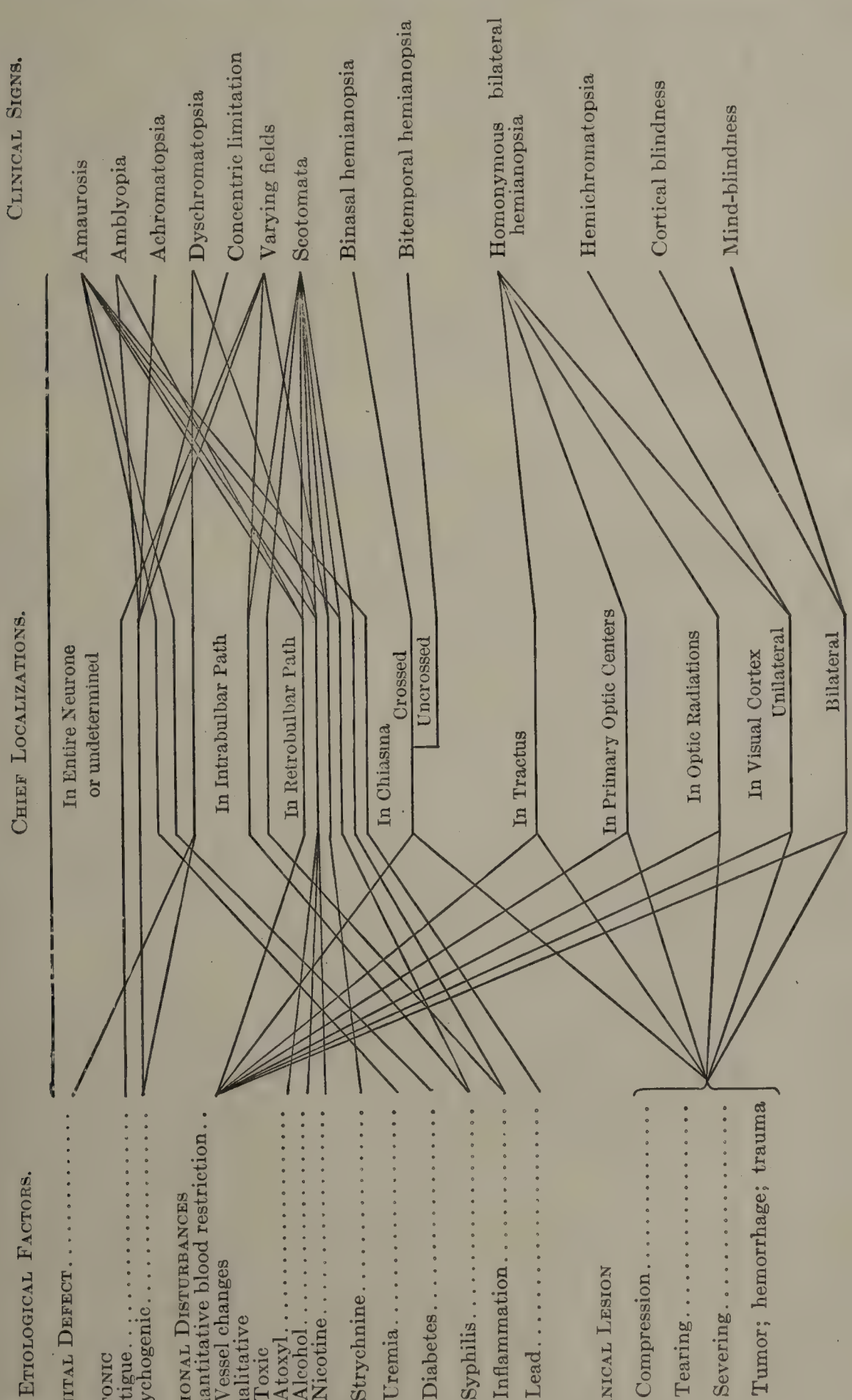


Fig. 171.—Summary of optical disturbances. (Veraguth.)

the occipital lobes or optic radiations of one side and passed through or near the tip of the occipital pole of the opposite hemisphere

(Fig. 170). Central vision is probably represented on either the mesial or the lateral surface of the posterior poles of the occipital lobes. The macular fibers are probably not represented bilaterally and are cortically localized in the posterior limits of the visual areas, probably on the margins and the lateral surfaces of the occipital lobes. It is probably because there is an overlapping of the middle and posterior cerebral arterial supply in this region that cerebral hemorrhage with visual defect so rarely involves the macular regions.

Annular scotomata are among the rarer anomalies seen in occipital lesions.

For further discussion of the cortical disturbances of vision from lesions of the temporal or occipital lobes see chapters on Aphasia, Syphilis of the Brain (Paresis), Brain Tumor, Hemiplegia, Thrombosis, Arteriosclerosis, etc.[1]

DISEASES OF THE OCULOMOTOR NERVES.

Ocular Nerves, Third, Fourth, Sixth.—Disorders of the sensorimotor functions of these nerves are best discussed under a general head, since the usual ocular palsies are often complex syndromes in which one or more of these nerves are involved. The vegetative functional disturbances are also usually associated.

The third nerve is a motor nerve for all of the muscles of the eyeball, save the external rectus, and the superior oblique, which latter received its motor fibers from the sixth and fourth nerves respectively. The third nerve also supplies the levator palpebræ, the ciliary muscle and the contracting fibers of the pupil. The dilating fibers of the pupil receive a branch from the sympathetic. Deep sensibility fibers also pass in the motor roots.[2] Numerous reflex pathways connect the eye muscles with other structures, notably the labyrinths.[3]

Third Nerve Palsies.—These are often very complicated and may be central or peripheral, complete or partial. Complete paralysis of both third nerves is rare, partial palsies are the rule. Unilateral palsy of all of the external muscles governed by the third nerve (often termed ophthalmoplegia externa) is due usually only to a lesion involving the second or peripheral motor neurons of the third nerve. Bilateral external ophthalmoplegia may occur also from lesions of the mesencephalon and cortical oculomotor paths.[4] The chief causes for central palsies are various types of encephalitis, polioencephalitis, either infectious poliomyelitis, influenza[5] or toxic as in alcoholism (Wernicke's polioencephalitis superior). Syphilitic thrombosis may cut off the blood supply of the nuclei. Pressure from the aqueduct above, or third ven-

[1] See Wilbrand and Sänger and Henschen in Lewandowsky's Handbuch, vol. **3.**

[2] Sherrington and Tozer: Proc. Royal Soc., 1910.

[3] See Bartels: Die Reflexbahnen f. d. Ohr.-Augenbewegungen, for recent discussion, Arch. f. Ophthalmol., 1926, **117**, 538. See also Discussion on Ocular Palsies, Proc. Royal Soc. Med., 1921, vol. **14**, Sect. Neur. and Ophthalmology. de Barenne and de Klijn: Ueber vestibuläre Augenreflexe, Arch. f. ophth., 1923, **111**, 374.

[4] Epidemic encephalitis gave rise to a host of complicated eye palsies. The literature is enormous. See Epidemic Encephalitis.

[5] Jelliffe: Loc. cit.

tricle may cause pressure palsies, usually of irregular distribution. (Nothnagel's Syndrome. Pineal Syndrome.)

Peripheral palsies are more frequently due to disease at the base, usually basal syphilitic meningitis, tumor, tuberculosis, hemorrhage, traumas (rarely), or are occasioned by involvement of the fibers as they pass through and about the red nucleus by tumor, multiple sclerosis, or when implicated in a thrombotic or hemorrhagic softening of the cerebral peduncle—Millard-Gubler, Benedict, Fovilles' syndromes, red nucleus syndromes. (See chapters on Midbrain for description of these syndromes, also see Fig. 174.) Infectious disease neuritis may also occasion peripheral palsies. Pressure from aneurism of the internal carotid, and thrombosis of the cerebral sinuses (sinus cavernosus) may also cause peripheral palsies. A special herpes zoster ophthalmicus is known. Exophthalmic goiter and diabetes are special causes. Transitory third nerve palsies occur in the disorder known as ophthalmoplegic migraine (*q. v.*).

Syndromes. — The compound character of the nuclei and the loosely arranged bundles making up the nerve explain the great range in symptomatology. The careful phyletic and histopathological studies of Brouwer[1] have enabled the student of the nervous system to better comprehend the anatomical relations. His schemes are here reproduced. (Fig. 173.)

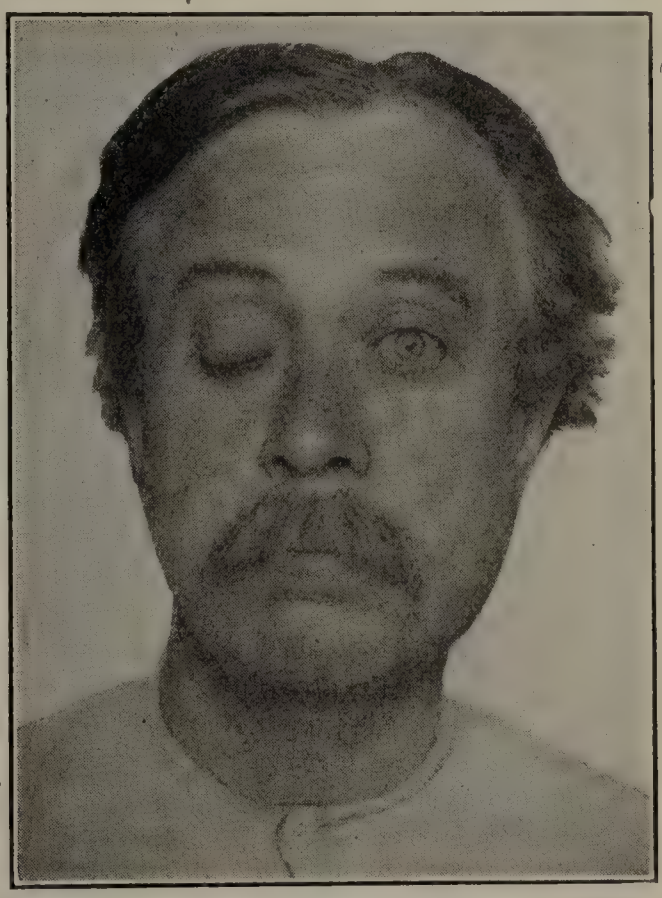
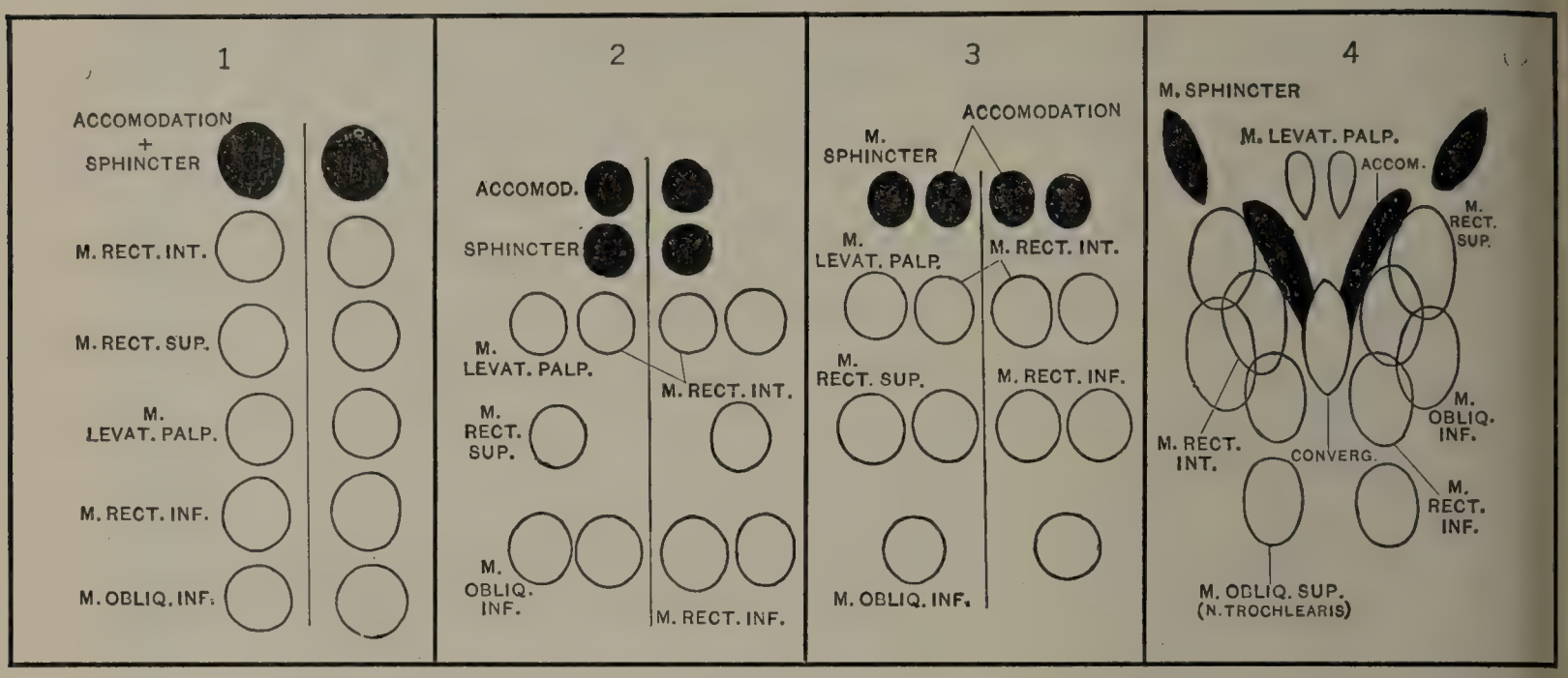


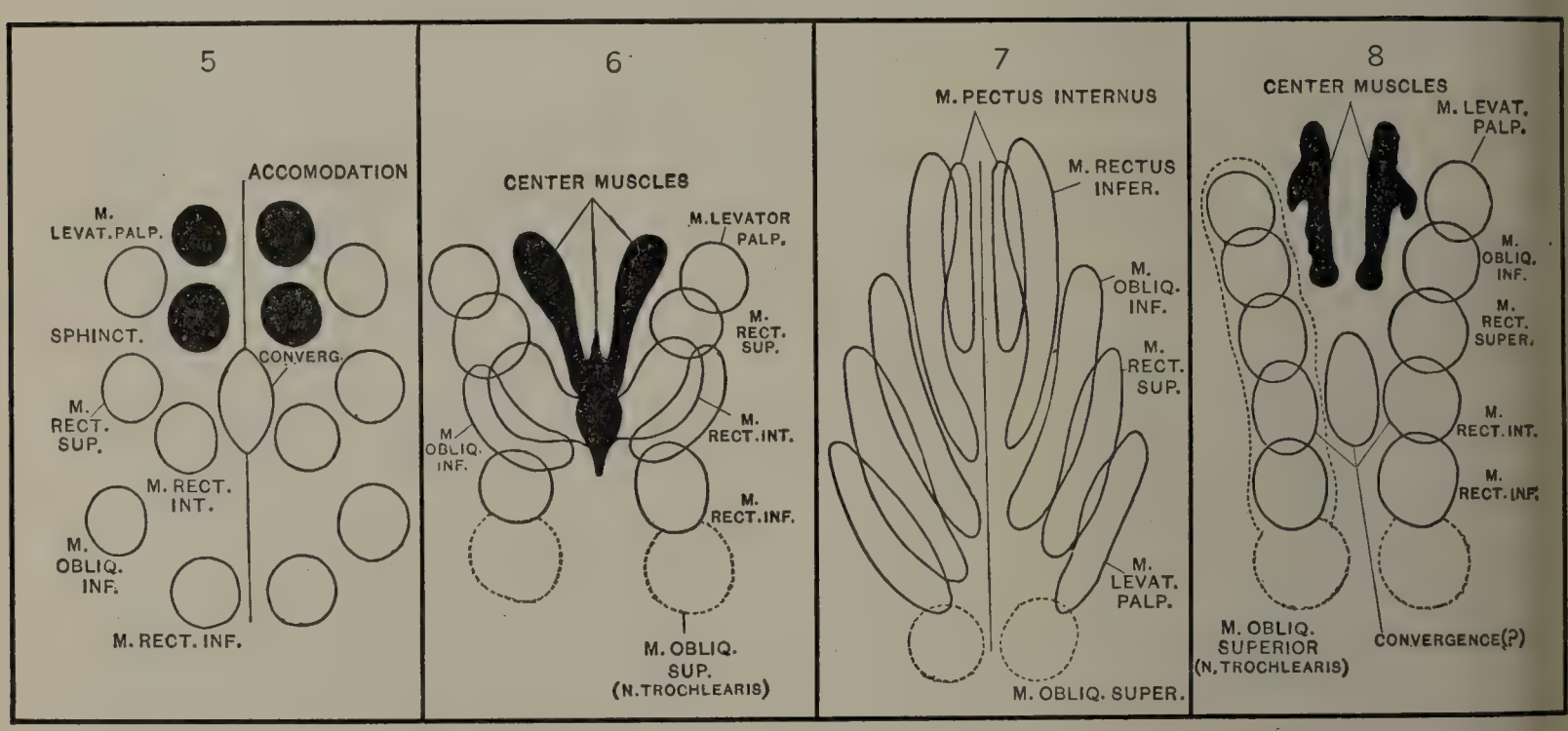
FIG. 172.—Cerebral syphilis ptosis. Third nerve palsy.

A complete unilateral palsy, probably nuclear (ophthalmoplegia completa), would then cause ptosis, wrinkling of forehead on same side (from effort to overcome ptosis by occipito-frontalis), wide pupil from involvement of the Edinger-Wesphal nucleus), irresponsive to light and accommodation, eye turned outward and slightly downward. Double vision is present and some dizziness in the early stages. A variety of individual muscle palsies may also result from either nuclear or peripheral involvement as indicated—ophthalmoplegia externa, when the pupil is not involved; ophthalmoplegia interna when only the internal muscles are involved—a rare condition.

The distinction of nuclear from peripheral palsies is usually made on the basis of accompanying symptoms—sensory or motor, due to implication of the red nucleus, or of the cerebral peduncles. In the absence of these accessory symptoms (Weber-Gubler, Benedict syndromes, rubrospinal syndromes) the distinction may be impossible.

[1] Brouwer: Zeit. f. d. g. Neur. u. Psych., 1918, **40**, 152.





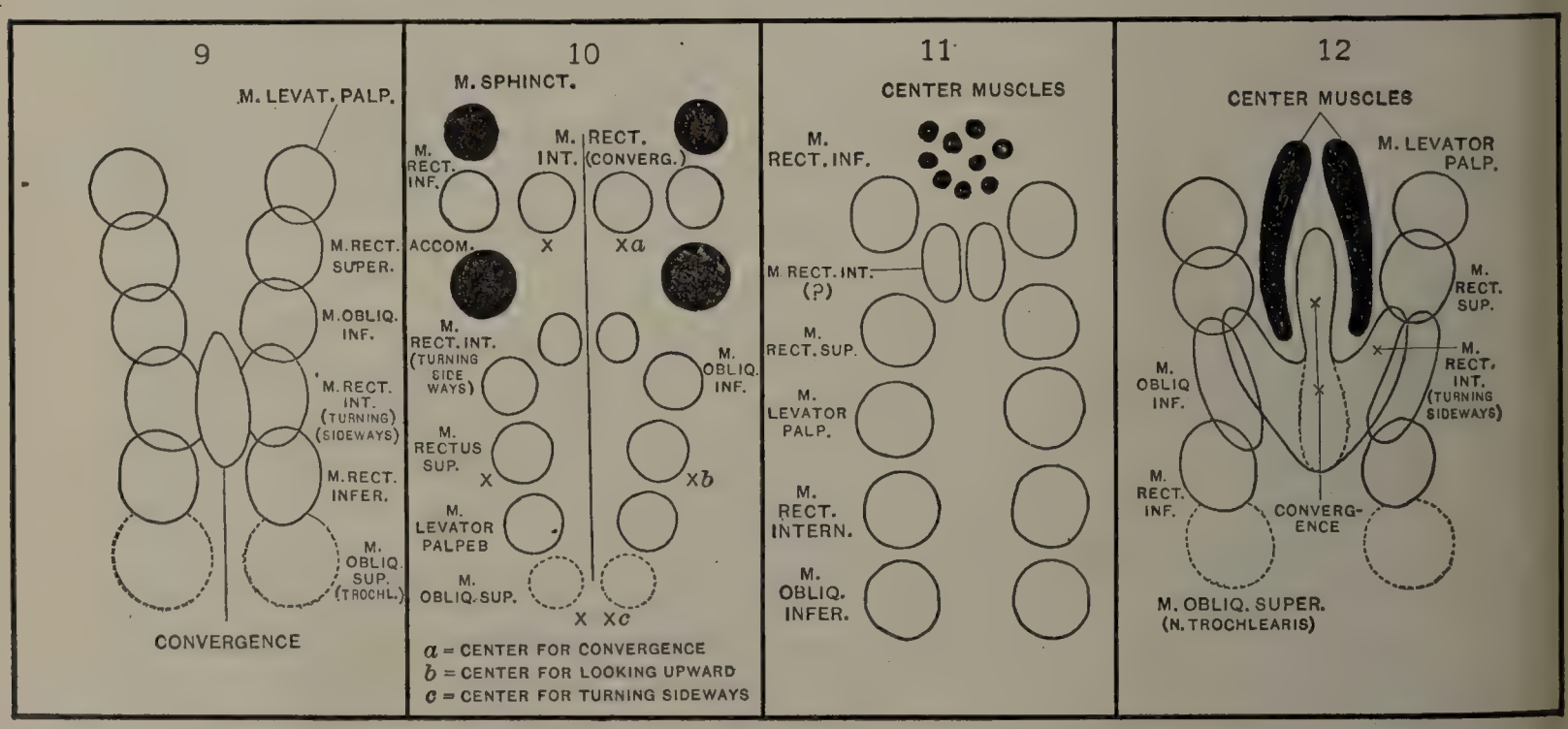


FIG. 173.—Scheme of historical development of ideas concerning the localization of oculomotor nuclei. (After Brouwer, loc. cit.) 1, Henschen u. Volkers, 1878; 2, Kahler and Pick, 1881; 3, Starr, 1888; 4, Knies, 1891; 5, Stuelp, 1891; 6, Bernheimer, 1897; 7, Bach, 1899; 8, Edinger, 1900; 9, Majano, 1903; 10, v. Monakow, 1905; 11, Tsuchida, 1905; 12, Brouwer, 1918. Center muscles = Binnenmuskeln = accommodation and vegetative synapses.

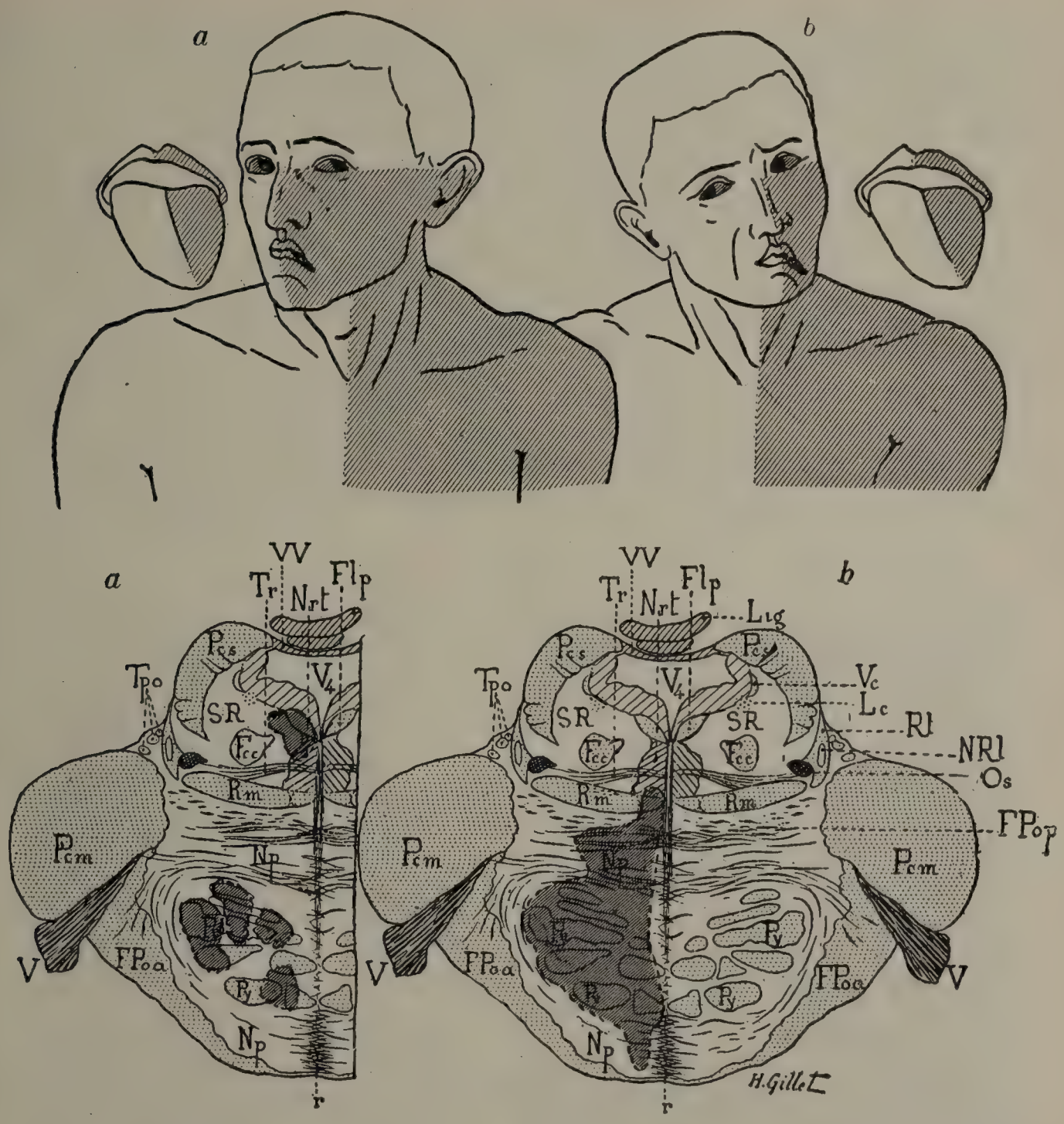


FIG. 174.—Fovilles' syndromes, with anterior and posterior pontine syndromes. Hemiplegia, cerebral type, with (*a*) conjugate deviation of the head and eyes, (*b*) by lesions of the upper portion of the pons, right side, involving the anterior portion of the pons and the region of the tegmentum. On the left side there is a contralateral hemiplegia of the limbs, of the lower part of the face and of the tongue, because of the involvement of the pontine pyramidal fibers *Py* (corticospinal pyramidal fibers, corticonuclear facial and hypoglossal fibers). In *C*, right-hand figure, there is a single lesion which involves the tegmentum at its antero-internal angle and destroys the head-turning (cephalogyric) and eye turning (oculogyric) fibers of the right side which at this level are situated in the pes lemniscus and the internal portion of the median fillet giving rise to conjugate deviation of the head and of the eyes. By reason of the predominant action of the antagonists the head is inclined to the right and the eyes look to the right, the patient looks to the side of the lesion. In *a* there are multiple isolated lesions. Four large foci in the anterior portion destroy the pontine pyramidal fibers with a resulting crossed contralateral hemiplegia of the extremities, the face, and the tongue. Another focus occupies the posterior internal portion of the tegmentum and destroys the internuclear oculogyric fibers of the posterior longitudinal bundle which directly unites the nuclei of the sixth and third nerves and *vice versa*. There results a paralysis of the eyeballs by which they cannot turn sidewise toward the right—right oculorotary paralysis—by reason of the predominance of the antagonists the patient looks to the left. The patient looks away from the lesion toward the paralyzed members. The cortical oculorotary fibers and the pes lemniscus are intact. For details of structure and abbreviations, see chapter on Midbrain Lesions. (Dejerine.)

There is no single disease process to which the term ophthalmoplegia may be rigidly applied. Hence there is no general course and no general treatment. The various palsies must be interpreted on the basis of the dynamic factors, and the treatment must be founded upon the causation. Syphilis is responsible for the majority of these palsies, and calls for verification by the cytobiological tests and prompt antisyphilitic treatment, best by arsphenamine and hypodermic injection of mercury. (See chapter on Syphilis of the Nervous System.)

Isolated involvement of the pupillary apparatus may be discussed here. Contracted pupils, irregular pupils, unequal pupils, dilated pupils, etc., have been discussed (see Symptomatology). A dilated pupil with loss of accommodation reflex is infrequently seen in severe alcoholism (Korsakov's syndrome). It may be present also in optic nerve disease—combined with loss of right reflex as well. A loss of



FIG. 175.—Third nerve paralysis. Ptosis and external strabismus.



FIG. 176.—Cerebral syphilis. External rectus palsy, right eye.

light reflex with preservation of the accommodation reflex (reflex iridoplegia, Argyll-Robertson pupil) is a frequent sign of syphilis. Its mechanism has been discussed. It is often unilateral in the beginning of a tabes or paresis, or other type of cerebrospinal syphilis and may occasionally occur in a number of other conditions, such as following gunshot wounds of midbrain, and brain tubercle, quite frequently in epidemic encephalitis, etc. Pupillary irregularity is frequent in pulmonary tuberculosis (vegetative),[1] cerebral syphilis usually becomes double.

[1] Fanelli, F.: Rif. Med., 1923, p. 991. Spiegel: Zur Lokalisation d. Argyll-Robertsonschen Phänomens, Wien. klin. Wchnschr., 1925.

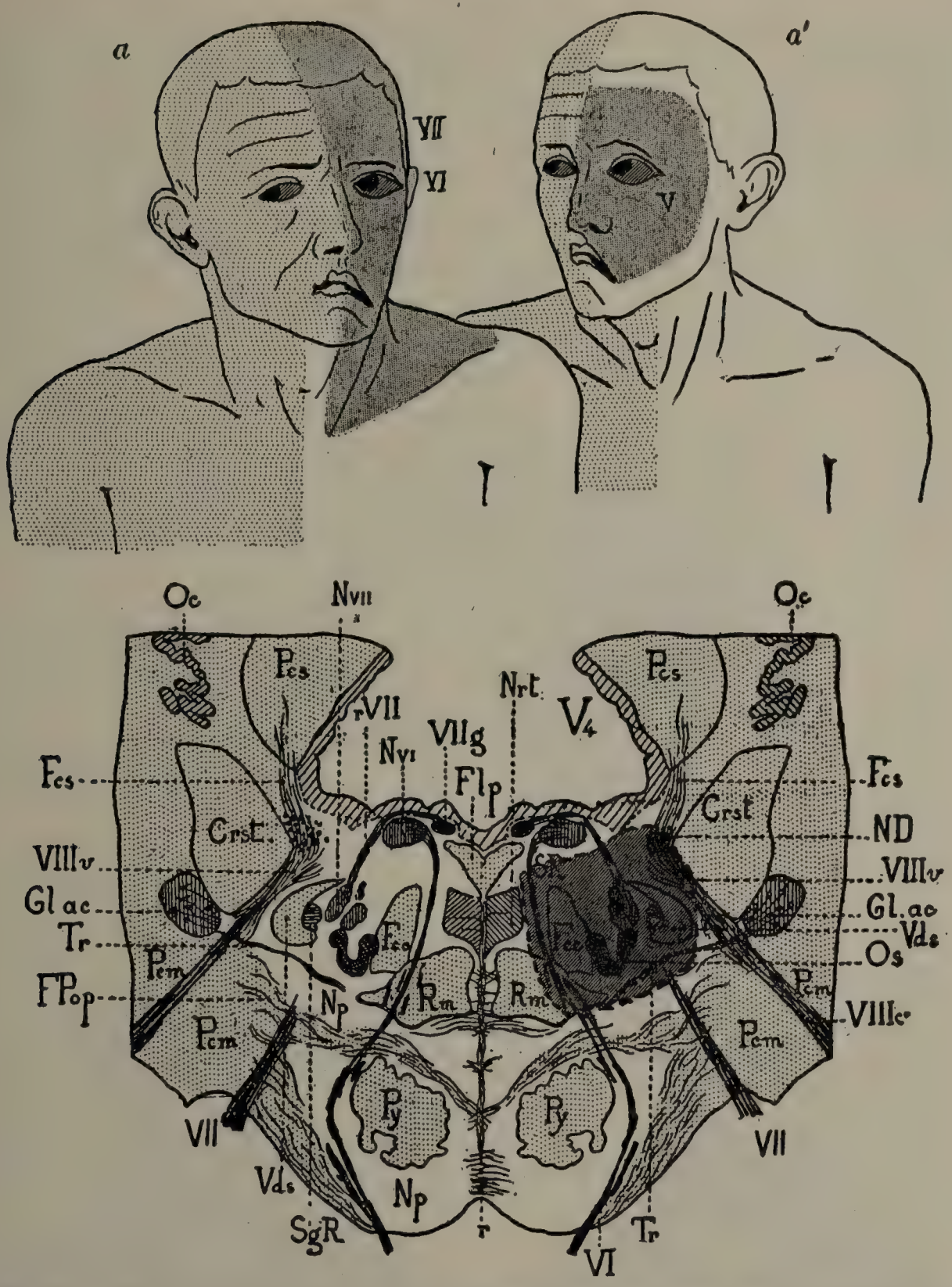


FIG. 177.—Pontine syndrome, with eye palsies of central origin and syringomyelic dissociation. There is here a crossed hemianesthesia with alternating paralysis of the VI and VII cranial nerves, anesthesia of the V nerve due to hemorrhage in the lateral and lower portion of the pontine tegmentum of the left side. The *right-hand figure* shows the hemianesthesia, dissociated as in *syringomyelia* (hemianalgesia and hemithermanesthesia due to lesion of the crossed sensory pathways of the lateral portion of the reticular formation). There is preservation of the tactile and postural sensibilities and of the stereognostic sense, because of the incomplete extension of the lesion to the median lemniscus (*Rm*). The *left-hand figure* shows (1) atrophic paralysis of the VII nerve with reaction of degeneration, lagophthalmia, drooping of the lips, loss of facial mimicry, paralysis of the entire left facial (VII) indicated (Fig. *a*); (2) anesthesia of the face, following involvement of the descending root of the trigeminus (see V on *a'*); (3) paralysis of the external rectus with convergent strabismus by reason of the overaction of the antagonists. Furthermore, there is a paralysis of the lateral movements of the eyeballs toward the left notwithstanding the integrity of the posterior longitudinal fasciculus (*Flp.*), of the nucleus of the VI and of the adjacent reticular formation. The lesion of Deiters's nucleus, and of the labyrinthine oculorotary fibers which unite Deiters's nucleus (*ND*) to the nuclei of the III and VI causes this. By reason of the overaction of the antagonists the patient looks to the right. (After Dejerine.) For abbreviations on the anatomical sketch see section on Midbrain and Plate X.

Isolated ptosis may also be seen. This may be due to oculomotor invasion (tabes), or it may be hysterical. In the former case there is usually a compensatory overaction of the corrugators, in the latter this is less likely to take place. Sympathetic disease, ptosis from palsy of Müller's muscle, is usually accompanied by pupillary disturbances and the eyeball lies somewhat retracted in the eyeball cavity. The cervical sympathetic is here implicated. Hypermetropia is the usual condition of the child at birth. At three emmetropic vision is the rule. Persistence of the hypermetropia is a vegative neurological sign and is frequent in vagotonic individuals.

Progressive myopias and hypermetropias may have psychogenic causes of far reaching importance. Some of these coming on or about puberty, often attributed to excussive study or reading are of great interest from the psychoanalytic point of view. They often point to definite unconscious regressive situations. In three progressive myopios under observation (J) two were stationery. One, as part of an unconscious homoerotic fixation, developed a mild diabetes mellitus, another a marked myasthenia; a third a progressive myopia, showed a typical paranoid formula chiefly compensated for by the eye situation.[1]

Chronic Progressive Eye Palsies.—These make up a special group, occasionally congenital, more often they are a part of a progressive anterior poliomyelitis. (Bulbar palsy, *q. v.*)



FIG. 178.—Paralysis of the fourth nerve. Inability to look down. It will be noted that as the eyeball does not turn downward the eyelid does not descend; at the same time the patient can close his eyes when told to do so. (Russell.)



FIG. 179.—Paralysis of the fourth nerve. The same patient closing his eyes at command. (Russell.)

Fourth Nerve Palsy.—The fourth (trochlearis) nerve supplies the superior oblique muscle with its motor fibers. Afferent fibers carry deep sensibility fibers from the muscle. The fibers are crossed and uncrossed, the latter being phylogenetically the first to appear, but later are overshadowed by the crossed fibers. Isolated palsy causes a marked diplopia, and some dizziness when the patient looks down-

[1] Jelliffe: Myopia as Castration Symbol, Int. Zeit. f. Psa., 1926.

ward and outward. The false image stands lower and nearer than the true one, its upper end inclined toward the true image. Looking upward or downward causes no diplopia. These patients have difficulty in descending stairs, and they incline the head forward and toward the sound side to adjust to their diplopia.

The fourth nerve is frequently involved with the third and sixth in basal inflammations or new growths, or may be involved independently from pressure in the posterior fossa (cerebellar tumor). It also is involved at its nuclear origin from encephalitis, poliomyelitis, toxemias, etc. (Figs. 178 and 179.)



FIG. 180.—Paralysis of upward movement of the eyes, showing the excessive wrinkling of the forehead in the attempt to look up. Skew deviation. (Holmes.)

Sixth Nerve Palsy.—This is perhaps the most frequent of the eye palsies. The peripheral motor neuron is exposed for three-fourths of an inch or more on the base of the skull, and is therefore subjected to greater possibility of local pressure than any other cranial nerve. When the external rectus muscle is paralyzed there is an internal strabismus and a slightly smaller pupil in the affected eye. There is also a diplopia.

Sixth nerve palsy more usually results from basilar disease, fracture of base, meningitis (syphilitic and other types), tumors not only of the base but also of the brain substance itself. Nuclear involvement is seen in encephalitis, poliomyelitis, toxemias, etc. External rectus palsy as a result of a myositis is not unknown. It may also be a complication of severe migraine (periodic palsy).

Central Motor Neurons.—Isolated eye palsies are due to nuclear or to peripheral involvement of the third, fourth and sixth nerves.

EXPLANATION OF PLATE X.

The Oculorotary Paths and in Particular the Innervation of the Muscles which Turn the Head.

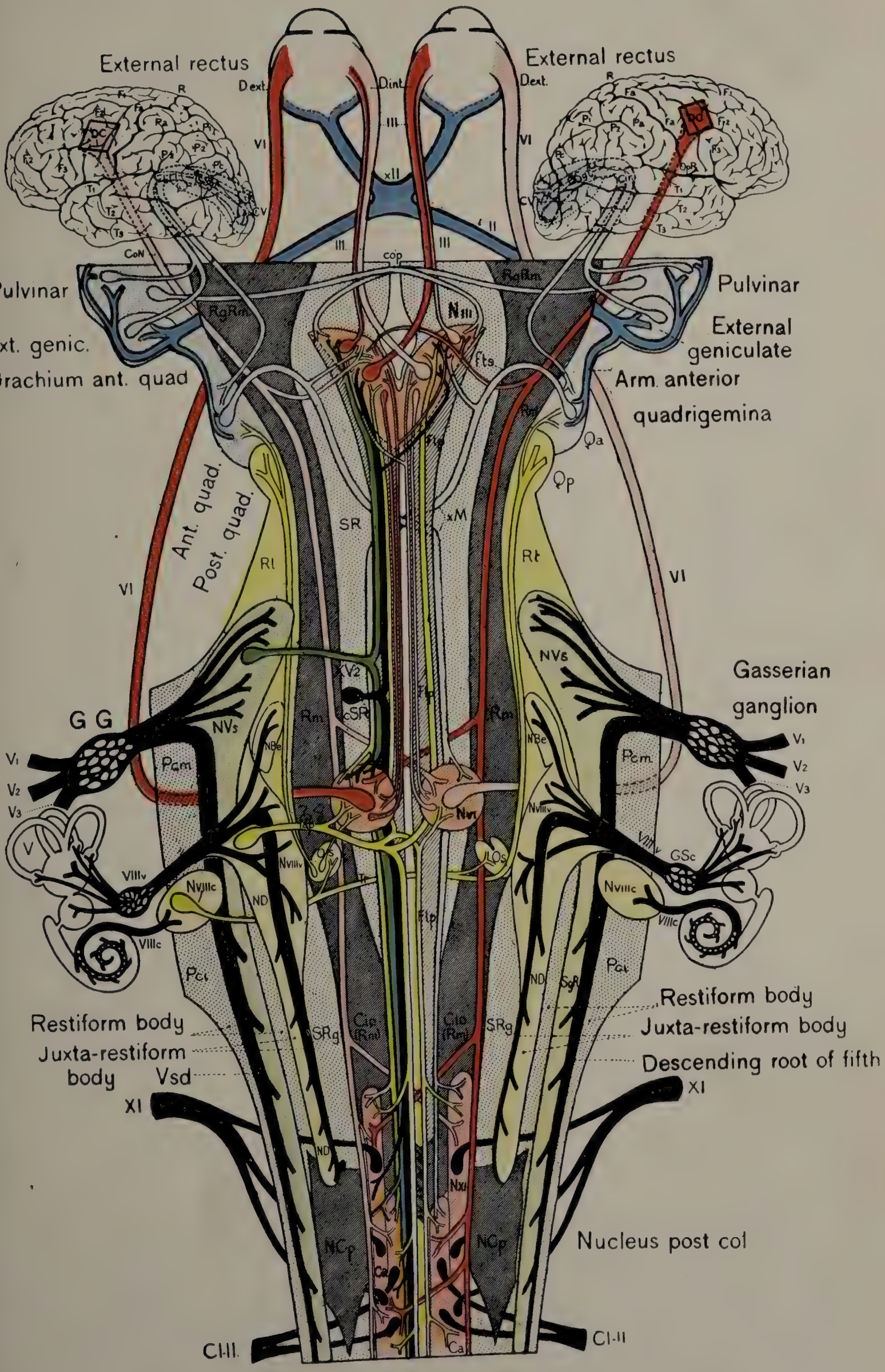
Abbreviations.—BrQa, arms of anterior corpora quadrigemina; *C*, the cochlea with spiral ganglion, the cochlear branch of the *VIII* pair; *Ca*, anterior horn of the spinal cord; *Cge*, external geniculate; *Cj*, juxta restiform body; *Cio(Rm)* interolivary region of the medulla containing the median lemniscus; *Cirl*, retrolenticular segment of the internal capsule; *CoN*, corticonuclear contingent of the pyramidal tract, *CSgt*, sagittal view of the occipitotemporal lobes; *CV*, visual area of the internal face of the hemisphere, transparent view; *CI–II*, first and second cervical pair; *DC*, center of conjugate deviation of the head and of the eyes; *Dext*, right external rectus muscle; *Dint*, right internal rectus muscle; *Fa*, ascending frontal convolution; F^1, F_2, F_3, three frontal convolutions; *fap*, posterior and internal arcuate fibers of the medulla; *Flp*, posterior longitudinal fasciculus; *fts*, tectospinal fibers; xV_2, central or secondary trigeminal pathway; *GG*, Gasserian ganglion; *G, Sc*, Scarpa's ganglion; *NBe*, Bechterew's nucleus; *ND*, Deiters' nucleus; *NVs*, sensory trigeminus nucleus; *NIII*, oculomotor nucleus; *III* pair; *NVI*, nucleus external rectus; *NVIIIc*, anterior terminal nucleus of the cochlear; *NVIIIv*, triangular nucleus of the vestibular; *NXI*, spinal accessory nucleus (trapezius-sternocleidomastoid); *Os*, superior olive; P_1, P_2, superior and inferior parietal lobe; *Pa*, ascending parietal convolution; *Pc*, angular gyrus; *Pci*, inferior cerebellar peduncle; *Pcm*, middle cerebellar peduncle; *Pul*, pulvinar; *Qa, Qp*, anterior and posterior corpora quadrigemina; *R*, fissure of Rolando; *RgRm*, region of median lemniscus; *Rl*, lateral lemniscus; *Rm*, median lemniscus; *SgR*, gelatinous substance of Rolando; *SR*, reticular substance; *SRg*, gray reticular substance; T_1, T_2, T_3, temporal convolutions; *Tr*, trapezoid body; *V*, vestibular nerve; V_1, V_2, V_3, three branches of the trigeminus, ophthalmic, superior and inferior maxillary; *Vsd*, descending root of the trigeminus; *xM*, tegmental crossing of Meynert; *XII*, optic chiasm; *III*, oculomotor; *VI*, external rectus; *VIIIc*, cochlear branches of the auditory; *VIIIv*, vestibular; *XI*, spinal accessory.

The tegmentum in its medullary, pontine, peduncular portions seen in projection at the level of the aqueduct of Sylvius and the fourth ventricle, with the reticular formation (*SR*), the posterior longitudinal fasciculus (*Flp*) and the median lemniscus (*Rm*). It is limited laterally by the lateral lemniscus (*Rl*), colored in yellow, and the long sensory nuclei of the trigeminus (*V*) and of the auditory (*VIII*) nerves (*NVs, SgR*) colored green, and in yellow (*NVIIIc, NBe, NVIIIv, ND*) and showing each side of the median line: (1) above the nuclei of the *III* pair (*NIII*) which innervates by crossed and by direct fibers the internal rectus of the eye; in the center, the nuclei of the *VI* pair (*NVI*) which innervates the external rectus of the eye, and (3) below, the cephalorotary nuclei which act to rotate and incline the head and neck; spinal nuclei and spinal accessory *NXI*, and motor centers of the cervical cord (*Ca*).

Myelinated early are the fibers which unite the nuclei of the sixth and of the third pair and of their associated fibers to enable the lateral movements of the eye to take place in the early stages of life. These *internuclear* fibers, colored in red, take their origin from small cells in the nuclei in the oculomotor, *III* and abducens *VI*, and pass by means of the posterior longitudinal fasciculus; the small ganglion cells of the nucleus of the left *VI* pair for example, can put into action the crossed and direct root fibers going to the left internal rectus; and at the same time the ganglion cells of the nucleus of the *III* left pair, can put into action the root fibers of the homolateral external rectus of the same side (left). Thus there is established a strict physiological association, permitting the action of a dextrorotary system, turning the eyes toward the right, or a levorotary system, turning the eyes to the left, an association which can be incited and activated possibly by the cortex, or by various sensory or sensorial paths, labyrinthine, tactile or optic.

1. The cortical oculorotary pathway (colored dark red on the right, pale red on

PLATE X


External rectus
External rectus
Dext.
Dint.
Dext.
III
VI
VI
XII
CV
CV
III
III
CoP
CoN
Pulvinar
Pulvinar
RgRm
RgRm
External geniculate
Ext. genic.
Brachium ant. quad
Arm. anterior quadrigemina
fts
Ftg
Qa
Qp
Ant. quad.
Post. quad.
SR
xM
Rl
Rt
VI
VI
XV2
NVs
Gasserian ganglion
G G
Rm
cSR
NBe
Pcm
V1
V2
V3
Nvi
Nviiiv
GSc
Nviiic
ND
Flp
Pcl
SgR
Restiform body
Juxta-restiform body
SRg
Cio (Rm)
Descending root of fifth
Vsd
XI
XI
Nxii
NCp
Nucleus post col
Ca
Cl-II
Cl-II

the left) belongs to the corticonuclear path (*CoN*) (see Plate I, *O*) and takes its origin from the center of deviate conjugation of the head and eyes (*Dc*) situated at the juxtaposition of the second and ascending frontal regions. It passes by way of the knee of the internal capsule, internal portion of the cerebral peduncles, descends with the aberrant fibers of the pyramidal tracts (see Plate I, *D*) in the median lemniscus and passes to the nuclei of the *III* pair, to the nuclei of the *VI* pair of the opposite sides, and to the cephalorotary nuclei of the two sides.

2. The labyrinthine oculorotary pathway, colored yellow, contains a vestibular oculorotary path, for static equilibrium and a cochlear, or acoustic, oculorotary pathway. The vestibular oculorotary pathway takes its origin in the cells of the terminal nuclei of the vestibular root (*NBe, NVIIIv, ND*). Its fibers belong to the posterior and interior arcuate fibers of the medullopontine tegmentum (*fap*). They actionate the two *VI* nuclei and give to the posterior longitudinal fasciculus (*Flp*) direct and crossed, ascending and descending fibers. The ascending crossed and the descending direct fibers are the most numerous. The ascending fibers actionate the nuclei of both *III* pairs; the descending fibers actionate the head-turning nuclei.

The acoustic rotary pathway is represented by the cells of the anterior nucleus of the acoustic (*NVIIIc*), the trapezoid body (*Tr*), the fibers destined to the superior olive of the same side (*Os*) and of the crossed side and the fibers which go in the lateral lemniscus (*Rl*). This pathway can incite the oculorotary system by the fibers of the hilum of the superior olive which actionate (a) the motor cells of the external rectus (*VI*), (b) the motor cells of the oculomotor (*III*) acting on the internal rectus of the opposite side by the intermediation of small cells of the intranuclear oculorotary system. The labyrinthine oculorotary pathways can be activated not only by peripheral sensations—equilibrium (vestibular), auditory (cochlear), but also by the cerebellum, by reason of the system of internal fibers of the semicircular canals and cerebellovestibular fibers which terminate in Bechterew's and Deiter's nuclei. These connections are not figured in the plate.

3. Lateral movements of the eyeballs can be induced by sensory stimuli acting on the skin of the head and body. The central trigeminal oculorotary pathway (green) takes its origin in the nuclei of the *V* pair (*NVs*), passes by the arcuate fibers of the medullopontine tegmentum and the posterior longitudinal fasciculus, and then is divided into ascending and descending fibers which actionate the oculomotor nuclei (*III* and *VI*) and the cephalorotary nuclei (*XI*, ant-horn cells *Ca*). The central sensory pathway (black) can actionate the oculorotary and cephalorotary nuclei by fibers which originating in the reticular formation (*cSR*) ascend and descend in the posterior longitudinal fasciculus.

4. The visual oculorotary pathway, tecto-spinal (*fts*) (light blue), leaves the anterior corpora quadrigemina, crosses the middle line at the level of the dorsal decussation of Meynert (*xM*) descends in the prelongitudinal fascicle, giving off terminal and collateral fibers to the nuclei of the *III* pair of the homolateral side, to the nuclei of the *VI* pair and to the cephalorotary nuclei (*NXI,Ca*) of the opposite side.

It is actionated in part by the peripheral visual pathway (retina, nerve, chiasm) (*xII, II*) (dark blue) and in part by the central visual pathway in the calcarine fissure (visual center) and the anterior corpora quadrigemina. It can also be indirectly stimulated by the efferent visual pathway, which originating in the primary optic centers (*Pul,Cge*) irradiates in the calcarine cortex, passing by way of the retrolenticular segment of the internal capsule (*Cirl*) and the optic radiation of the occipitotemporal lobes.

A destruction of the cortical oculorotary pathway (right) for example, determines a paralysis of the levo-oculorotary and levocephalorotary systems; that is to say, a conjugate deviation of the head and of the eyes to the opposite side. A lesion of the labyrinthine oculorotary pathway, left, will also determine a paralysis of the levo-oculorotary system by abolition of the lateral movements of looking of the direct homolateral side. In both instances the patient looks to the right because of the predominance of the antagonists (Dejerine).

Supranuclear disease of the oculomotor paths does not result in the loss of function of a single eye, much less of a single eye muscle. Such lesions between the oculomotor cortex and the nuclei in the midbrain cause complicated disorders of the *associated movements* of the eyes. The most frequent of these are: (*a*) conjugate deviation, (*b*) lateral associated palsy, (*c*) vertical associated palsy, (*d*) loss of convergence, (*e*) central nystagmus, (*f*) irregular types. (See Plate X.)

(*a*) *Conjugate Deviation.*—Here both of the eyes are directed to the side of the lesion, and cannot be voluntarily moved in an opposite direction. Yet, if the eyes are fixed upon an object and the head is turned away from the lesion, the eyes will turn in the direction which voluntarily is impossible. This is termed conjugate deviation of the eyes and head. The eyes' axes may not be truly parallel, but may diverge slightly. In acute apoplexies this symptom is occasionally seen—*i. e.*, forced deviation of the head to the side of the lesion. (See Fig. 174.)



Fig. 181.—Trochlearis palsy in cerebrospinal syphilis.

Lesions of the inferior parietal, angular gyrus, and possibly the foot of the second frontal gyrus may produce or occasion this type of forced position of the eyeball.

Lesion of the centrum ovale, and of the internal capsule involving the projection fibers of the oculomotor may cause conjugate deviations, here associated with hemiplegia as a rule. The chief lesions causing conjugate deviation are: hemorrhage or softening, abscess, encephalitis, occasionally tumor.

(*b*) *Lateral Associated Palsy.*—Here the eyes are unable to pass the middle line. The altered position of the head as seen in conjugate deviation is absent and movements of the head are unavailing in bringing the eyes past the middle line. Convergence, however, may remain intact. Certain incomplete conjugate deviations are found here.

Lateral conjugate palsy is usually due to a pontine lesion on the side of the palsy, and which implicates the abducens fibers near the nucleus, and the synapses of the posterior longitudinal bundle, possibly Lewandowsky's tractus pontis ascendens. Pressure at a distance may also occasionally cause a lateral conjugate palsy.

Fractures, pontine tumors, multiple sclerosis, softening (after labyrinthine infection) are among the causes of this comparatively rare condition.

(*c*) *Vertical Associated Palsy.*—Here the motion of both eyes is hindered only on looking up or down—all other associated movements are possible. When there is loss of ability to look down usually the eyelids do not descend as they normally do. In some patients the palsy is not symmetrical, one eye moving up or down more than the other.

Certain forced positions of the eye, one being higher than the other—(Magendie-Hertwig position) may be mentioned here. This syndrome often points to implication of the middle cerebellar peduncle. The side involved is indicated usually by the lower-lying eye. There is also nystagmus.

In the majority of the cases lesions have been found implicating the corpora quadrigemina (pineal) either directly or by tumor, or by direct pressure. So-called hysterical cases are usually mistakes in diagno-

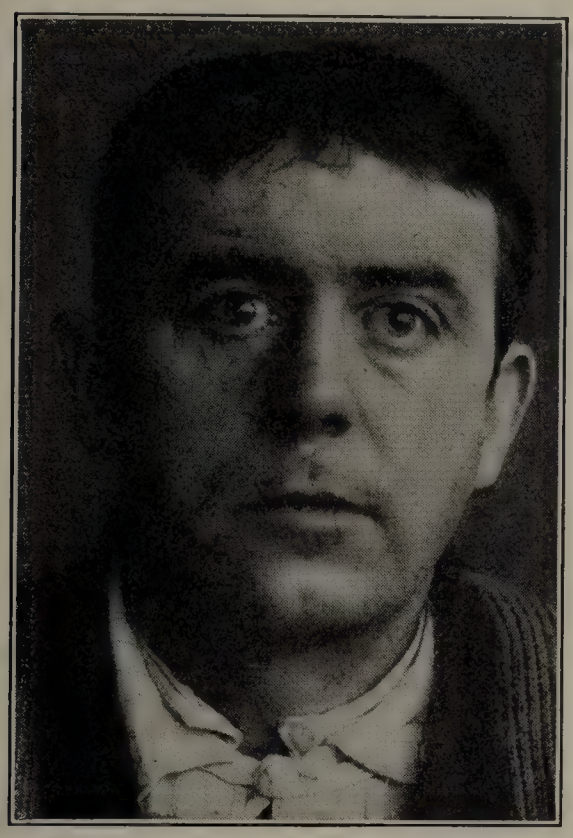


FIG. 182.—Inequality of pupils. Left pupil larger than right. Cerebral tumor. Immobile.

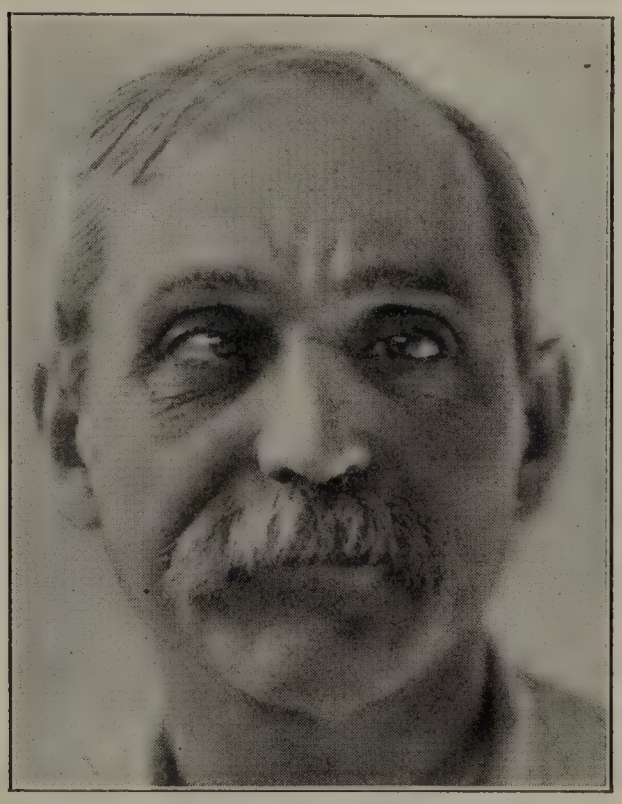


FIG. 183.—Tabes. External rectus palsy.

sis. One such of Lewandowsky's proved to be a cysticercus of the corpora quadrigemina. A personal case showed a sarcoma of the third ventricle pressing upon the anterior corpora quadrigemina.

Oculogyric spasms,[1] usually upward and to right, are occasionally seen in chronic encephalitis cases. The precise mechanisms are still unknown. Symbolic factors may play a rôle.

(*d*) *Paralysis of Convergence.*—As an isolated symptom this is rare, it is usually accompanied by other associated palsies. It is found in multiple sclerosis and encephalitis. A closely related phenomenon—weakness of the internus muscles, Möbius' symptom in exophthalmic goiter, is thus far difficult of explanation.

[1] Ballet, 1907; G. Lévy, Encephalitis, 1921; Wimmer, Chronic Encephalitis, Acta. Neurolog. Skand, 1927, vol. **2**; Wilder and Silbermann. Beiträge z. Tic Probleme, Berlin, 1927, p. 70.

(*e*) *Central Nystagmus.*—The extremely complicated subject of central nystagmus is more fully discussed in the section on Vestibular Disease. When rhythmic, *i. e.*, possessing a quick and a slow excur-

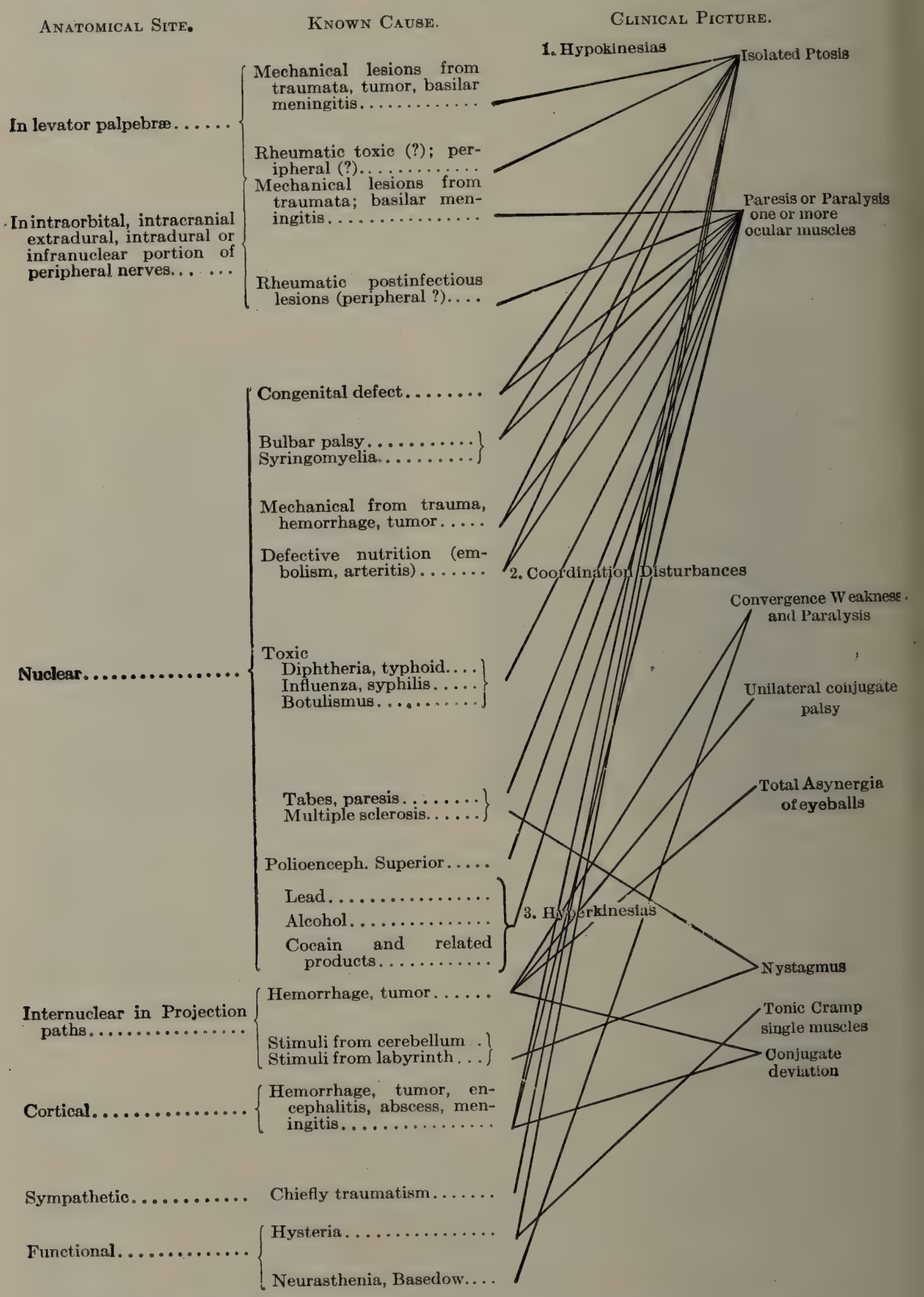


Fig. 184.—Summary of disturbances of external ocular nerves. (Veraguth.)

sion, it is usually vestibular. Undulating nystagmus, *i. e.*, with uniform backward-and-forward movements, is more apt to be due to involvement of the central or peripheral eye muscle nervous pathways. Possibly vestibular associations must always be involved. Undulating nystagmus is occasionally seen in severe fatigue, in myasthenia. in progressive muscular atrophy, alcoholism, hydrocephalus, etc. Undulating nystagmus as well as dissociated eye movements are normal in infants, and are frequently seen in congenital defects (idiots, imbeciles, congenital blindness).

(*f*) *Irregular Palsies.*—Dissociated eye movements in which the eyes move irregularly, each according to its wish, as in crustacea, in the very young infant, in congenital defective developments, is seen coming on in adults from destruction of the associative mechanism of the eye movements, more particularly from separation of the nuclei of the oculomotorius. Lesions which cut the nuclei apart one from another (multiple sclerosis, epidemic encephalitis, tumor) will cause this asynergia or ataxia of the eye.

Skew deviations are conditions in which one eye is directed outward and downward, the other inward and upward. Such a compulsory eye position is usually due either to a middle cerebellar peduncle affection or to a cerebellar lesion elsewhere.

Eyeball apraxias, so-called, or ideomotor dissociated movements offer certain complex analogies with similar disturbances of the tongue muscles in speech, the facial muscles in mimicry, or the arm muscles in expression. They are usually due to lesion of the projection fibers in the centrum ovale or internal capsule.

Psychogenic dissociation of the ocular movements frequently occurs. The oculogyric crises of postencephalitis may belong here.

Treatment.—The treatment of these various syndromes depends entirely upon the causative factors. These have been discussed under the respective syndromes. Also see chapter on Midbrain Disorders.

DISEASES OF THE TRIGEMINAL NERVE.

Fifth or Trigeminal Nerve.—The symptomatology of lesions of the fifth nerve is diverse, as it has both a sensory and a motor part, and has many synaptic junctions with cranial, spinal and vegetative nerves.[1]

Motor Part.—The cortical synapses of the motor part are bilateral, and are located in the lower third of the central convolution. From here the fibers pass through the corona radiata enter the internal capsule with the pyramidal tract, and make their first synapsis with the chief motor nuclei, in the dorso-lateral part of the tegmentum of the pons. Most of the fibers cross about the level of the posterior corpora quadrigemina. From here the second motor neuron passes with the inferior maxillary branch through the foramen ovale, and is distributed to the masseter, temporal, pterygoids, tensor tympani, tensor veli palati, mylohyoid and the anterior belly of the digastric.

[1] Map scheme of the Sensory Distribution of the Vth Nerve. L. H. Pegler, 1914.

Affection of the *cortical motor* neurons occurs in pseudobulbar palsy. Here the lesion is bilateral also. Unilateral interruption of the tract causes little disorder in mastication. (Hirt claims that a left-sided lesion may cause bilateral palsy.) Bilateral disorder is nearly always associated with the other features of pseudobulbar palsy (*q. v.*, Fig. 192). The paralytic signs are the half-open mouth, with inability to approximate the teeth. The jaw cannot be protruded, and the lateral movements are impaired. The food is apt to fall out of the mouth, cannot be held by the lips and cheeks or tongue, and has to be manipulated by the fingers. Food is often pushed up to the pharynx and nose. There is no atrophy of the muscles of the jaw, and no reaction of degeneration. The jaw-jerk is increased.

Cortical foci may give rise to chattering movements of the jaw. Grinding movements of the jaw, so frequent in paresis and occasionally present in senility, may accompany cortical loss. The champing movements of the jaw in paralysis agitans are possibly to be interpreted similarly to the general tremor of the other muscles; namely, as an interruption of the tonic impulses passing through the midbrain (corpora striata) structures. (See Paralysis Agitans.) Grinding of the jaw is not infrequent as a reflex in children, and it occasionally is seen as a result of basal meningeal irritation of the motor root, as in tuberculosis, syphilis, or even tumor formation.



FIG. 185.—Paralysis of both the motor and sensory fifth. Trophic ulcer of the right eye and deviation of the jaw to the paralyzed side on opening the mouth. (Holmes.)

Prolonged spasm of the muscles of mastication is seen in certain toxemias, such as strychnine poisoning, tetanus, tetany. Here the interpretation is not simple. It is a result possibly of the marked

lowering of the synaptic threshold in the pontine motor nuclei, causing overresponse to the cerebral or reflex motor impulses. The violent convulsive movements of the epileptic discharge are very complex.

Irregular or anomalous spasmodic movements of the jaws occur in multiple sclerosis, usually from midbrain or pontine localizations of the plaques, or they may be reflex or psychogenic (hysteria or dementia precox). In the latter instances in the few cases analyzed the biting symbolizes unconscious sadism (cannibalism.) The clenching of the jaw in food refusal as in schizophrenia, depressed manic-depressives, fever deliria or confusion, expresses various symbolizations. Fear of being poisoned is here a frequent motive at the conscious level. Jaw-grinding at night in sleep is usually symbolic of protests and is frequently interpretable from other features of the dream phenomena by psychoanalysis.

Nuclear disease of the motor neuron of the trigeminus may be unilateral or bilateral, partial or complete. In unilateral monoplegia masticatoria, the lateral movements of the jaw take place to the paralyzed side. Bilateral lesion causes the jaw to fall, and abolishes all lateral movements. The floor of the mouth is flaccid from the mylohyoid and digastric palsy, and there is difficulty of hearing notes of low-pitched tuning-forks. The muscles show atrophy, reaction of degeneration and the jaw-jerk is absent.

Peripheral trigeminus motor palsy is usually associated with sensory, sympathetic and taste phenomena.

Nuclear disease of the motor neurons is comparatively rare. It may occur in multiple sclerosis, in syphilis of the pons, hemorrhage, poliomyelitis, syringomyelia. Peripheral palsies are more frequent, and are due to trauma, to pressure of carotid aneurisms, tumors, chronic meningitis and at times to infections and toxemias.

Sensory Part.—Affections here are much more intricate and complex. and are often combined with motor symptoms. The sensory receptors of the trigeminus are widely distributed over the face, the mucous membranes of the superior and anterior nasal fossa, the frontal and ethmoid sinuses, tentorium cerebelli, teeth, mucosa of posterior inferior nares, the sinuses of the jaw, the dura mater, the mucous membranes of the lips, cheeks, posterior and inferior portion of the mouth, and the filiform papillæ, which function as tactile receptors. The taste buds are receptors for the glossopalatine and glosso-pharyngeal nerves. The fifth nerve probably does not function as a gustatory nerve.[1] The sensory ganglion is the Gasserian.

Collateral synapses occur with the ciliary ganglion for the passage of impulses from the cornea and sclera receptors. Impulses from the ethmoid and sphenoid sinuses, the pharynx, posterior nares, hard and soft palates, maxillary sinus, uvula, pharyngeal walls, tonsils and related mucous parts pass by way of the sphenopalatine ganglion and come into relation with the glossopalatine and glosso-pharyngeal. The submaxillary ganglia connections are intricate and probably pass with the vegetative nervous system fibers.

[1] Kappers: Psych. Neur. Bladen, 1914. Winkler: Manuel de Neurologie, 1918.

The chief reflexes arising from these connections are:

1. Winking reflex.
2. Pupillary (sympathetic reflex)—pinching the cheek or neck causes a dilatation of the pupil on the same side.
3. Jaw reflex.
4. Sneezing reflex.
5. Pharyngeal reflex—(gagging and swallowing).

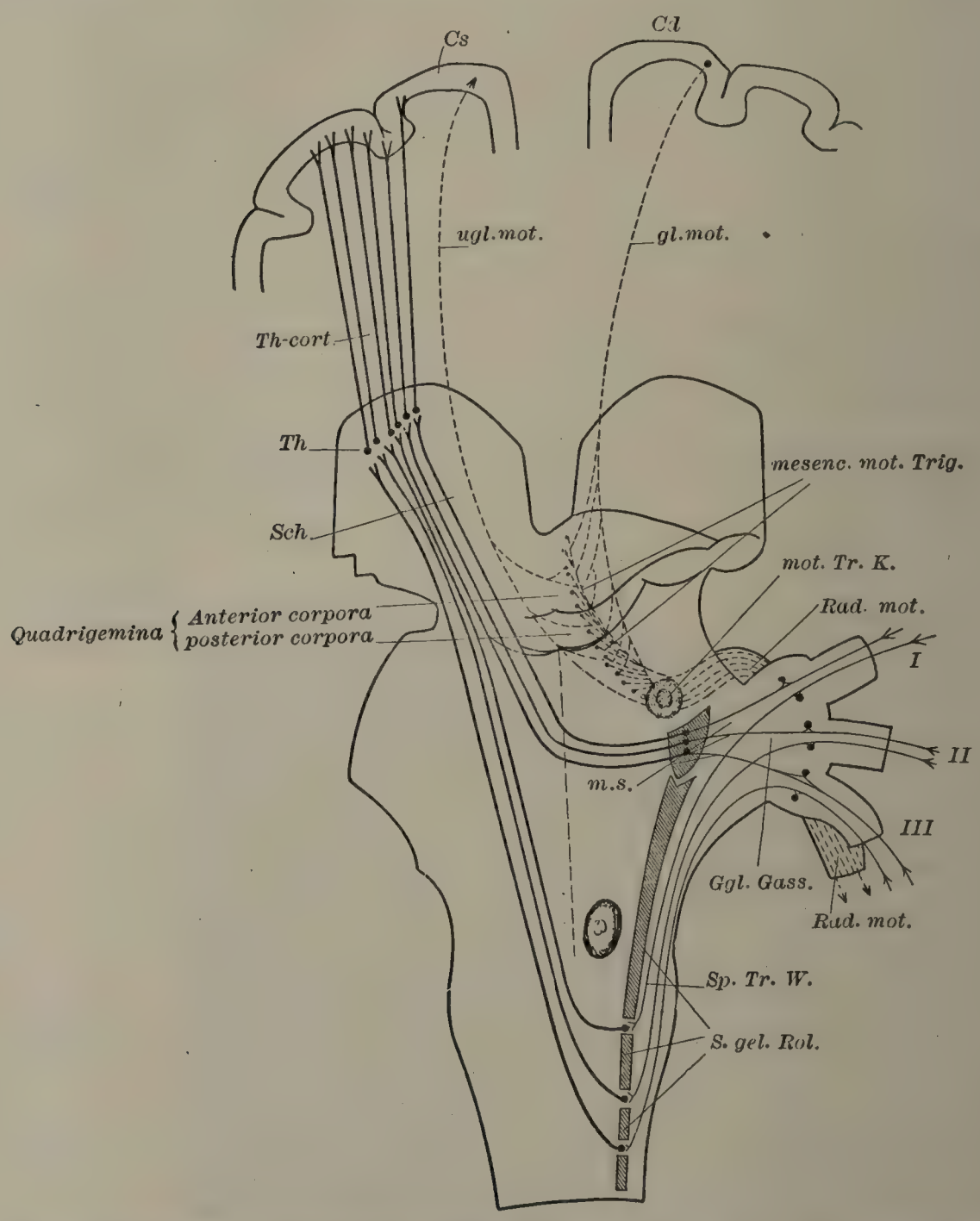


FIG. 186.—Scheme of intracerebral trigeminus pathways. *Cs*, left hemisphere; *Cd*, right hemisphere; *Th-cort.*, thalamocortical trigeminus tracts; *Th*, synapses of trigemenus in thalamus; *Sch*, mesencephalic lemniscus; *mot. Trig.*, motor trigeminus root nuclei in midbrain; *mot. Tr. K.*, motor trigeminus nucleus in locus cœruleus; *Rad. mot.* motor roots; *Ggl. Gass.*, Gasserian ganglion; *I*, *II*, *III*, three trigeminus branches; *m.s.*, mesencephalic sensory trigeminus roots; *sp. Tr. W.*, spinal trigeminus branches; *S. gel. Rol.*, substantia gelatinosa Rolando. Dotted line, motor; solid line, sensory. (Veraguth-Bing.)

The centripetal pathways from the Gasserian ganglion join to form a large sensory root which is distributed to two main end-stations: a mesencephalic and a spinal one with numerous collaterals. From these roots the second sensory neuron passes through the median

lemniscus to the opposite side to end in the thalamus. A further neuron then passes to the sensory brain area.



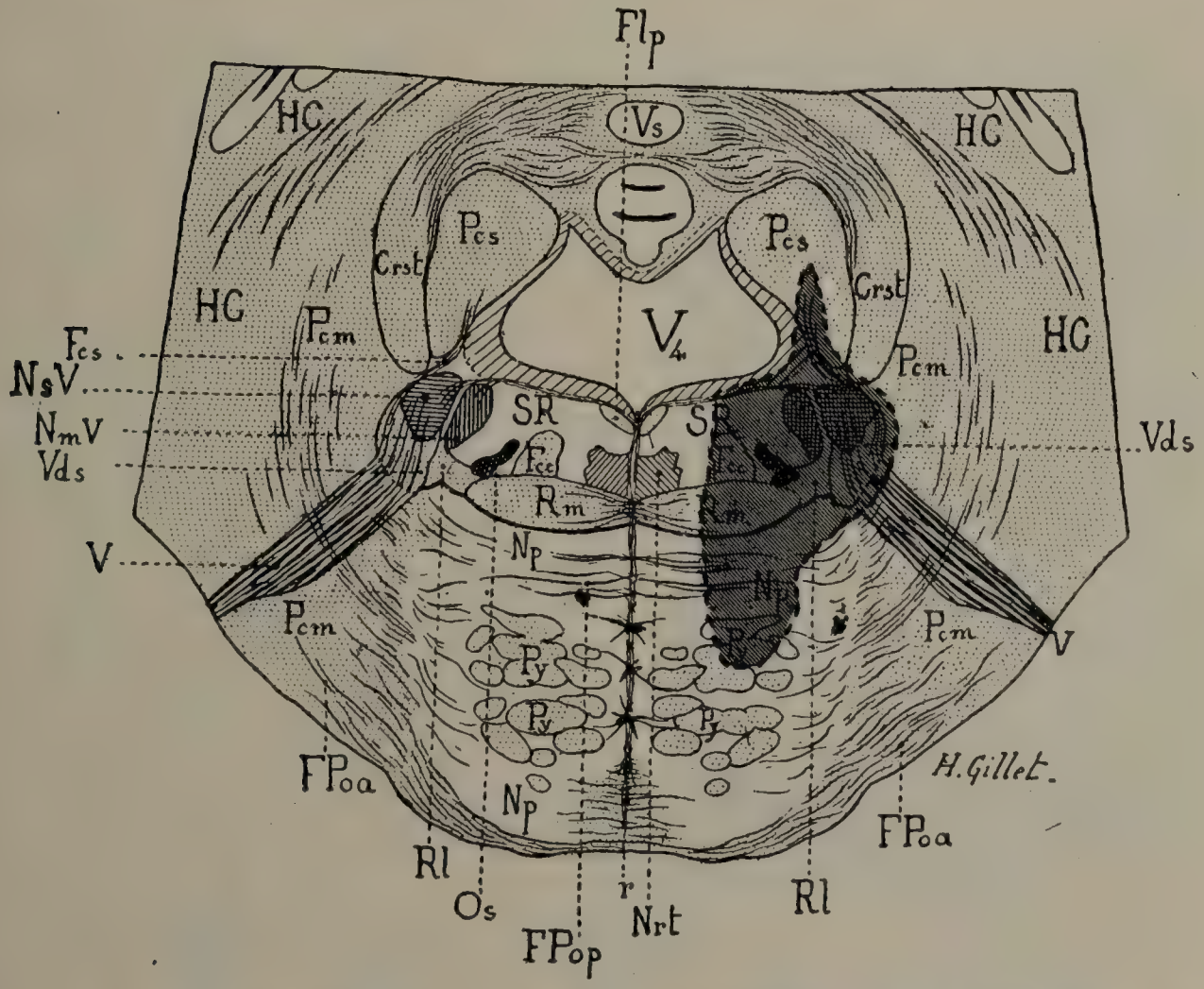


FIG. 187.—Pontine syndrome. Alternate hemianesthesia of the trigeminus by lesion of the median portion of the left pontine tegmentum involving the motor and sensory roots of the trigeminus, their root fibers, the external portion of the median lemniscus, the crossed sensory paths of the tegmentum and extending as far as the superior cerebellar peduncle behind and in front as far as the pons implicating peduncular pyramidal fibers. On the *right* side there is hemiparesis of the extremities and of the inferior facial (oblique cross hatchings), hemianesthesia of the extremities, neck and head, especially of pain and temperature sense (vertical lines). On the *left* side there is anesthesia of the trigeminus; paralysis of the masticators, the pterygoids especially, the masseter and the temporal. There are choreo-athetoid movements of the body due to lesion of the superior cerebellar peduncle (*Pcs*). (Dejerine.) (See Midbrain Section.)

The chief sensory symptoms of fifth nerve involvement are hyperesthesiæ and neuralgia, hypesthesiæ, anesthesiæ. Loss of taste, trophic and secretory disturbances, hay fever, rose colds, and psychogenic

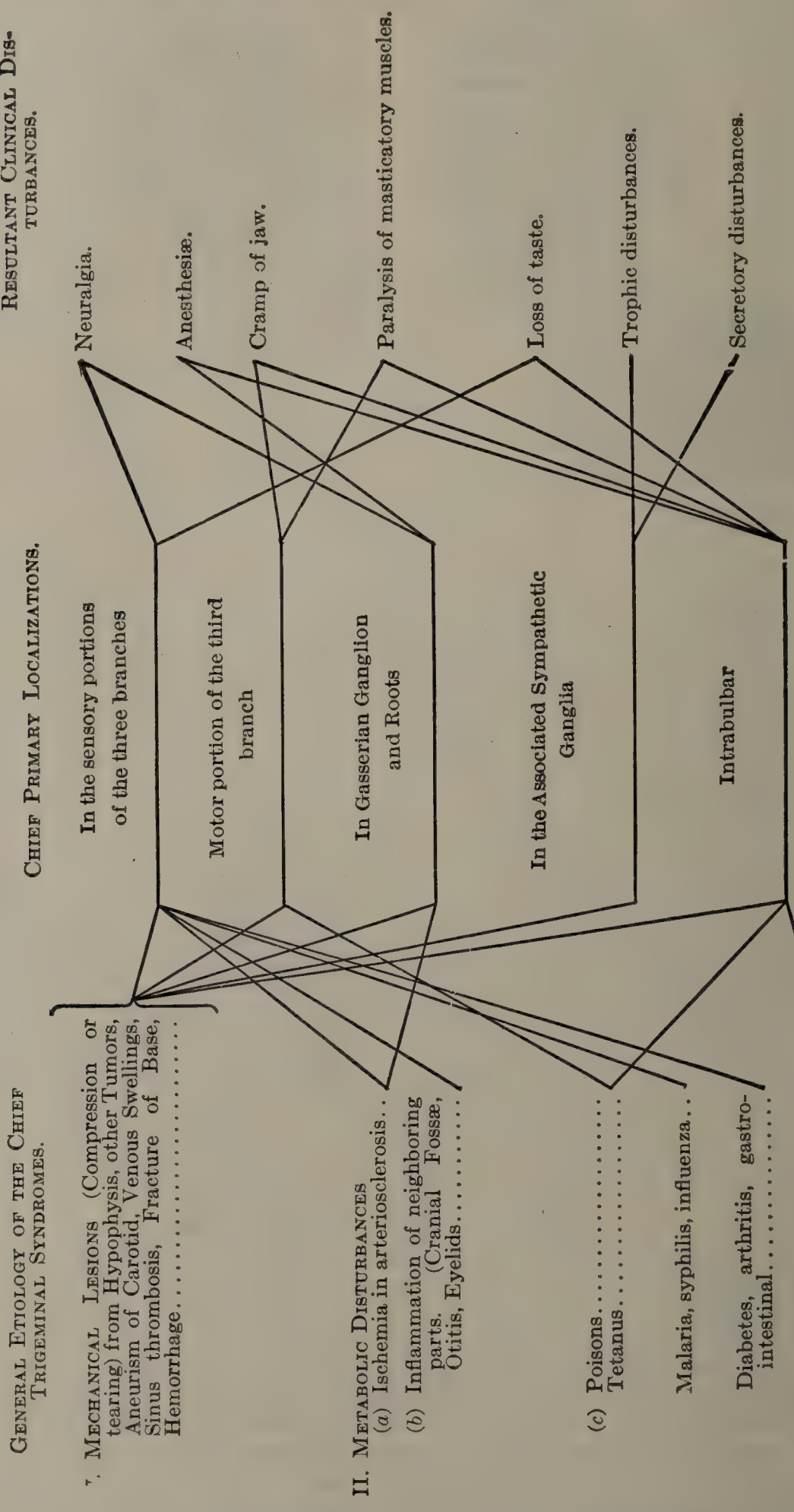


Fig. 188.—Summary of trigeminal [and related, J.] disturbances. (Veraguth.)

colds occur from implication of mixed vegetative, glossopalatine (chorda tympani) and glossopharyngeal pathways. The apparent loss of taste in trigeminus (Gasserian) operations is frequently failure to

separate tactile from gustatory impressions, and also failure to realize the great amount of damage done to contiguous structures. Gasserian operation always involves other structures than the Gasserian ganglion itself.

The most frequent of the syndromes is *trigeminal neuritis* or *tic douloureux* (*q. v.*). Here the Gasserian ganglion is often involved or the neuralgia may be due to pressure upon one or all of the roots. The distribution of the hyperesthesia is of value in determining the branch or branches involved. It is comparatively rare to find reflex neuritic pains from disease of the teeth, hence the hope that removal of healthy teeth will cure a tic douloureux is usually doomed to disappointment. This is an extremely common error and needs to be emphasized.

Central or thalamic trigeminal pains are possible. Clinically little is known of them. Trigeminal agnosia is a curiosity merely. Anesthesia may be due to interruption of peripheral, pontine, thalamic or cortical pathways. The diagnosis as to localization must be made on the basis of the accompanying symptoms, sensory, secretory, trophic, and motor.

An inflammation of the Gasserian ganglion causes a trigeminal herpes zoster. Dryness of the eyes, with hyperesthesia or anesthesia is due to a peripheral lesion of the superior or first branch of the nerve. Unilateral myosis may also point to trigeminal irritation here. Related dryness of the mucous membranes of the nose, lips, and cheeks with anesthesiæ, usually point to peripheral disease of the second branch, while taste impairment of the anterior two-thirds of the tongue may be, but is not invariably, associated with lesions of both second and third branches, in which chorda tympani fibers of the glossopalatine are also implicated.

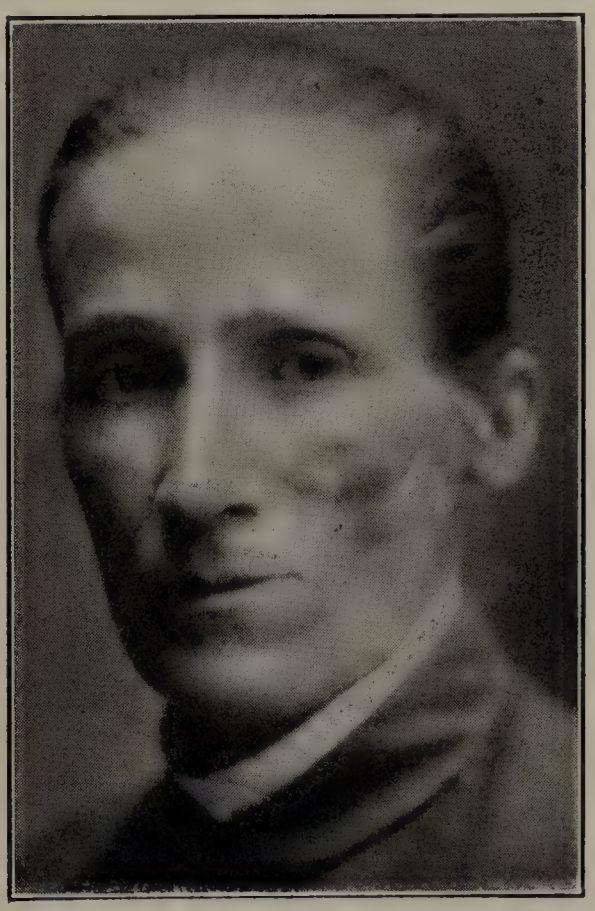


FIG. 189.—Syringomyelia, beginning as hemifacial atrophy, then developing "Morvan's disease," and finally showing classical syringomyelic end lesions. (Hammond.)

In root lesions, the epicritic loss is usually less than the protopathic loss, while the reverse is usually true for peripheral lesions. Pontine lesions show a more general loss of epicritic sensibility on the side of the lesion with mono- or hemihypesthesiæ or anesthesiæ on the opposite side of the body, while thalamic lesions are associated often with anesthesia and analgesia to pin-prick, central pain and affective overresponse. (See Thalamus.)

Dissociation of pain and temperature from epicritic touch sensibility

may take place in the trigeminus distribution. For lack of space here a complete analysis of sensibility disturbances of the trigeminus should be sought in special monographs. (See section on Neuralgia.)

A true psychogenic tic douloureux is always a possibility. It is often curable by psychoanalysis.

Trophic disturbances, usually due to peripheral disease (?), cause changes in the gums and mucous membranes, ulcerations, herpetic eruptions. Corneal ulceration and loosening of the teeth are often present, but whether trophic or not is not certain.

Progressive Facial Hemiatrophy.—This rare condition, which shows at its onset a gradual thinning, with wrinkling of the skin about the orbit or jaws, with later progressive atrophy of the bones, cartilages and muscles, also of the tongue and soft palate, without sensory signs or reaction of degeneration is at times a result of peripheral or pontine (nuclear) disease of the fifth nerve.

DISEASES OF THE FACIAL NERVE.

Seventh Nerve.—The seventh nerve is possibly a mixed nerve. The cortical origin of the motor neuron occupies the lower third of the precentral convolution, from here the fibers pass through the knee of the internal capsule, through the middle third of the peduncle and make their first junction (possibly by means of intercalated neurons) with the homo- and contralateral seventh nerve nuclei in the tegmentum or the pons, just ventrolateral to the abducens nerve nucleus. From these nuclei, four are usually described, the second motor neuron fibers make a dorsal upward curve (genu facialis) (see Fig. 175) around the abducens nucleus, then pass ventrally and emerge at the posterior border of the pons, lateral to the olive, where they lie in close relation to the fifth and eighth nerves in the cerebellopontine angles. They are finally distributed (three ventral nuclei) to the muscles of expression of the face, to the muscles of the external ear, the stapedius, the posterior belly of the digastric and to the stylohyoid. The frontalis, corrugator supercilii, and orbicularis palpebrarum are innervated by fibers coming from the dorsal group.

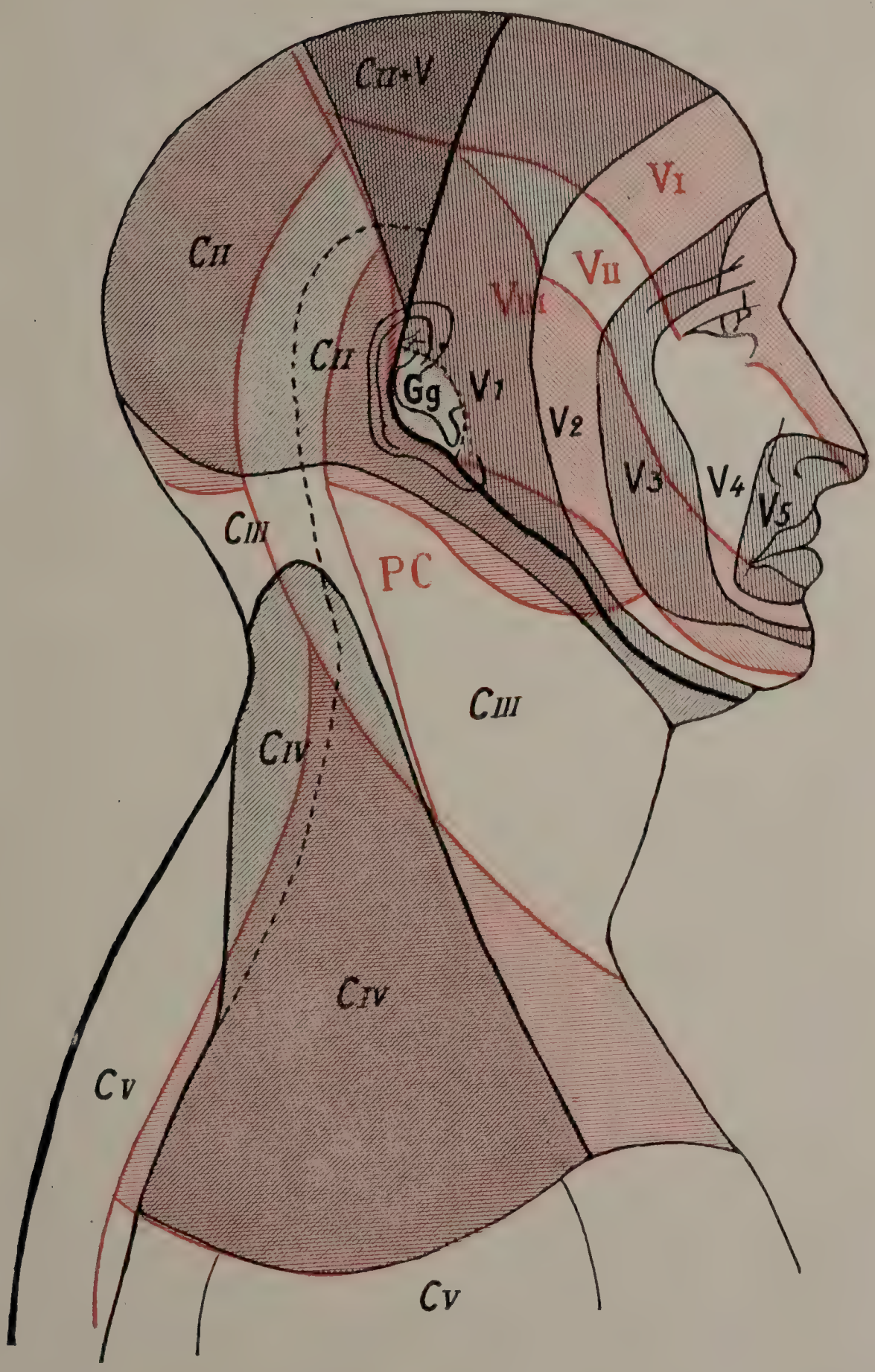
In its peripheral distribution the nerve passes through the facial canal in the temporal bone (aqueduct of Fallopius), coming into intimate relations with other cranial nerves, eighth, pars intermedia, and also forming collateral associations with vegetative fibers of more than usual complexity, a study of which is of value in the local diagnosis of lesions of this nerve and contiguous parts. (See Fig. 191).

The anatomy of the possible sensory portion of the nerve has not been definitely homologized. The comparative studies of Herrick and Johnson fail as yet to show sensory components in forms higher than the amphibia.[1] Reinhardt[2] describes part of the gustatory apparatus as facial sensory.

[1] Jour. Comp. Neurol., 1914. Mills, C. K.: Sensory Components of Seventh Nerve, Jour. Nerv. and Ment. Dis., 1910. Kidd: Review of Neurology and Psychiatry, 1914.

[2] Reinhardt: Jour. Comp. Neurol., 1918, vol. **30.**

PLATE XI



Superposition of the Peripheral Sensory Distribution (in red) and of the Nuclear (in black) of the Trigeminus.

V 1-5 shows gradual involvement of the area supplied by the trigeminus in a case of syringobulbia; VI, VII, VIII, the three branches of the trigeminus; CI-V, distribution of the cervical nerves; Gg, the territory of the geniculate ganglion (Hunt's zoster zone of the facial). (Dejerine.)

By some its sensory ganglion is considered to be the geniculate, which is thought to contain the afferent fibers from touch receptors located in the auricle of the ear, the floor of the external auditory canal, the tympanum, and from certain soft parts of the internal ear. The

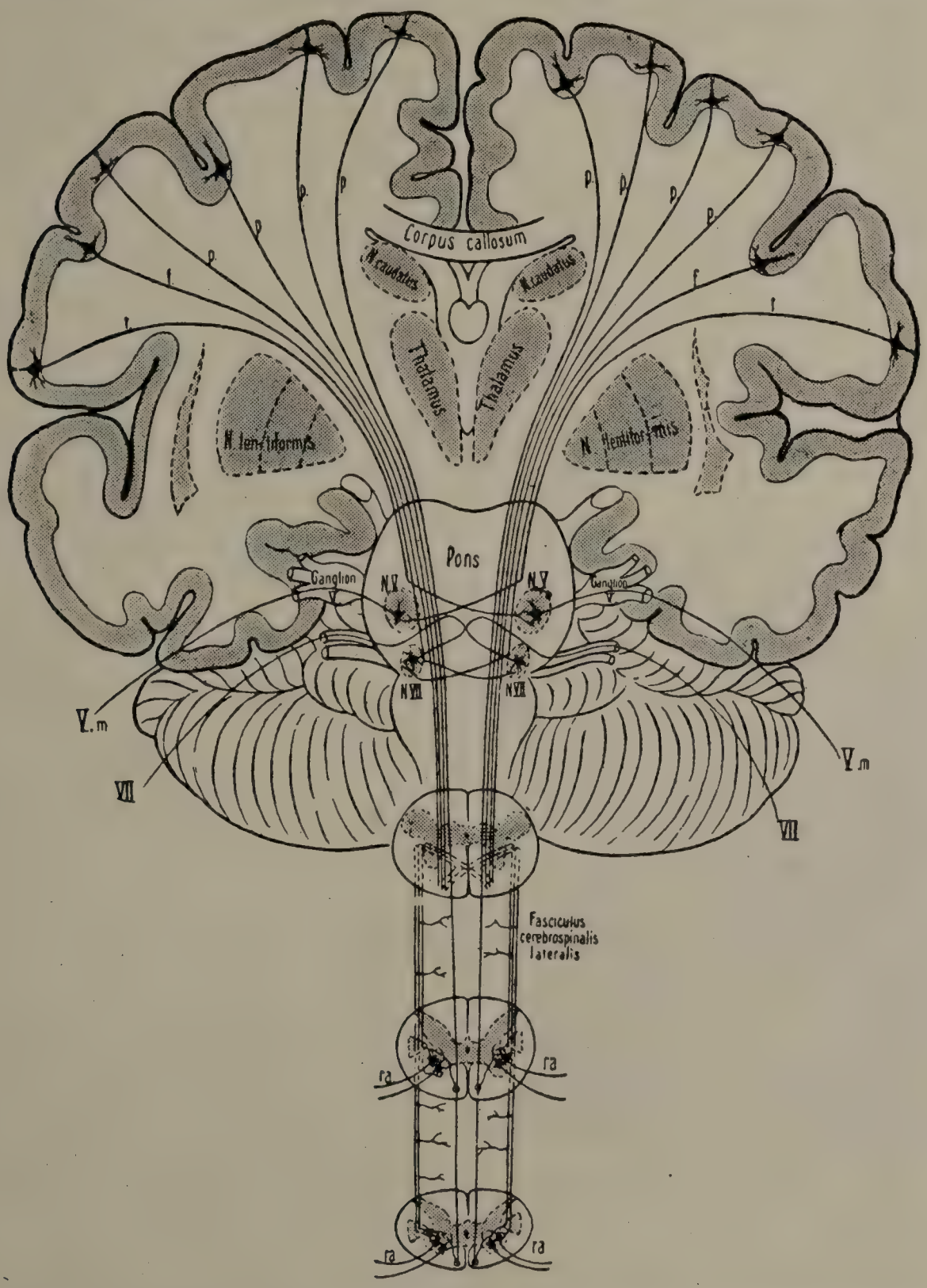


FIG. 190.—General scheme of the course of the pyramidal fibers and the central paths of the motor trigeminus and facial. *Vm.*, lower division of the trigeminus; *VII*, root fibers of the facial; *f*, central facial fibers; *NV*, motor fifth nucleus; *NVII*, facial nucleus; *p*, corticospinal fibers, lateral and anterior; *ra*, anterior roots; *t*, central tract of the motor fifth. (Bechterew.)

nerve of Wrisberg is considered to be the sensory portion of the nerve. *Hunt's syndrome* is a clinical picture thought to uphold this interpretation. Modern studies seem to support this conception.

Cortical Palsies.—Cortical or capsular facial palsy is usually unilateral, and most characteristically affects only the lower facial distribu-

tion. This is the typical facial palsy of the upper neuron (hemiplegic) type. The face is drawn to the sound side, the angle of the mouth droops and the nasolabial fold is flattened, but the eyes can be closed and the forehead wrinkled. There is paresis or paralysis of the lower muscles varying with the severity of the lesion. The soft palate may show palsy, pulling to the sound side on phonation. Babinski further described a loss of the contractions of the platysma of the affected side on forcing the mouth open against slight resistance.

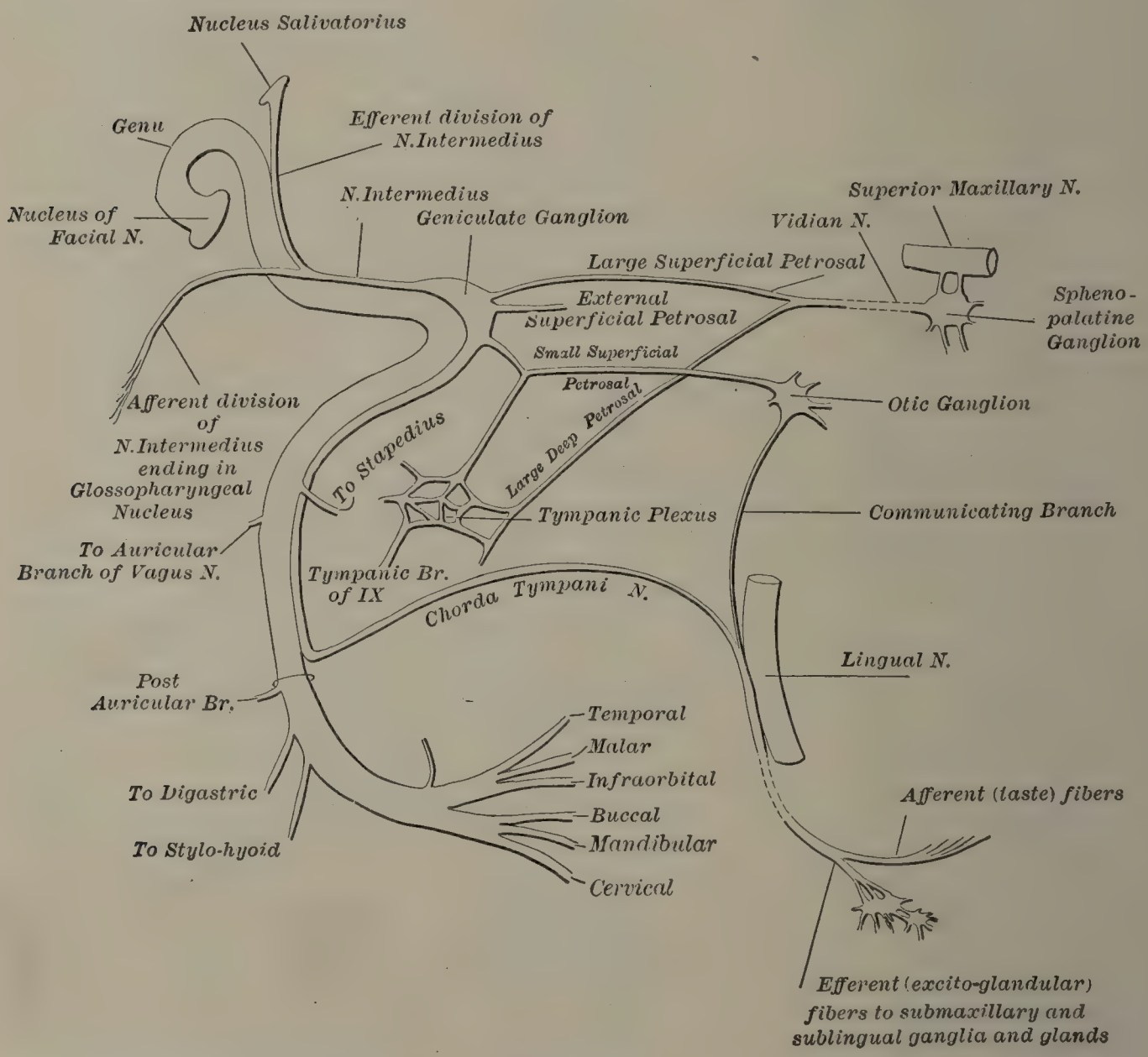


FIG. 191.—Plan of the facial and intermedius nerves and their communications with other nerves. (Gray.)

In certain widespread cortical neuron palsies, however, the upper branches may be involved, with narrowing (at times widening) of the palpebral fissure, and a drooping of the outer angle of the eyebrows on the affected side. Pontine syndromes frequently show these signs. (See Midbrain Section.)

Apraxia of the facial musculature is met with in cortical, or corpus callosum lesions. Here the patient loses the power to make proper mimetic movements. He may not be able to close the eye on the paralyzed side, independently one of the other. Furthermore, in cortical neuron palsy the tongue protrudes to the paralyzed side or cannot be protruded at all. Speech disturbances are frequent.

In cortical facial monoplegias—or hemiplegias with facial involvement—there are no atrophies, the electrical reactions are not involved, and secretory and taste modifications are absent.

Cortical and subcortical irritation may give rise to facial convulsive movements—spontaneous laughing or crying movements.

The mimetic movements of mixed psychogenic-somatic origin, tics, silly grimaces of the schizophrenic, drawn expression of the depressed manic, tenseness of the paranoid types, etc., are numerous.

Pontine Facial Lesions.—When the nuclei of the peripheral neurons are involved all of the branches may be affected, but inasmuch as there are different groups of nuclei, occasionally, as in poliomyelitis for example, certain muscles are involved and others are not. A general lesion here will cause a total palsy of the muscles with atrophy and loss of electrical reactions. There are no changes in taste, nor of hearing in the pure nuclear cases.

Lesions here are apt also to involve the third nerve, also the pyramidal tract fibers, and the sensory fibers of the fillet, hence a variety of hemiplegic or hemianesthetic syndromes—crossed or lower alternate hemiplegias (Figs. 179, 199, 205). (See Section on Midbrain.) In epidemic encephalitis a variety of facial muscle disturbances may occur. Grave myasthenias are among them, choreas, tics, etc. These tics should be separated from the pure symbolic tics. (See Striatum lesions.)

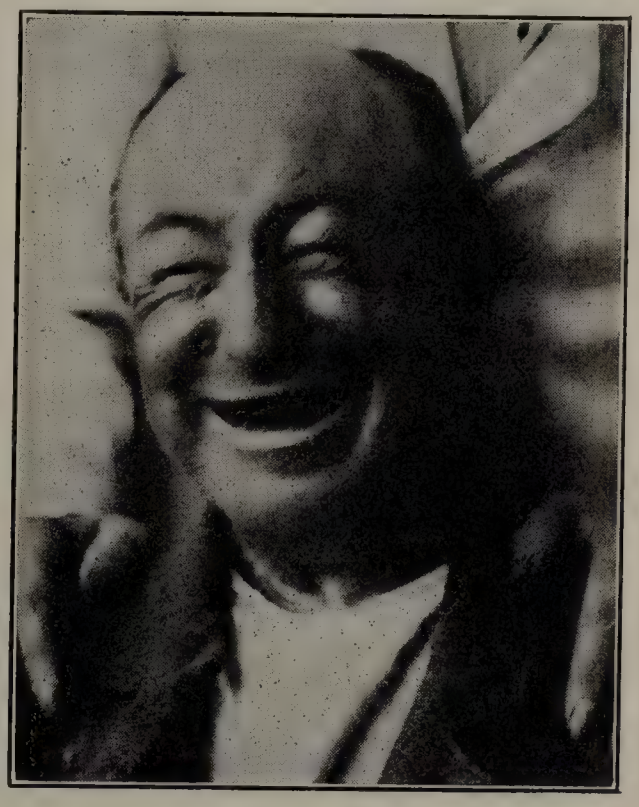


FIG. 192.—Pseudobulbar palsy. (Tilney.)

Supranuclear pontine lesions may rarely be double (Pseudobulbar palsy types).

Peripheral Facial Palsies.—These are called Bell's palsies since first described by Bell. Here a great variety of syndromes may occur, depending on the exact site of the lesion. These may be conveniently divided into five syndromes (see Figs. 191 and 193). 1. Mostly peripheral, due to disease or pressure at or outside of the stylomastoid foramen. This results in a complete paralysis of the muscles of the side of the face. At rest the asymmetry is marked in proportion to the severity of the palsy—all grades are found. The muscles of the forehead cannot be contracted horizontally or vertically, the eye remains partly or widely open on attempts at closure, closing at night in sleep; the nasal orifice is narrowed, the nasolabial fold is obliterated, the angle of the mouth droops and shows the teeth, and there is pulling of the mouth to the sound side. Puffing the cheek is impossible, holding food and saliva are difficult, and on attempting to whistle the air comes out

on the paralyzed side. Tears run down the cheek but the reddening of the eye is secondary. There is less sweating on the paralyzed side. Pressure-pain sensibility is unimpaired. The palate and tongue may be apparently involved, but careful scrutiny shows otherwise. Reaction of degeneration sets in as a rule in the severe cases. Slight speech disturbance is apparent, especially in the beginning, and is very marked with a (rarely occurring) double peripheral palsy (see Figs. 194, 195, 196 and 197).

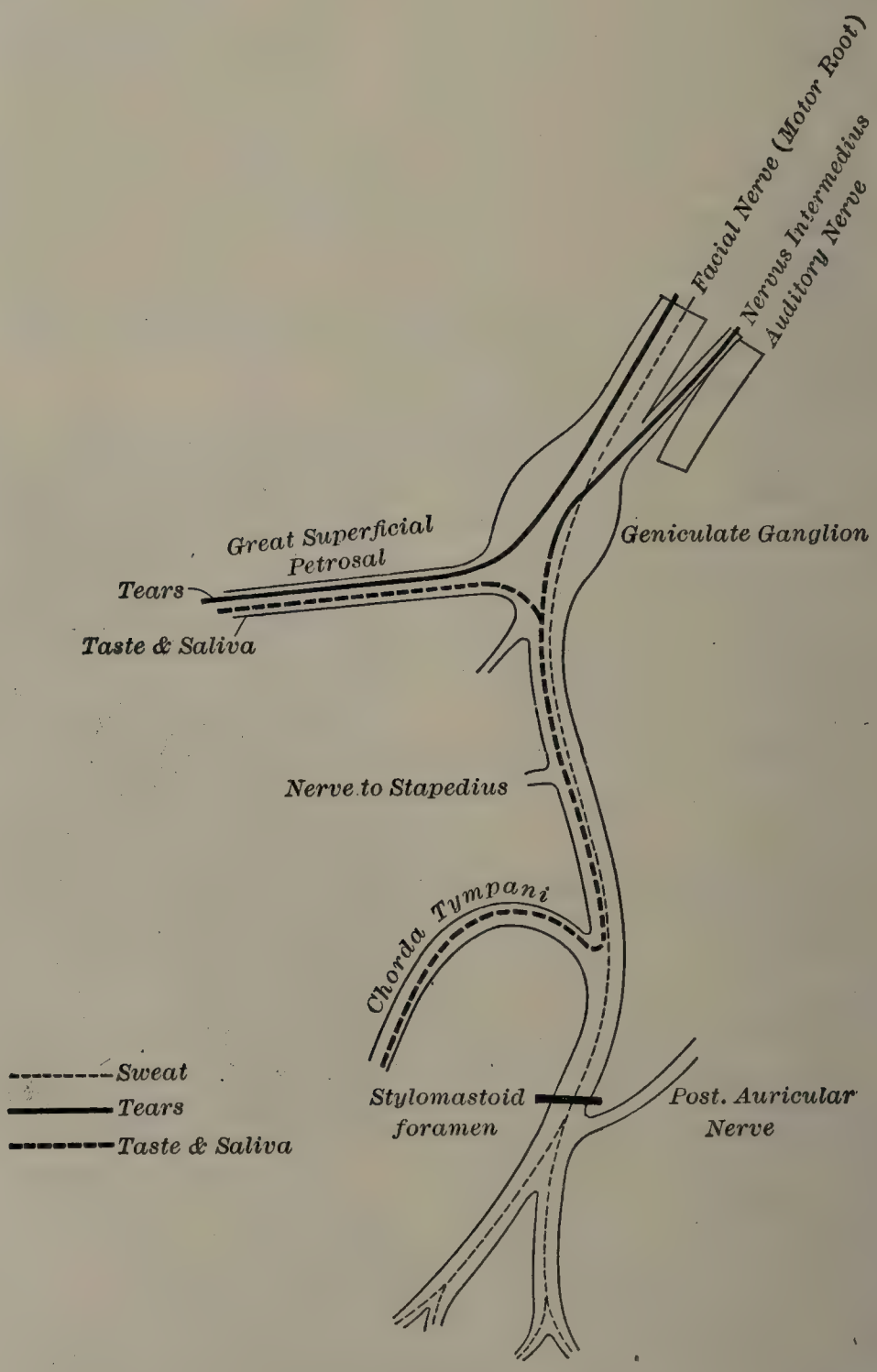


FIG. 193.—Diagram of facial nerve, showing course of secretory and of taste fibers [of glossopalatine, J.]. (Stewart.)

These palsies are due to trauma or pressure from a tumor, possibly a perichondritis of or swelling about the stylomastoid foramen (called rheumatic or refrigeration palsy). The effect of cold upon the facial nerve itself, *i. e.*, by exposure in riding with one side exposed to open windows, etc., looms large in statistical enquiries.

2. Just within the stylomastoid foramen, and within the Fallopian

canal, the chorda tympani (part of the glossopalatine) travels with the facial nerve. A lesion here will cause all of the symptoms just enumerated, and in addition there will be an impairment or loss of

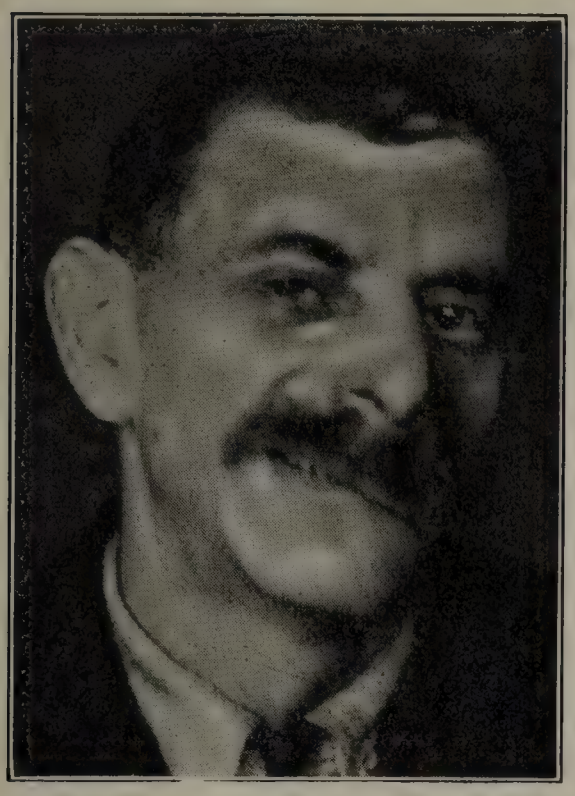


FIG. 194.—Peripheral facial palsy. Smiling.



FIG. 195.—Peripheral facial palsy. Whistling.

taste of the anterior two-thirds of the tongue. There will also be a diminution of the salivary secretions. Pressure-pain sensibility is not impaired.

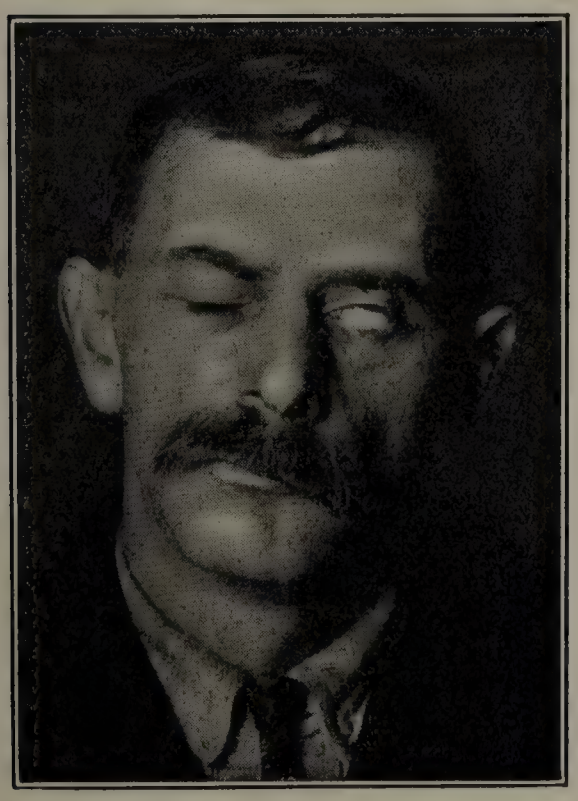


FIG. 196.—Peripheral facial palsy. Closing the eyes.

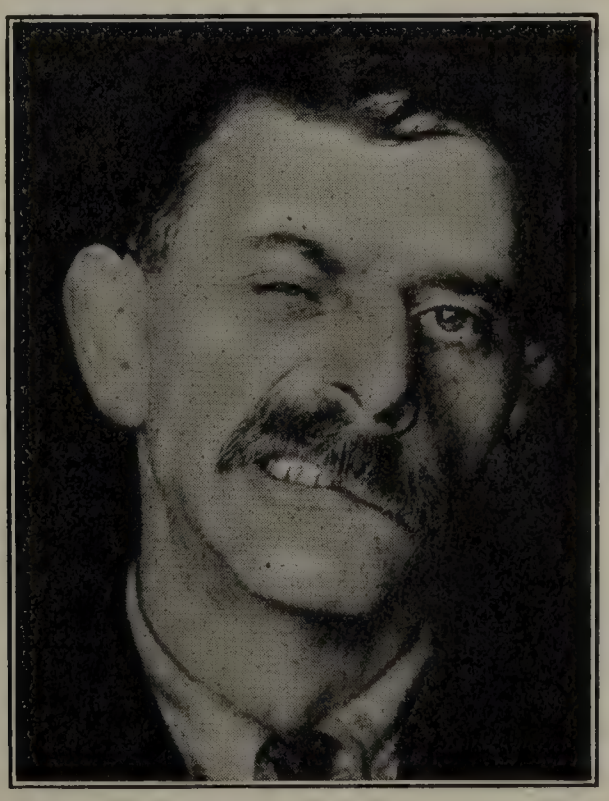


FIG. 197.—Peripheral facial palsy. Showing the teeth.

3. If to this last syndrome hyperacusis and tinnitus alone be added the lesion is slightly farther back in the canal involving the branch given off to the stapedius muscle (see Figs. 191 and 193).

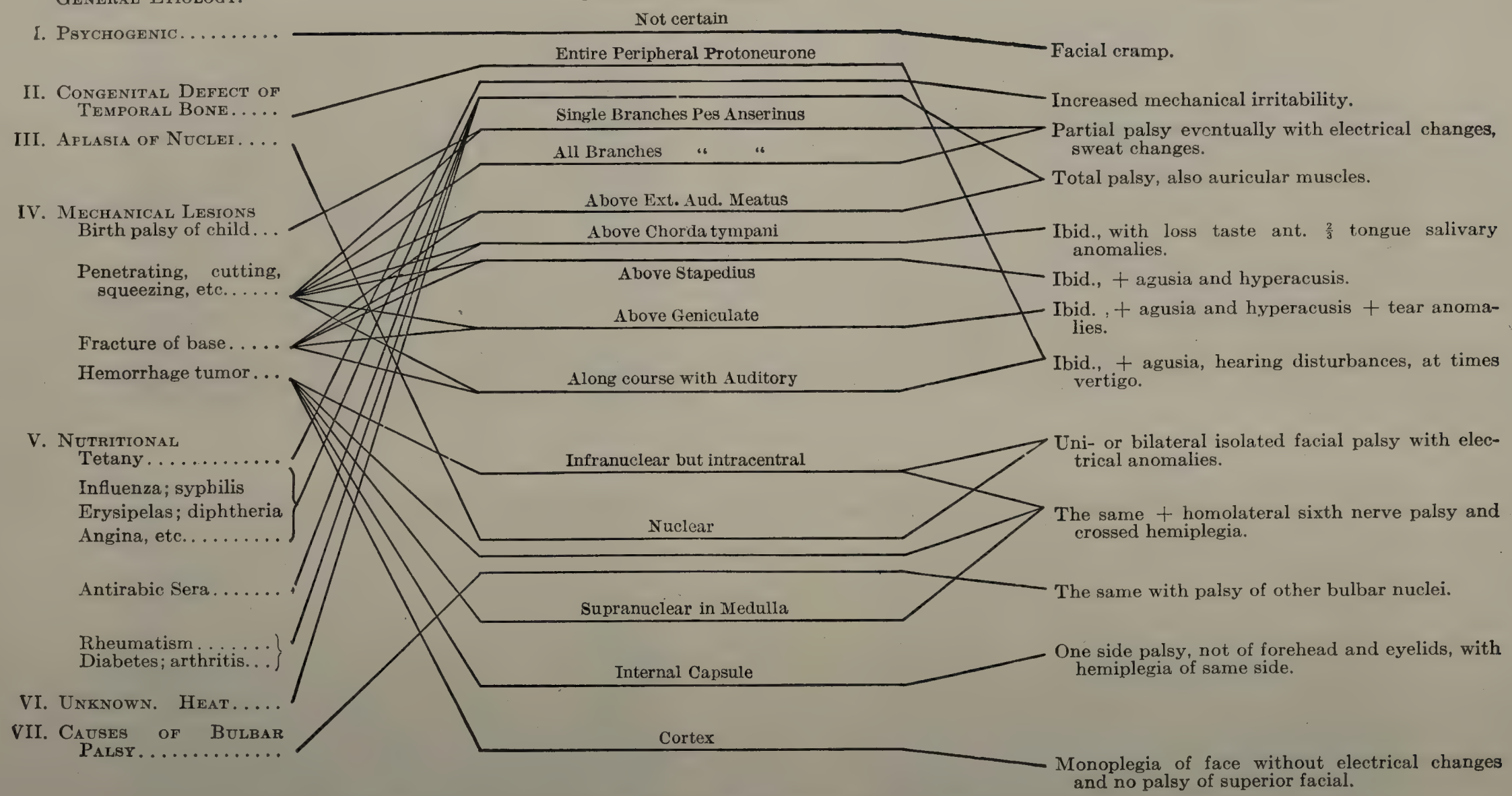


Fig. 198.—Summary of the chief seventh [and glossopalatine, J.] nerve syndromes. (Veraguth.)

4. Lesions lying between the geniculate and the stapedius within or at the internal entrance to the Fallopian canal, cause a variety of additional symptoms, the exact anatomical relations of which are still somewhat obscure.

As the geniculate ganglion of the glossopalatine nerve lies in the sheath carrying the facial also, an inflammatory swelling of this ganglion, may cause a pressure palsy of this nerve. In addition to the facial palsy other symptoms of the glossopalatine disorder are added, chiefly taste loss. If the acoustic is also pressed upon there may be tinnitus or hearing disturbances. A herpetic eruption may occur also, from involvement of either the geniculate or other posterior ganglia. Herpes of the auricle, may occur from involvement of the cephalic sensory ganglia.

Facial palsy of non-geniculate origin from lesions in the same region may or may not be accompanied by loss of hearing. The chief additional diagnostic feature of lesions here is the lowering of the threshold of deep sensibility (Maloney) from involvement of vegetative fibers.

Lesions of the seventh nerve at its emergence from the pons usually implicate other structures, notably the fifth or eighth nerves, at times the sixth, eleventh, twelfth. The facial palsy is of the peripheral type with no loss of taste, changes in the secretions, or suppression of the lachrymal secretions. Basal symptoms such as anorexia, nausea, headache, and optic disk changes are often present. The chief pathological processes are basal meningitis, usually syphilitic, or tumor formation.

DISEASES IN THE VESTIBULAR AND AUDITORY PATHWAYS.

To adequately summarize, even for the purposes of a text-book, the chief functional defects in this vast complex would require many, many pages. In the evolutionary scheme of things the function of capturing and transforming stimuli from gravity, inertia and sound wave sources, has built up a vast connecting mechanism to manage bodily displacement, hunting food and mates, in which scheme of things the auditory mechanism has been the latest to evolve. Its latest functions have been those to handle speech symbols in their relation to the social mechanisms of modifications of bodily displacement. What mankind, for instance, has built up in a vast symbol of financial interchange by means of draughts, letters of credit, checks, etc.; the bird carried out by going after the worm and bringing it to the nest, etc.

The studies of Winkler, Sherrington, Magnus, de Kleijn and others have shown that the vestibular and auditory systems have a closer origin than that which has been heretofore thought. The story may well begin with the statocyst of lower animals, where are found complex functional relationships to gravity and inertia stimuli (displacement). It gathers the local effects of external excitations, rhythmic or non-rhythmic; it registeres the movements of the surrounding liquid whether

due to the liquid or air shock, provided such stimuli determine a displacement of the body and in its latest development and most specialized form the organ is sensitive to other stimuli. Thus in Pterotrachea, a simple mollusc, the otoliths respond to air vibrations which have been transmitted to water. In a much simpler animal Eucharis, Verworn has shown analogous mechanisms. In the mammalia there are to be distinguished at least four neuro-epithelial structures: (*a*) The striæ nervosæ in the semicircular canals; (*b*) the maculæ, or taches sensorielles of the utricle and saccule; (*c*) the cristæ, or cretes sensorielles, of the ampoules; (*d*) organ of Corti, in the cochlea.

The most recent of these is the organ of Corti which reaches its complete development only in the mammalia. The cretes sensorielles of the ampoules are older, the oldest of all the structures are those of the maculæ.

Winkler compares the maculæ to the primitive statocyst of invertebrates. These organs primarily function for the maintenance of general muscular tonus. They collaborate with the proprioceptive stimuli from the body. The function of the cretes sensorielles of the ampoules is of later evolution, and is more specialized. They have appeared for the registration of the movements of the head and for the head organs, chiefly the eye movements. They remain in close liaison with the general tonus functions of the maculæ. These regulatory functions are strictly condensations of geotropic reflex activities and are purely unconscious. Rhythmic activities have not yet taken on their social role. These are still to develop as various forms of more conscious bodily displacement, marching, dancing, unison actions, etc., chiefly initiated through sound waves and closely related with development of laryngeal and buccal proprioceptive stimuli associated with the developing speech function. Thus the organ of Corti has come to be developed in response to affording a wider conscious acquisition for bodily movement, including tongue movements, speech, in its social orientation function. These reflexes for bodily orientation are no longer geotropic and entirely unconscious, they are taking on sociotropic activities, partly conscious, and in the highest cultural types becoming unconsciously socially valuable. The organ of Corti then is functioning in response to rhythmic or interrupted stimuli from the bodily organs and the tongue symbols. Thus are built up the affective responses to sound stimuli through muscular displacement and imitation. From this point of view the newer psychopathology of the unconscious can develop the muscular tensions that arise in social relations, in the responses to tone, to attitude, and to speech symbols.

It can thus be seen how extremely valuable language has become as a type of release mechanism through gradual condensation of language symbols. The body and its organs thereby are not required to go through severe and dangerous displacements if through language the necessary socially valuable protective reflex be found. Thus the symbol has grown to have a dynamic significance for affecting both

metabolism and conduct. In fact speech has become the most dynamic of all the agencies which play upon the human being.[1]

Vestibular Apparatus.—The study of the evolution of bodily displacement from the lowest form of vertebrates shows that the labyrinthine mechanism is a complex synthesis whose chief concern is regulating the impressions of space relations of the body to that of the head. The *meaning* for the human body of the physical laws of gravity and inertia is its chief concern. It has developed in response to the mechanical stimuli of gravity and inertia, acting largely through a highly developed otolith organ, which reacts to changes in the incidence and degree of pressure upon its sensory end-organs, due to changes in the specific gravity of its surrounding fluids; and also through the semicircular canals which react to changes in position in the three planes of space. The slightest change of the body in space is felt by this apparatus, and in the healthy central nervous system any such change is automatically reacted to by appropriate (proprioceptive) motor response.

The chief members of this complicated series of reflexes have been studied very exhaustively in the past ten years, and the results of animal experimentation of Magnus and de Kleijn on lower animals may be summarized as follows. Magnus'[2] large work may be consulted for details; also the studies of Lewy, and of Rademaker and of Spiegel.

I. Reflexes responding to movements	
1. Rotatory reactions	
(*a*) On head	Apparatus of semicircular canals. Otolithic apparatus may also be stimulated by movement.
(*b*) On eyes	
(*c*) On limbs	
(*d*) On trunk	
II. Reflexes resulting from position	Otolithic apparatus.
1. Tonic labyrinthine reflexes on the bodily musculature	
(*a*) On limbs	Utricle.
(*b*) On neck (and trunk)	Utricle.
2. Labyrinthine righting reflexes	
(*a*) Asymmetrical	Saccule.
(*b*) Symmetrical	Utricle.
3. Compensatory eye positions	
(*a*) Vertical	Saccule.
(*b*) Rotatory	

These motor responses, however, are complicated mechanisms, and all of their elements are not thoroughly analyzed. One of its parts is that of a reflex muscular tonus, by which the ordinary posture of the body is maintained. It is this function that has entitled it to the appellation

[1] Jelliffe: The Symbol as an Energy Condensor, Jour. Nerv. and Ment. Dis., December 1919.

[2] Magnus: Die Körperstellung, Springer, Berlin, 1924. Spiegel: Der Tonus der Skelettmuskulatur, Berlin, 1927, for latest references,

of the labyrinthine tonus. Sherrington[1] has analyzed the complicated interrelations between the proprioceptors of the limbs, muscles, joints, etc., which carry impressions of movements, strains, tensions, etc., and the receptors in the labyrinth. It is impossible to enter into them in detail here. Suffice it to say that the result is the reflex maintenance of the posture of the body, including the compensatory reflexes of the head, and those muscles of the head capable of changing the sense of consciousness of position, particularly the muscles of the eyeballs.

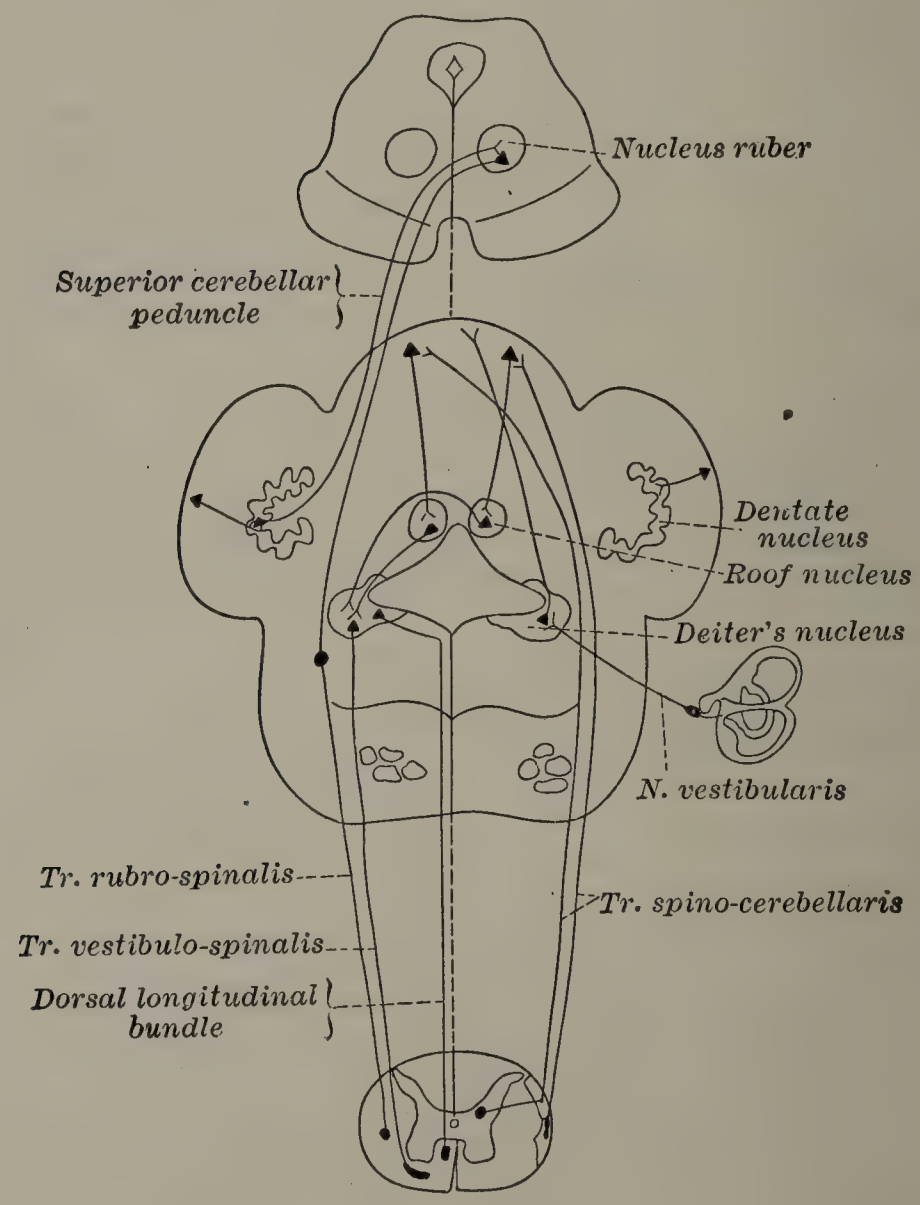


FIG. 199.—Diagram to illustrate the chief spinal connections of the cerebellum. On the right the afferent tracts are represented, on the left the efferent cerebellar tracts. (Holmes.)

The labyrinth is a part of a great system of connections—which Sherrington has designated as the proprioceptive system—which gives animals, human as well as others, a definite attitude toward the external world of space. It is the most important of these organs. It is connected in a system with other nervous structures performing their part in the same general function, and each segment of the body

[1] Sherrington: The Integrative Action of the Nervous System. de Kleijn and Magnus: Pflüger's Arch. f. d. ges. Physiol., 1921, vol. **116**. Magnus: Körperstellung, Berlin, 1924 (see summary, Proc. Royal Soc. Med., 1924, **17**, 11. Winkler: Manual de Neurologie, vols. **1**, **2**, **3**. Lewy, Tonus und Bewegung, 1923. Spiegel, loc. cit.

is caught up in the chain of connections from the lower end of the spinal cord to the frontal area of the cortex.

This whole complicated system of end-organs, fiber connections, long and short fiber tracts, has its chief center, just as every other reflex system has its center. The chief center or head ganglion of this whole proprioceptive system is the cerebellum. The cerebellar connections of the vestibular system, the vestibulo-spinal, vestibulo-bulbar, vestibulo-cerebellar, and, finally, the cerebello-rubro-cortical components which carry those fibers whose functioning is recognized in the consciousness of space relations, are now fairly well known, not in their entirety, but in their main tracts and connections. Hence, disease or disorder which shows any perturbation of the function of

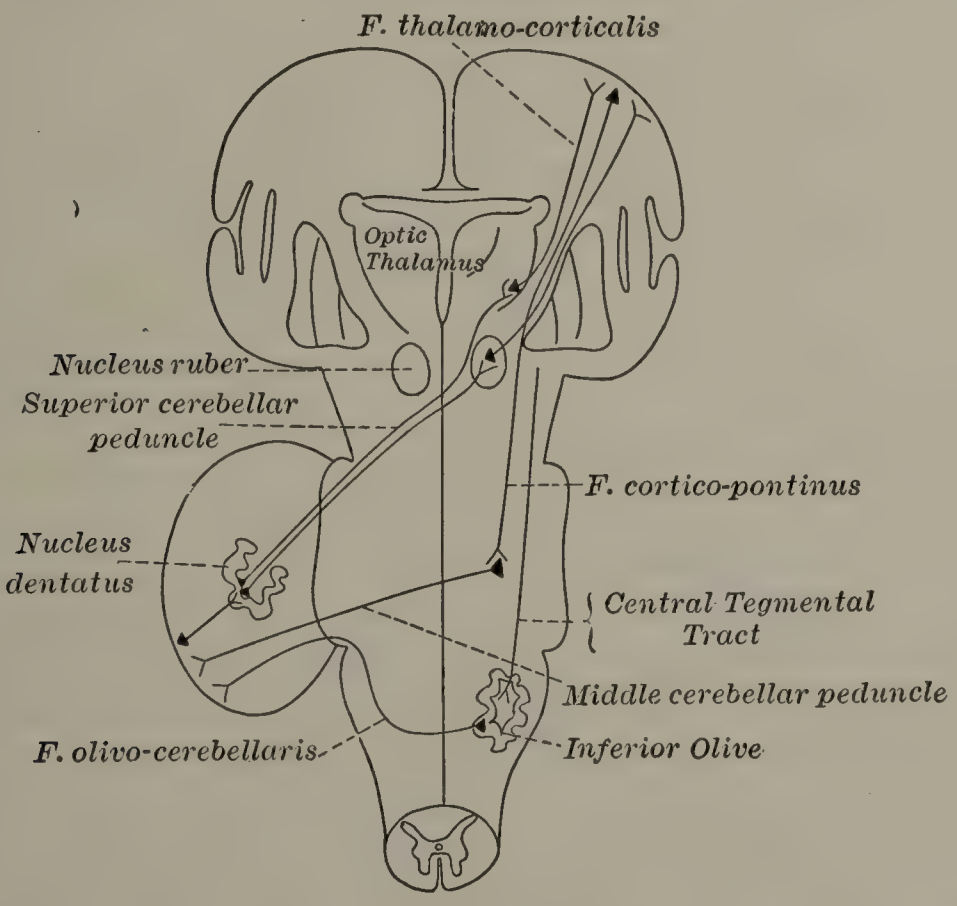


FIG. 200.—Diagram to illustrate the afferent and efferent connections of the cerebellum with the forebrain. (Holmes.)

orientation in space may be more or less accurately localized along the fiber tracts, carrying the necessary impulses underlying these functions, and an appropriate therapy adopted (see Plates X, XII and XIII).

Nervus Vestibularis.—The fibers of the median acoustic root (Lewandowsky—mixed) constitute the central prolongation of the bipolar ganglion cells which make up the vestibular or Scarpa's ganglion. The peripheral prolongations of the cells are the receptors (hair cells of the ampullæ) in the walls of the semicircular canal.. Movement of the head or of the body causes a flow of the endolymph which gives rise to the specific stimuli in the receptor organs. Three stages in the central synapses are recognized. Oblongata, nuclear cerebellar and cortico-cerebellar. The oblongata synapses are the nuclei of Bechterew, Deiters, Roller and the triangular nucleus. The cerebellar nuclear

stations are the nuclei tecti, nucleus globosus, and nucleus emboliformis (paleocerebellum) and the cerebellar cortical stations are in the vermis (lingula, uvula and nodulus) and in the hemispheres, flocculus. These are the chief primary distributions of the vestibular stimuli in the building up of the complex mechanism of automatic muscular plastic control and synergy. For a further consideration of the reflex arcs see chapter on Cerebellum.[1]

Vestibular Vertigoes.—At one time loosely grouped together under the name Ménière's disease, the analyses of later years have shown a great variety of these affections depending upon the anatomical sites of the lesions. One must distinguish between:

1. Disease of the peripheral end-organ, (*a*) partial, or (*b*) complete; these are the vertigoes of partial or complete labyrinthine disease.
2. Disease of the first neuron, (*a*) paresis, (*b*) paralysis of the vestibularis.
3. Disease of the primary end-nuclei in the medulla and of Deiter's nucleus. Lesions of the latter give a special symptomatology termed Bonnier's syndrome.
4. Disease in the region of the posterior longitudinal bundle associated with eye-movement vertigoes.
5. Disease of the nuclear region of the eye-muscles in the corpora quadrigemina.
6. Disease of the pontine eye nuclei.
7. Disease of central eye paths.
8. Disease of cerebellum.

In disease of all these regions vertigoes are to be expected by implication of the vestibular nerve; the character of the accompanying phenomena, especially the nystagmus, aids in the localization. (See chapter on Examination Methods: Vestibular Tests.)

In partial or circumscribed disturbance of the vestibular end-organs in the labyrinth the vertigo is associated with nystagmus. The nystagmus is spontaneous and shows a long slow movement, due to the vestibular, and a quick return movement due to the tegmental nuclei, the direction of the quick movement naming the nystagmus. Vestibular nystagmus usually increases when the eyes are directed in the direction of the quick movement, and usually diminishes or ceases on looking in the opposite direction. There is almost always a combination of horizontal and of rotary nystagmus. Bárány states that every other form of spontaneous nystagmus is of intracranial origin. If the nystagmus movement is rotary and horizontal it must be determined whether it is peripheral or central. A peripheral nystagmus to the right should show on caloric, pressure, and rotation tests that the right vestibule is functionally active. Should such tests show an inactive right vestibular, then the nystagmus must be of central origin. If the right vestibular is active, then continued observation of the nystagmus will alone determine. Should the nystagmus continue uninterruptedly for twenty-four hours or more, it is of intracranial

[1] Spitzer: Arb. Neur. Inst., 1923, vol. **25.**

origin. If it lasts a shorter interval and is uninterrupted by quiet intervals, it may be either peripheral or central. When there is also nystagmus of the well side, which lasts about two weeks, gradually decreasing, then a peripheral disturbance seems certain. Intracranial nystagmus is not so apt to diminish.

The Ménière-like attacks are either mild or marked. Buzzing in the ears is rare in mild attacks. There is no impairment of hearing. In the severer attacks there is little buzzing, but hearing is apt to be impaired. In free intervals the nystagmus diminishes or disappears, the Bárány caloric reaction is diminished on the affected side.

Total destruction of the labyrinth may be acute or chronic; the latter may show no symptoms. The former sets in with violent vertigo, nausea, vomiting. There is marked horizontal and rotary nystagmus of the sound side. The slightest movement of the head increases the vertigo and nystagmus during the first forty-eight hours; the latter gradually disappears in three or four weeks. There is marked loss of coördination, with tendency to fall to one or the other side. After the period of quiescence of the nystagmus, caloric and rotation tests show the defective unction. The galvanic reaction is not usually affected.

Disease of the vestibular nerve, usually due to tumor of base (acoustic, cerebellopontine angle), leads to similar reactions. Here, however, there seems to be a difference in that Neumann has found that the galvanic reaction is reduced or lost, according to a partial or complete destruction of the vestibular ganglion. Other cranial nerves are here involved as a rule. The cochlearis is frequently implicated. Complete deafness does not result. The trigeminus is also often involved and pain, paresthesiæ, or motor defects appear. Cerebellar symptoms may also complicate the picture. The nystagmus is apt to continue in intensity with tumors, and may be on the sound as well as the affected side.[1]

Involvement of the nuclei (encephalitis, abscess, syphilis, tumor) brings about similar attacks of nausea, vomiting, vertigo, and nystagmus. The symptoms continue and increase, as a rule, beyond the three weeks ordinarily seen in labyrinthine disease.

The method of continuous observation aids in locating the diseased focus.

Bonnier's Syndrome, due to implication of Deiters' nucleus and contiguous structures, usually causes a marked attack of nausea, vomiting, vertigo, and nystagmus with buzzing in ears and deafness (Ménière's syndrome), with irradiations to the ninth and tenth nerves causing anxiety, tachycardia, and hemiplegic weakness. The trigeminus and oculomotor are also apt to be involved. Bonnier has also described peculiar somnolent attacks accompanying his syndrome. Little can be done for these cases unless the focus is of syphilitic origin.

Here vertigo and nystagmus are associated in various ways, but the vertigo disappears on closing the eyes, and forced movements,

[1] Cushing: Tumors of the Acousticus and Cerebellopontine Angle, 1917.

conjugate deviations, and various skew deviations afford a clue to diagnosis. Caloric and other tests determine the integrity of the labyrinthine functions.

TABLE OF DIFFERENTIAL DIAGNOSIS OF LABYRINTHINE AND CEREBELLAR DISTURBANCES.

Tests.	I. Circumscribed labyrinthitis.	II. Diffuse labyrinthitis.			III. Diffuse purulent labyrinthitis and cerebellar abscess.	
	Fistula formation.	(a) Serous labyrinthitis.	(b) Acute purulent labyrinthitis.	(c) Chronic purulent labyrinthitis.	Type A.	Type B.
Spontaneous nystagmus	May be absent on sound or diseased side	Mostly on sound side; also on both sides	Rotatory and horizontal; strong on the well side	Weak on well side, or on both sides	Strong rotary and horizontal on sick side	Rotatory and horizontal on well side.
Turning nystagmus	Produced both sides same intensity	Both sides with same intensity	Cannot be tested because of excessive reaction	Residual: on well side, normal time; on sick side shortened	Produced both sides, same intensity.	
Caloric nystagmus	Diminished or not present	Absent	Absent.	Absent	Absent.	
Compression	Present	Absent	Absent	Absent	Absent.	
Hearing	Hears with difficulty or deaf	Hears with difficulty or deaf	Deaf	Deaf	Deaf.	
Other symptoms	Vertigo, nausea and nystagmus on rapid movement of head	Marked vertigo, nausea, fever	Vertigo, nausea and vomiting	Slight vertigo, also slight equilibrium disturbances	Changeable vertigo and equilibrium disturbances; finger test positive, eyegrounds positive	
Treatment	Radical operation	No operation	Radical and labyrinthine operation	Radical and labyrinthine operation	Radical, labyrinth and cerebellar operation	Radical and labyrinth operation; if nystagmus continues, cerebellar operation.

Cerebellar vertigoes have a number of special features. So far as the vertigo is concerned they may not be separable from the labyrinthine or vestibular vertigoes. Hearing symptoms are usually absent. The nystagmus is less apt to be horizontal and rotary, but may be up or down or oblique, and is usually directed toward the affected side.

There are usually also symptoms of a tumbling gait toward the side of the lesion; there is asynergia and usually adiadokokinesia. No real distinction as to the side of the lesion affected can be gained from the fact as to the subjective or objective motion of the objects during a vertiginous attack. Closure of the eyes has no marked affect upon the vertigo, nor upon the gait. Caloric and other tests determine a normal labyrinth.

In conclusion it is emphasized that the subject of vertigo is a very large one and it is necessarily only very hastily summarized in these few paragraphs. More complete knowledge may be gained from a study of the various works mentioned in the footnotes.[1]

[1] Jones: Equilibrium and Vertigo, Philadelphia, 1918, Marburg, et al. Handbuch der Neurologie des Ohres. Magnus. Körperstellung, 1924.

Treatment.—Here there comes into consideration the surgery of the ear and the surgery of the cerebellum and the cerebellopontine angle. The ear specialist should treat the labyrinthine cases, not the neurologist. Rest in bed, quinin, and an expectant treatment which shuts one's eyes to the danger of a suppurative labyrinthitis, brain abscess, etc., is folly.

In the apoplectic form of Ménière's syndrome (hemorrhagic labyrinthitis) often mistaken for a cerebral, or cerebellar hemorrhage, the patient must be kept absolutely quiet, the eyes should be kept closed, the room darkened, and all noises excluded as far as possible—telephone, house bell, etc., shut off. Ice should be applied to the mastoid. Leeches are at times of value. The continuous vomiting may be in part relieved by swallowing cracked ice. Surgical interference may be called for.

In syphilitic cases mercurial injections, salvarsan, or inunctions are called for. It may be noted that the acute labyrinthine disturbance which has been known to occur after the use of salvarsan is possibly due to the syphilis and not to the arsenic (Benario).

Treatment of Seasickness.—Seasickness is a special form of disturbance of the labyrinth due to the continuous movements of the endolymph and irritation of the receptors. As the stomach has little or nothing to do with seasickness, diet has little or no effect upon this malady, and the ocean traveller need pay no more attention to the question of food than that dictated by common sense. Eat one should, for there is nothing more distressing than continued retching with an empty stomach.

If one is predisposed to seasickness, morning walks on deck before breakfast should be dispensed with. One should try to breakfast immediately upon rising, and a little fruit or other light food eaten before rising may be found helpful. What one eats is of small moment; the great thing is to eat; but one should avoid food which one does not like. There is no potency in any particular food in the prevention of seasickness.

Nor is alcohol of any use, unless enough be taken to anesthetize the patient. Indeed it 's far more likely to prove an irritant, especially if the individual be unaccustomed to its use. The value of champagne is largely to allay fear. It is useful.

One should not go to dinner until it is just about to be served, thus avoiding the discomfort of waiting in a stuffy and perhaps overheated dining room. When the meal is over it is well to lie down, rather than go for a tramp on deck in the hope that it will aid digestion.

Warm clothing and wraps should be taken on a sea voyage even in very warm weather. Cold, damp, and foggy weather is apt to be met with on the ocean at any time of the year, and the consequent chilliness, added to that of an unstable vasomotor control, through the labyrinth, is a great cause of discomfort, which may be removed or alleviated by wearing warm outer garments.

If the sea be at all rough and the motion of the vessel appreciable,

the sensitive traveller should lie down at once, as it is easier to accustom oneself to the labyrinthine hyperstimulation in a recumbent position, especially if one adopts the position in which the motion is least felt in the superior canals, *i. e.*, one should lie down as flat as possible—semireclining does not so place the plane of the semicircular canal as to cause the least possible flow of fluid within it. One flat pillow is all that one should use since half-sitting up is as bad as standing up. Chairs should be shifted, if possible, according to the pitch or roll of the vessel.

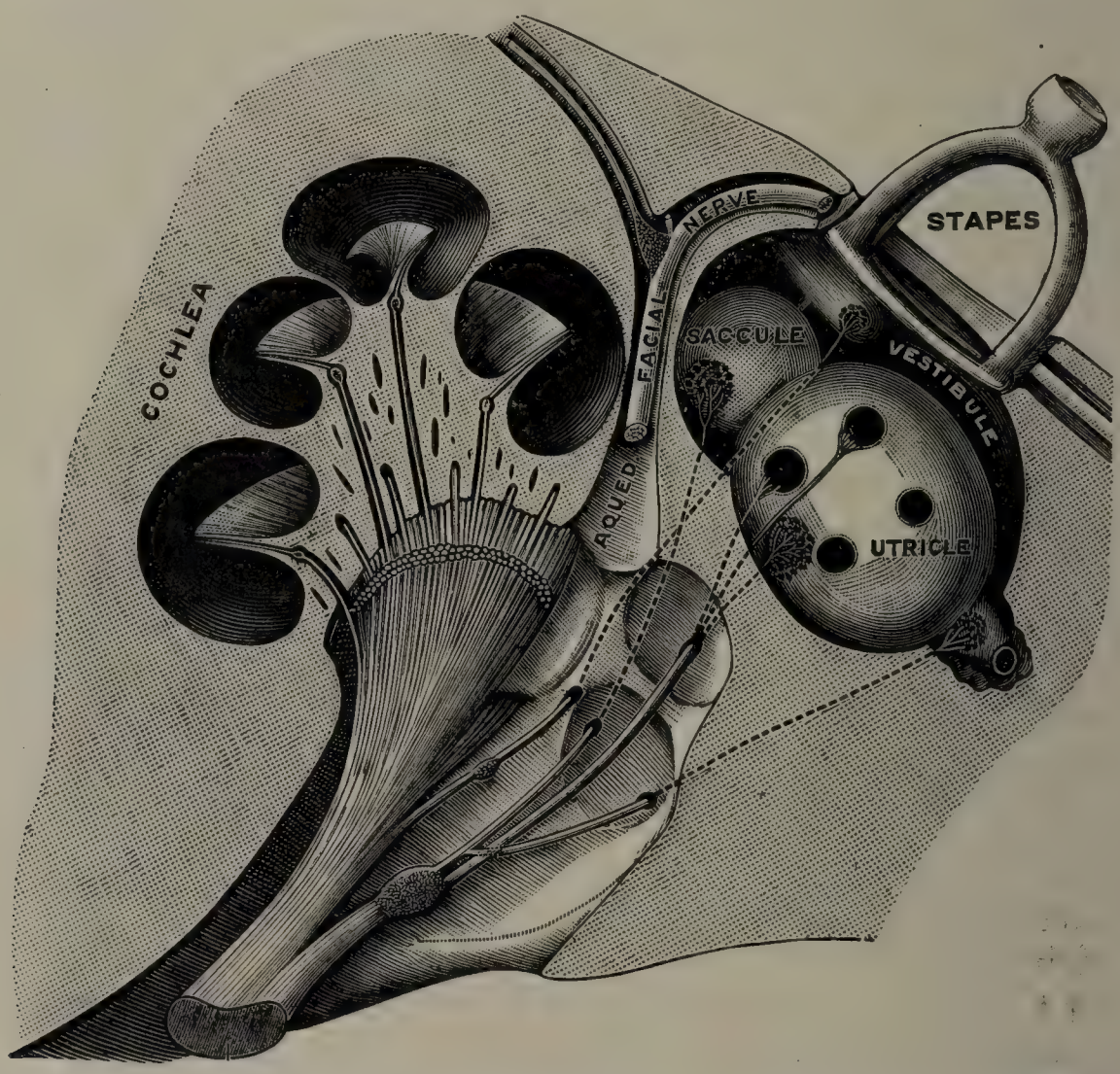


FIG. 201.—Receptor topography of the acoustic nerve. (Semidiagrammatic.) (Testut.)

As soon as the first symptoms of seasickness are felt the patient should lie down, if possible, on deck. As eye movements aid in causing seasickness, one should close the eyes if there is much motion of the ship, so as to relieve the muscles from the constant adjustment necessary in watching a rising and falling horizon, and in very bright weather, colored glasses should be worn to subdue the glare. It is often a good plan to face the cabin rather than the sea.

Reading continuously is rather to be avoided, therefore books should be chosen which will allow one to close one's eyes and meditate. Cards or other games which hold the attention are very helpful.

In making choice of rooms, one should give preference to those in the middle of the boat where the motion is less. To overcome the smells and stuffiness incident to ocean travel, one should keep plenty

of air circulating in one's stateroom, unmindful of drafts, which are of much less consequence that one is prone to think them.

Eating fruits and salads, drinking plenty of liquids, and occasionally taking a pill of aloes, aloes and mastiche, or similar laxative, is generally sufficient to counteract the constipation which is a frequent consequence of the unusual change of habits, especially when one eats very little.

The headache of seasickness is best combated by eating, by coffee, and by small doses of bromids and phenacetin. The widely used headache mixtures incorporating caffein and antipyrin in the elixir of sodium bromid are useful. The sodium salt of barbital in doses of from 8 to 10 grains, given by rectum in suppository, is a very useful remedy in causing sleep and in relieving excessive irritability of the labyrinth.

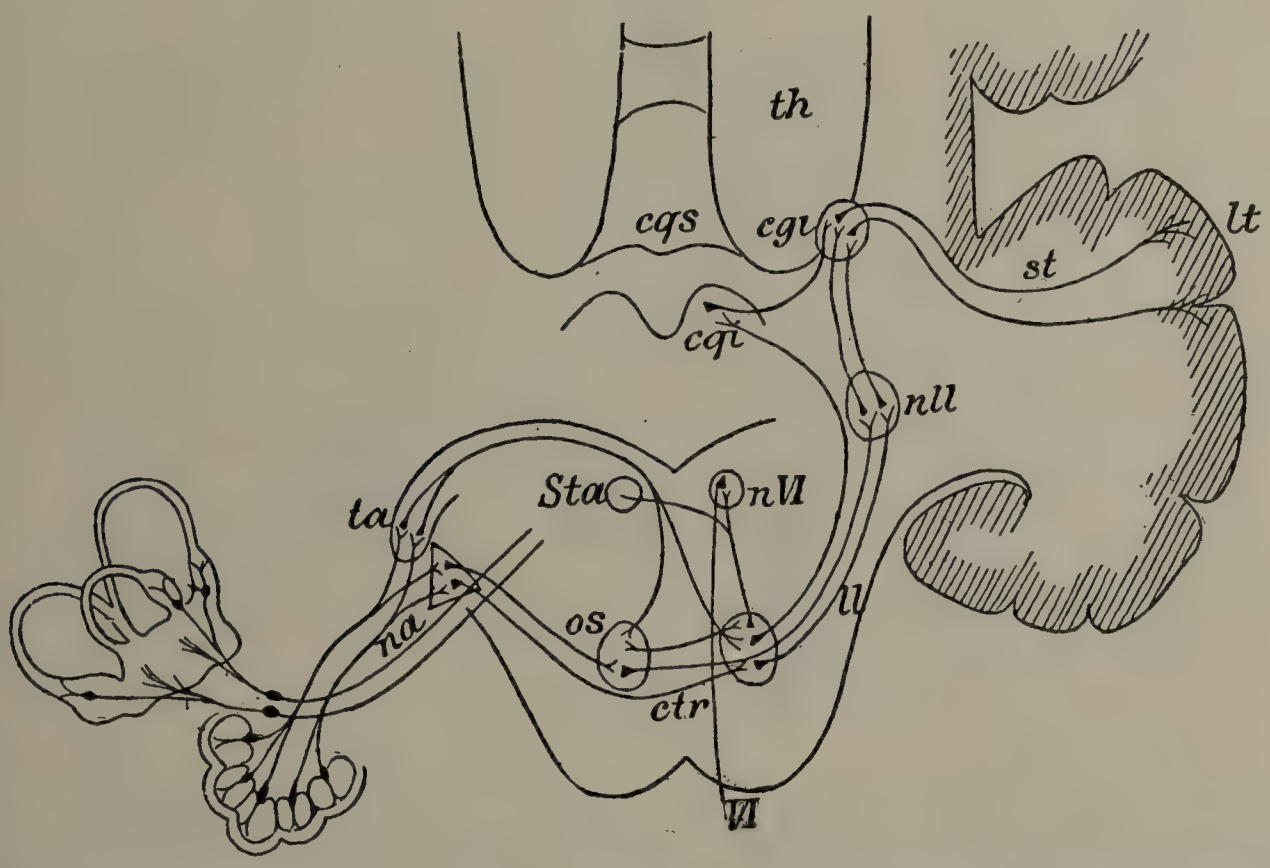


FIG. 202.—Central acoustic paths. *na*, anterior acoustic nucleus; *ta*, acoustic tubercle; *Sta*, acoustic striæ; *os*, superior olive; *ctr*, trapezoid body; *nVI*, abducens nucleus; *nll*, lateral lemniscus; *cqi*, *cqs*, posterior and anterior corpora quadrigemina; *cgi*, external geniculate body; *st*, acoustic path to cortex; *lt*, temporal lobes; *th*, thalamus. (Bechterew.)

Auditory Nerve.—The receptors for sound stimuli lie in the organ of Corti in the cochlea. They respond in the average human to sound stimuli of 11 octaves, *i. e.*, from 10 to 7840 double vibrations. Ordinary conversational or musical sounds usually lie within 16 to 4032 vibrations. Space orientation through sound is purely associative. The chief avenues for sound conduction to the receptors is the auditory canal, but sound conduction is also possible by way of the bones of the skull and in part by other bony structures. From the cochlea the branches coalesce to form the acoustic nerve which, passing in the auditory canal with the facial, enters the medulla at about the cerebellopontine angle. The sensory ganglion is the tuberculum acusticum.

The further course of the pathways is illustrated in Figs. 202 and 203, also see Plate X.

The chief disturbances of the auditory nerve are: various forms of deafness and of tinnitus. Deafness may vary considerably and may be absolute or partial. Certain tones may be cut out and not others, upper or lower tones, sometimes intermediate tones drop out (hearing scotomata, analogous to optic scotomata, are not infrequent in hysterical reactions, dementia precox, in multiple sclerosis, paresis, tabes, etc.). These anomalies of hearing are chiefly peripheral, either in the primary receptors or occasionally in the ganglion. Paracusia (buzzing, whistling, crackling), are for the most part peripheral, but may also be

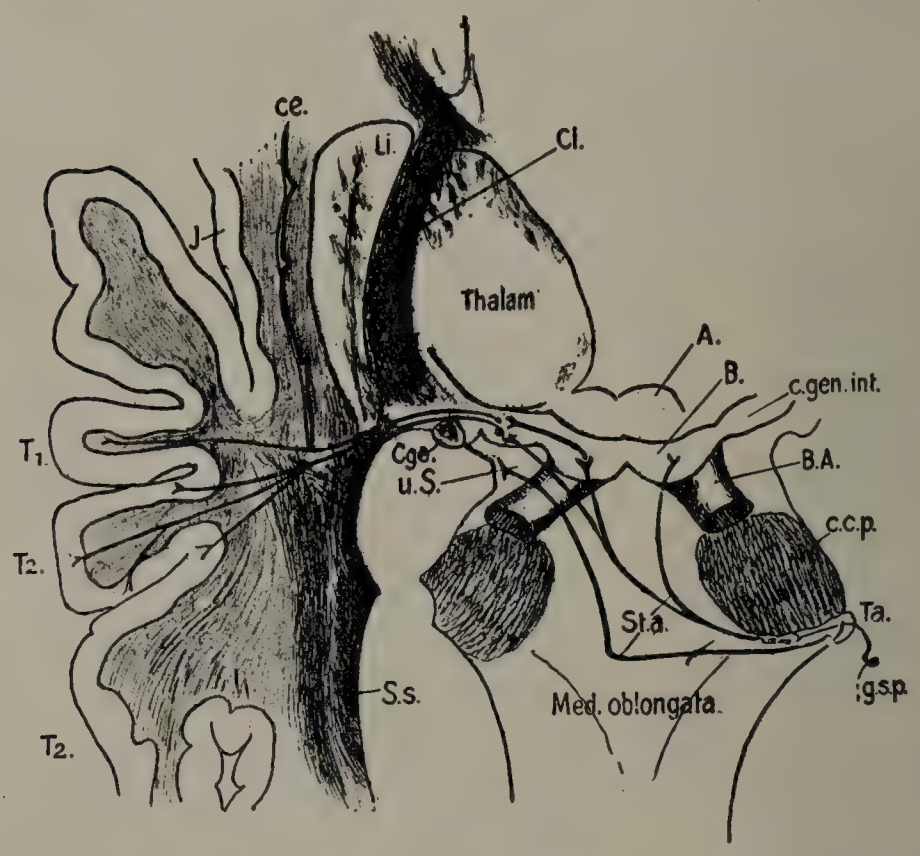


FIG. 203.—Scheme of the central acoustic pathways. T_1, T_2, first and second temporal lobes; *J*, island of Reil; *ce*, claustrum; *Li*, lenticular nucleus; *Ci*, internal capsule; *Thalam.*, thalamus; *u.S.*, median lemniscus; *S.s.*, optic radiations; *V.L.*, posterior quadrigemina; *B.A.*, middle cerebellar peduncle; *c.c.p.*, crus cerebelli ad pontem; *c.gen. int.*, internal geniculate; *Cge.*, external geniculate; *Sta.*, striæ acousticæ; *g.s.p.*, ganglion spirale; *Ta.*, acoustic tubercle. (v. Monakow.)

central, as in psychotic or psychoneurotic syndromes. Psychogenic deafness is a frequent complication of traumata. These are frequently encountered, particularly in war times—detonation deafness. The presence of intact auditory pathways may be detected by the persistence of the auditory-orbicularis reflex (*q. v.*). Also see paragraph on malingering of hearing in Examination Method chapter.

Complete deafness from medullary lesions is rare for anatomical reasons already indicated.

Cortical or subcortical involvement of the auditory pathways cause various forms of amnesic aphasia. (See under Speech Mechanism.)

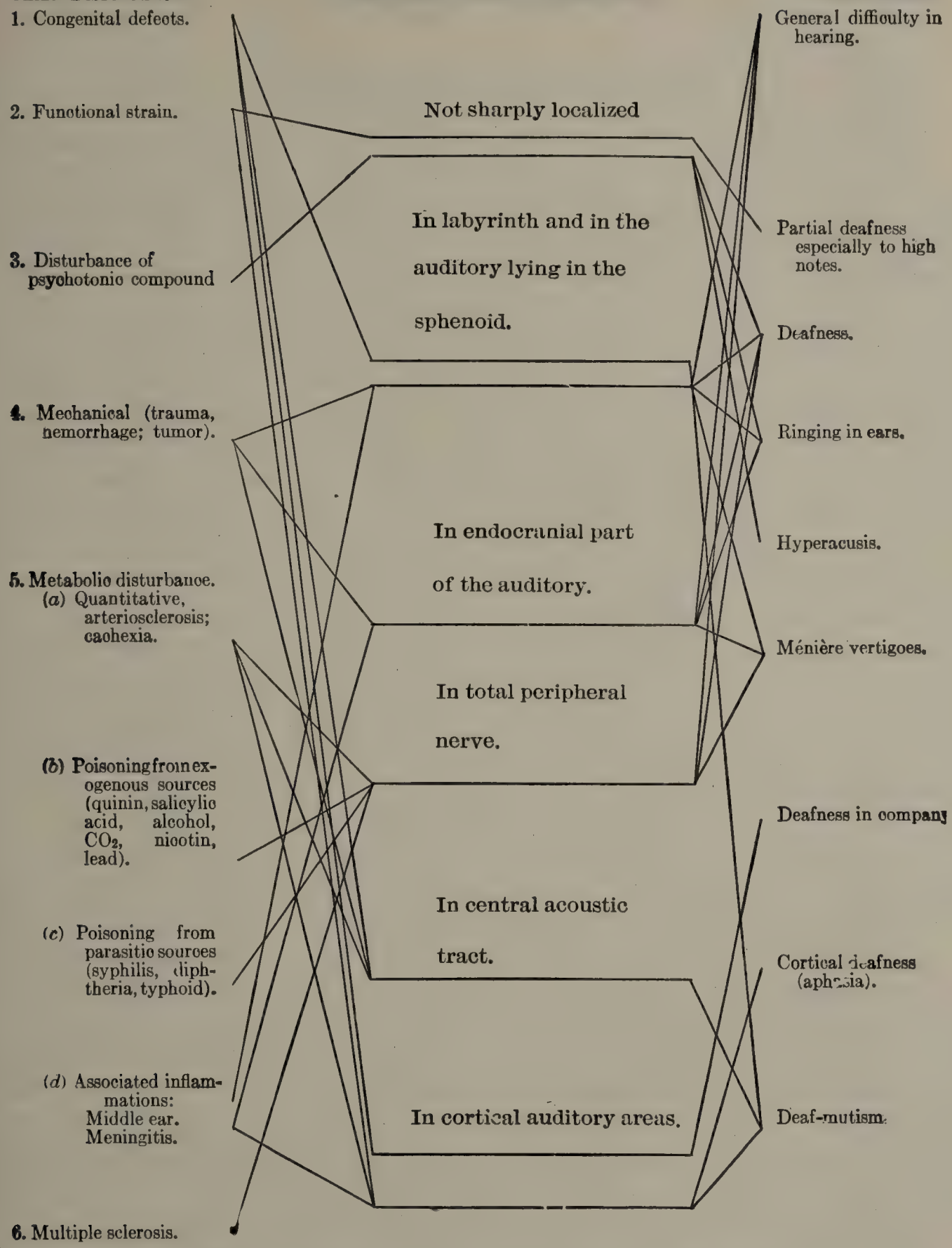


Fig. 204.—Summary of auditory disturbances. (Veraguth.)

Chart for Differentiations of Peripheral and Central Lesions.

Speech test.	Disease of sound-conducting apparatus.	Disease of sound-perception apparatus.
	Deep tones heard worse than higher ones.	Deep tones heard worse or better than higher ones.
Air Conductor for forks c, c^1, c^2, c^3, c^4 and for Galton's whistle.	Deep tones heard worse than higher ones. Lower tone threshold raised.	High tones heard worse than deep ones. Upper tone threshold lowered.
Weber (c^1).	Median or localized in worse ear.	Median or localized in better ear.
Schwabach (c^1).	Lengthened.	Rarely normal, mostly shortened.
Rinné (c. c^1, c^2).	Negative. According to grade of retardation only for c. or for c. and the higher tones to c^2.	Positive.

VAGUS NERVE DISTURBANCES.

The vegetative disturbances of the vagus have already been discussed but in surgical injuries of the neck, fractures of the skull, etc., a number of vagus signs, associated with other cranial nerve disorders, are observed. Vernet[1] has called attention to some of the more striking features of vagus involvement which have thrown fresh light on certain points in its physiology. Clinically the following are to be observed: (1) Cases of hemianesthesia or of hemihyperesthesia of the soft palate, pharynx, and larynx (*a*) without sensory changes in the trigeminal distribution, (*b*) without paralysis of the glossopharyngeal nerve (paralysis of the superior constrictor [Vernet] or gustatory disorders); (2) cases of glossopharyngeal paralysis with profound alteration of the sense of taste without concomitant sensory disturbances of the soft palate or pharynx of the same side. In cases of paralysis of the last four crania nerves (9 to 12)—in which the trigeminus nerve is not involved—Vernet has found that the zone of hemianesthesia or of hemihyperesthesia of the soft palate extends over the soft part of the velum only (at the level of the arc between the uvula and the faucial pillars) without in any way affecting the mucous membrane of the palatine arch. Unilateral injury to the sensory nerves of the soft palate, pharynx, and larynx arise from a corresponding injury to the vagus nerve in its peripheral course. These sensory disorders may be accompanied by certain symptoms which may throw light on the localization of the lesion. Vernet groups these symptoms under five headings: (1) The state of the auricular branch of the vagus. Escat's "sign of the tragus" or hyperesthesia of this branch signifies irritation of the vagus; in some patients it is so marked that cough is produced by simple pressure on the hinder wall of the external meatus of the injured side. In a paralysis of the vagus, revealed by a hemianesthesia or by a palato-pharyngo-laryngeal hemihyperesthesia, there is present also anesthesia of the auricular branch of the vagus of the same side; the limits of the zone of anesthesia can be accurately determined by exploring with a pin both the healthy and the paralyzed side. (2) Pain. This may be spontaneous or induced. It is felt on the paralyzed side. Spontaneous pain is felt as a painful feeling of tickling or constriction. Pain can be induced by pressure on the wing of the thyroid cartilage on the injured side. Vernet has found it in every case of vagus irritation. There is a true neuralgia of the vagus, characterized by subjective and objective sensory signs at the level of the soft palate, pharynx, and larynx. Severe pharyngeal pain cured by surgical division of the pharyngeal branch of the vagus or the vagus itself is a corollary. (3) Cough. This is one of the most constant symptoms of vagus irritation; it occurs as a coughing fit simulating pertussis (Escat); it may be spontaneous or induced. It may be induced by pressure on the front of the sternocleidomastoid behind the angle of the jaw on the

[1] Paralysis of the Vagus Nerve, Med. Rev. of Reviews, June, 1919, **25**, 330.

paralyzed side. (4) Respiratory disorders, (*a*) the exertion-dyspnea, (*b*) the pseudoasthmatic. The exertion-tachypnea depends on a hyperexcitability or irritation of the vagus. In wounds of the cervical vagus this symptom is present as long as the hyperesthesia persists. It is brought on by any slight exertion. The pseudoasthmatic type of dyspnea is a frequent accompaniment of destructive lesions of the vagus. The patients usually complain of a slight dyspnea, chiefly nocturnal; they may have to sit up, or may be suddenly wakened by this symptom when lying on the uninjured side. These disturbances are probably reflex, and the vagus is to be considered as the sensory nerve of the lung, and the spinal accessory as the broncho-pulmonary motor nerve. (5) Disturbances of salivation. In nearly all of the war-wounded cases salivary disorders occured at least during the first few days after the injury; hypersecretion of saliva indicates vagus irritation, deficiency of secretion, vagus palsy. The vagus takes no part in the motor innervation of the palato-pharyngo-laryngeal muscles, the motor innervation being supplied by the internal branch of the spinal accessory; and the vagus must be regarded as a sensory nerve only, even the cardio-moderator fibers being branches of the internal branch of the spinal accessory nerve.

Syndrome of the Posterior Lacerated Foramen.[1]—This syndrome, widely observed during the world war occurs from penetrating wounds just beneath the lower jaw below the base of the skull in front of the anterior part of the atlas to the right of the middle line. Palsy of the right ninth, tenth, eleventh, and twelfth cranial nerves thus occur. It is shown by difficulty in swallowing solids; paralysis of the right half of the constrictor superior pharyngis (Vernet's "curtain movement," *i. e.*, elevation of the sound side of the pharynx during phonation, due to the unantagonized action of the normal left half of the superior constrictor); agenesia of right hinder third of tongue; hemianesthesia of right velum palati, palate, and larynx; salivary hypersecretion which gradually disappeared; cough on swallowing; dyspnea with slowed respiration; palsy of right velum palati; palsy of right half of larynx with vocal cord in cadaveric position; acceleration of pulse; palsy of right sternocleidomastoid and trapezius which rapidly atrophied; and by palsy of right half of tongue, with slight atrophy. This syndrome occasionally follows the mastoid operation.[2]

DISORDERS OF THE TASTE APPARATUS.

Glossopalatine and Glossopharyngeal Nerves.—The taste apparatus is a highly complex one. It is intimately bound up with the olfactory apparatus and in many respects its anatomical developments have followed along homologous lines. Taste discrimination is largely a

[1] Laurens, P.: Associated Laryngeal Palsies from Injury to the Four Hindmost Cranial Nerves, Rev. de Laryng., Otol., et Rhinol., November 30, 1919, 624.

[2] Lannois and Molinié: Paralysis of the Four Hindmost Cranial Nerves Following Operation for Acute Mastoiditis, Lyon Médical, April, 1919, **128**, 193.

chemical differentiation which from the lower animals up has become more and more circumscribed. In fishes the skin contains taste buds which have certain powers of differentiation and the gills of fishes also contain receptors to react to chemical stimuli. The comparative physiology of taste cannot be followed further here, important though it be, for an understanding of the different physiological functions of man in that extremely important of all vegetative needs, food selection.[1]

The chief receptors for taste in man are distributed in the tongue, the sides of the mouth at the lateral edge of the tongue, the posterior part of the mouth and in parts of the nasopharynx. These taste buds are chiefly located in the fungiform, the vallate and circumvallate papillæ. The receptor organ proper, by which chemical stimuli are reacted to and provide means of chemical discrimination is the gustatory filament from which the nerve fiber proceeds to make up the two chief nerve structures destined to carry the various energy stimuli, the glossopalatine and the glossopharyngeal. Vegetative fibers are closely intermingled in the peripheral course and cannot be more fully discussed. (See Winkler, Kappers, Cajal, etc.).

The first synapses are located in and around the papillæ and present many analogies with the olfactory synapses of the Malpighian glomeruli of the smell apparatus, and the neuro-epithelial synapses of the light-receiving apparatus. Both vegetative and sensorimotor synapses are probably present already combined.

The centripetal system begins with the perigemmal plexus and both types of nerve fibers and cells are found in its course.

The discussion as to the taste nerves has undergone many vicissitudes, but it seems fairly well established that, apart from the vegetative systems, the anterior two-thirds of the tongue gives rise to a special gustatory nerve, the glossopalatine, and the posterior part of the buccal cavity gives rise to the gustatory parts of the glossopharyngeal nerve. The trigeminus probably carries no taste fibers. It does carry tactile sensations from the tongue, however, which tactile sensations are often mistaken by patients, or confused with taste sensations. There exists a certain correlation between the two sets of sensations: the tactile are largely exteroceptive, whereas the fibers of the glossopalatine have an interoceptive significance.

The *glossopalatine* nerve is made up of the peripheral branches from the anterior two-thirds of the tongue, the chorda tympani, the intermediary nerve of Wrisberg, and its chief ganglionic synapse is the geniculate. Certain gustatory paths from the nose, and the palate possibly pass by way of the sphenopalatine ganglion, the vidian nerves, and the large superficial petrosal to reach the glossopalatine.

The *glossopharyngeal* carries the major portion of the taste fibers from the circumvallate papillæ of the posterior part of the tongue and buccal cavity. Certain fibers from the pharynx seem to traverse by

[1] Kappers: Der Geschmack, 1914. See also Smith, G. E.: The Cerebral Cortex. Croonian Lectures, 1919. Bean: The Cephalic Nerves, Anatomical Record, July, 1913, See works of Bethe and Skramlik, loc. cit.

way of the superior laryngeal branch of the vagus, but the larger portion pass by the dorsal radicular root of the glossopharyngeal to the petrosal ganglion.

The third (?) neuron proceeds from the petrosal and geniculate ganglia to make up a circumscribed tract, the tractus solitarius, which runs along beneath the ependyma of the fourth ventricle.

Winkler distinguishes at least four sets of fibers in the tractus solitarius:[1] (1) Some small myelinated fibers, coming from the region of the skin innervated by the vagus or the glossopharyngeal. (2) A larger group of larger myelinated fibers follow the tractus solitarius to form synapses in its ventral nucleus. These carry proprioceptive impulses and enter into the formation of the median lemniscus. (3) A still larger group of very fine fibers myelinated and unmyelinated,



FIG. 205.—The taste pathways. (Winkler.)

entering the dorsal nucleus in part and other nuclei, the sensory nuclei of the IX, X the nucleus rotundus X and the nucleus commissuralis, XI. These fibers conduct interoceptive impressions derived from chemical receptors of the internal surfaces of the body determining very important conditioned reflexes for the digestive functions, saliva, gastric juice, etc., and are related to the vegetative reactions of vomiting, hiccoughing, strangling, etc. (4) A group of fine fibers with little or no myelin sheathing, which coming from the glossopalatine and glossopharyngeal nerves are the chief gustatory fibers. This portion of the tractus solitarius containing chiefly the gustatory fibers is termed the pars prævagalis tractus solitarii and enters the primary gustatory nucleus. This nucleus is a gelatinous circumscribed mass situated

[1] Winkler: Manual de Neurologie, vol. 1.

along the para prævagalis between the nucleus of the trigeminus and the dorsal sensory nucleus of the vagus.

The mesencephalic tract seems to pass from the gustatory nucleus by way of fine myelinated fibers in the field of the tractus dorsali tegmenti of Schultz, and pursues a course somewhat analogous to the fibers of the olfactory apparatus, both forming synapses with the ganglion dorsalis tegmenti proximalis (Gudden).

The synaptic collaterals of this tract are of considerable importance. Those to the hypoglossal nucleus furnish stimuli for projicient action of the tongue to respond to the conditioned reflexes of taste and of smell. A tractus reuniens of Fuse permits salivary reflexes to the same stimuli. An important set of fibers are in connection with the vegetative (autonomic) nucleus of the vagus. Vascular, gastric, cardiac and visceral vegetative conditioned reflex possibilities pass this way. The cortical synapses for taste are not completely worked out. In all probability they are contained in the rhinencephalon[1] reaching them by paths similar to, if not identical with the olfactory.

The tests for taste have already been described in the examination methods.[2]

The chief disturbances are loss of taste of the glossopalatine and glossopharyngeal regions respectively, hyperesthesiæ, various vegetative reflex association disorders, often termed subjective disorders of taste.

Peripheral lesions of the glossopalatine, as in the course of the chorda tympani in the auditory canal cause loss of taste in the anterior portion of the tongue. The salivary secretions may or may not be involved at the same time from simultaneous involvement of vegetative fibers. Peripheral lesions of the glossopharyngeal (rare, save from war wounds) causes loss of taste of the posterior third of the tongue and the pharynx.

Total loss of or one-sided loss of taste may occur from lesions of the tractus solitarius (syphilis, tumor, multiple sclerosis, syringomyelia or from involvement of the primary gustatory nucleus from similar processes (influenza or poliomyelitis, etc.).

Taste and smell anomalies are usually combined in lesions from the mesencephalic to the cortical synapses (corpus mamillaria, tractus tegmenti dorsalis of Schultz and from rhinencephalon lesions (Ammon's horn); taste alterations in epilepsy resembling smell hallucinations. A large group of taste reflex perversions are found in vagotonic individuals chiefly of gastric origin.

Irregular and complex central lesions may occur in encephalitis, syphilis,[3] with difficulties in swallowing, anesthesia of pharynx, posterior part of tongue, tonsils, etc.

[1] Henschen, S. E.: Ueber die Geruchs und Geschmackszentren. Monat. f. Psych. u. N., 1919, **45**, 121.

[2] See also Frankl-Hochwart, Die nervöse Erkrankungen des Geschmackes und Geruches.

[3] Alpers, B.: Isolated Bilateral Paralysis of Glossopharyngeal Neuritis Due to Cerebral Syphilis, Am. Jour. Med. Sci., May, 1926, p. 740.

Psychogenic taste modifications, such as metallic tastes, interpreted as poisons, are frequent in hysterical conversions and in certain projections of paranoid individuals. (Cyclothymias, various compulsion neuroses.)

DISEASES OF THE LARYNGEAL NERVES.

Laryngeal Disorders.—The laryngeal muscles are supplied by the inferior or recurrent laryngeal nerve. The cortical synapses are as yet not definitely known in spite of the immense amount of experimentation.[1] That they are in the frontal region seems undoubted but where is uncertain. The conduction paths pass with other corticomedullary fibers through the knee of the internal capsule, apparently near the cortico-medullary spinal pathways for voluntary breathing. The medullary stations are better known, and correspond with the nucleus ambiguus. Laryngeal respiratory movements have their bulbar nuclei in the nucleus reticularis. Both nuclei are intimately associated.

An interesting pathological series from Ziehen's clinic[2] from a patient with tabes and complete laryngeal palsy showed degenerative changes in the nucleus ambiguus. The peripheral fibers seem definitely to pass with the vagus rather than with the spinal accessory.

The larynx also has a rich and complicated vegetative innervation.

In unilateral paralysis of the vagus, usually peripheral or bulbar, at times capsular (Avellis syndrome), there is an associated anesthesia of the paralyzed side. In recurrent laryngeal palsy, from neuritis, aortic aneurism, involvement by carcinoma in neck, poliomyelitis, etc., the voice is rough or harsh, the vocal cord in immobile, half-way fixed between abduction and adduction. In double-sided palsy the phonation is lost.

The chief laryngeal palsies are (1) abductor, unilateral or bilateral, (2) adductor, and (3) thyroarytenoid. In unilateral abductor palsy the voice is unaffected or it breaks readily or may be harsh at times, the involved vocal cord is immobile during aspiration. In the bilateral palsies the voice is unchanged, inspiration is made difficult and inspiration choking and coughing frequent.

Adductor palsy is frequently psychogenic. The patient loses the voice and talks in a whisper. The cords can move outward but do not come together.

Thyroarytenoid palsy causes hoarseness, the cords are orally margined although freely movable. The complexities of neurological laryngeal disturbances would require a volume to describe and must be sought for in special works.

[1] v. H. Grabower: Zeitschr. f. N. u. P., ref. l, p. 611.
[2] Wyscheslawtzewa: Berlin Thesis, 1909.

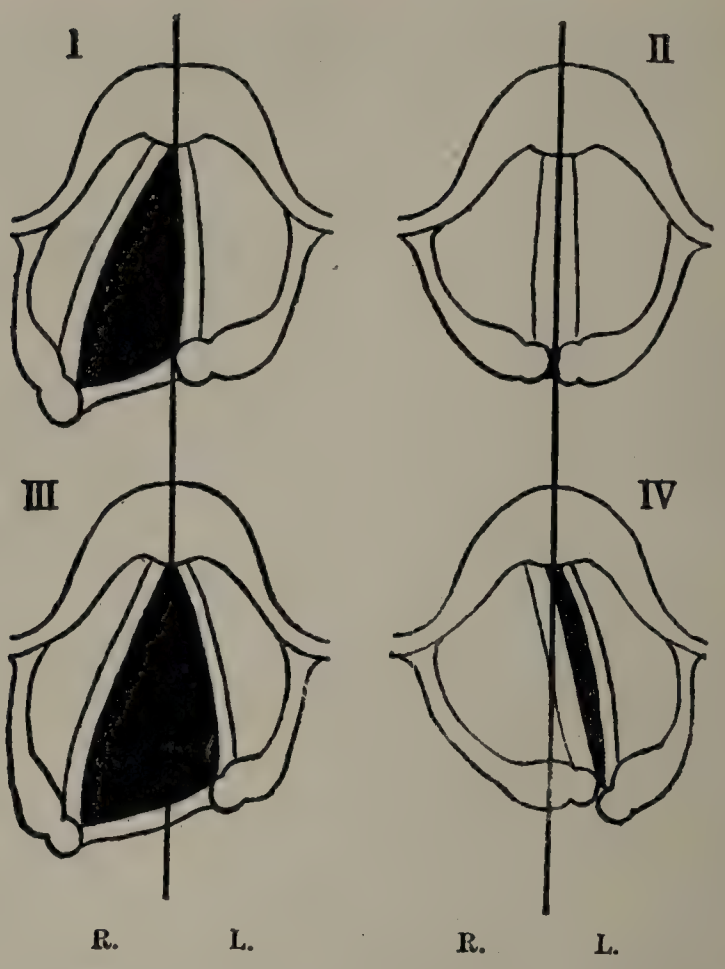


FIG. 206.—Organic laryngeal paralyses. *I*, left adductor paralysis during inspiration; *II*, left adductor paralysis during phonation; *III*, left recurrent laryngeal paralysis, during inspiration; *IV*, left recurrent laryngeal paralysis, during phonation. (Barwell.)

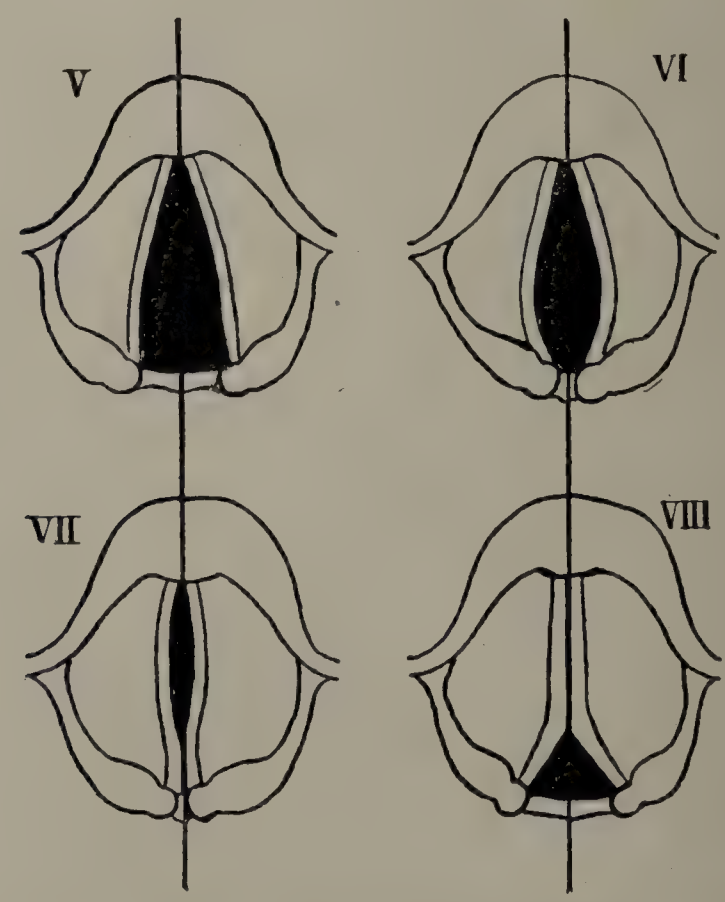


FIG. 207.—Psychogenic laryngeal paralyses. *V*, abductor paresis; all the adductors affected—phonation; *VI*, adductor paresis; arytenoideus unaffected—phonation; *VII*, Paralysis of the thyroarytenoide—phonation; *VIII*, Paralysis of the arytenoideus—phonation. (Barwell.)

DISEASES OF THE SPINAL ACCESSORY AND HYPOGLOSSAL NERVES.

Accessorius.—The spinal accessory nerve innervates the sternocleidomastoid and the upper fibers of the trapezius. Branches to the vagus are known. Its cortical synapses are not definitely localized. The supranuclear pathways pass through the internal capsule to end in part in the medulla near the olive and vagus nuclei, in part in the anterior horns of the six upper cervical "spinal" segments. The combined branch passes by way of the jugular foramen to its muscle distribution, being combined with vegetative fibers from the cervical plexus.



FIG. 208.—Spasmodic torticollis. Head drawn backward and chin up, due to involvement of the right trapezius, probably with certain deep neck muscles, in addition to the manifest spasm of the sternomastoid. (Massachusetts General Hospital.)

The chief lesions causing disorder of the spinal accessory functions are traumata (bullets, operations for tuberculous glands), lesions of the cervical cord, syringomyelia of the cervical vertebræ, multiple sclerosis, neuritides and poliomyelitides. See Chapter on *Fracture of the Base of Skull* for more complete discussion of a number of complicating lesions of the pneumogastric, spinal accessory, glassopharyngeus, etc.

Clinical.—Cortical disturbances (first motor neuron) cause irregular and spasmodic actions. These are seen in certain epilepsies, conditioned by cerebral syphilis, multiple sclerosis or other brain disorder.

The various tics (wry-neck, etc.) are cortical, mostly psychogenic in origin. They represent compulsion neuroses for the most part. Some may represent after effects of an encephalitis. (See Psychoneuroses.)

Nuclear affections of the accessorius are apparently rare.

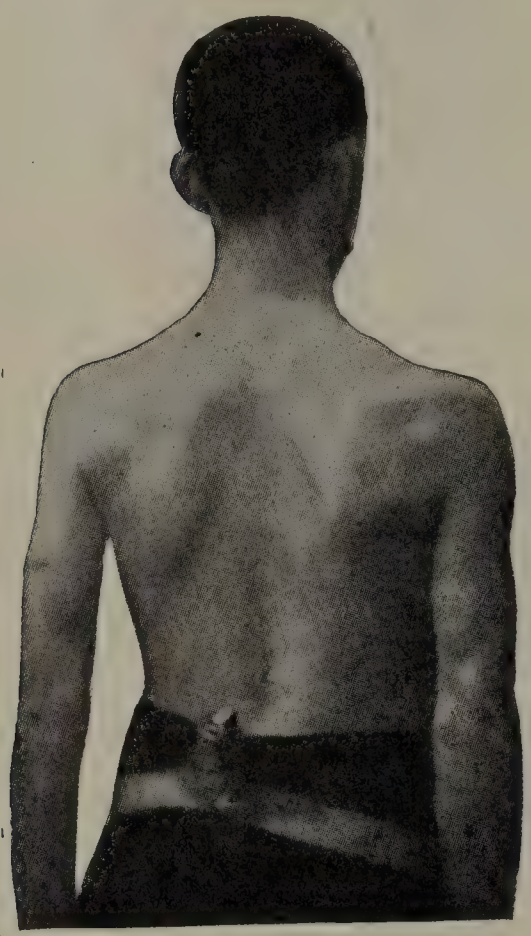


FIG. 209.—Paralysis of right spinal accessory nerve.

Peripheral palsies are not infrequent and are due to various injuries, direct, through disease of the base of the skull, syphilis, osteitis. They cause degrees of loss of power to pull the face to one side, with tendency to contraction of the opposite side (caput obstipum). Electrical changes, R. D., atrophy, loss of reflexes, are present in the nuclear and peripheral palsies, but are absent in the central palsies or centrally induced torticollis. Trapezius palsy causes an altered neck line from prominence of the levator anguli scapulæ, the scapula is also displaced outward and downward and rotated outward, the inner border running upward and outward rather than parallel with the spine.

Treatment will vary with cause. It should be emphasized that the surgical treatment of spasmodic torticollis, which in the overwhelming majority of cases is a psychogenic reaction, usually a compulsion neurosis, is useless. Psychoanalysis and reëducation have been much more valuable.

Hypoglossus.—The hypoglossal mediates somatic motor fibers to the tongue and to the sternohyoid and sternothyroid muscles. Vasoconstrictor fibers from superior cervical ganglion join. Proprioceptor fibers are also present, intrinsic and from Roller's nucleus. The cortical origins lie in the lower portion of the central convolutions.

The supranuclear pathways are followed with considerable difficulty. In the cerebral peduncles they lie in the center somewhat more median than the facial; within the internal capsule they lie at the knee. The supranuclear pathways decussate freely and make their medullary synapse (nucleus of the hypoglossus) in the lower two-thirds of the medulla, stretching as far down as the pyramidal crossing, ventrally from the central canal to the midline. At least ten to fifteen root bundles pass from the hypoglossal nuclei between the pyramidal tracts and the olive, and join together for a short distance within the hypoglossal canal, at the orifice of which the hypoglossal vein, which is in connection with the occipital sinus, surrounds it. The canal is narrow and short—less than half an inch—lying close to the occipito-atlantic articulation, at a place where fracture of the base of the skull is very apt to affect it.

At its exit from the skull the hypoglossal lies median to and dorsal of the vagus and of the internal jugular vein, proceeds laterally, passes

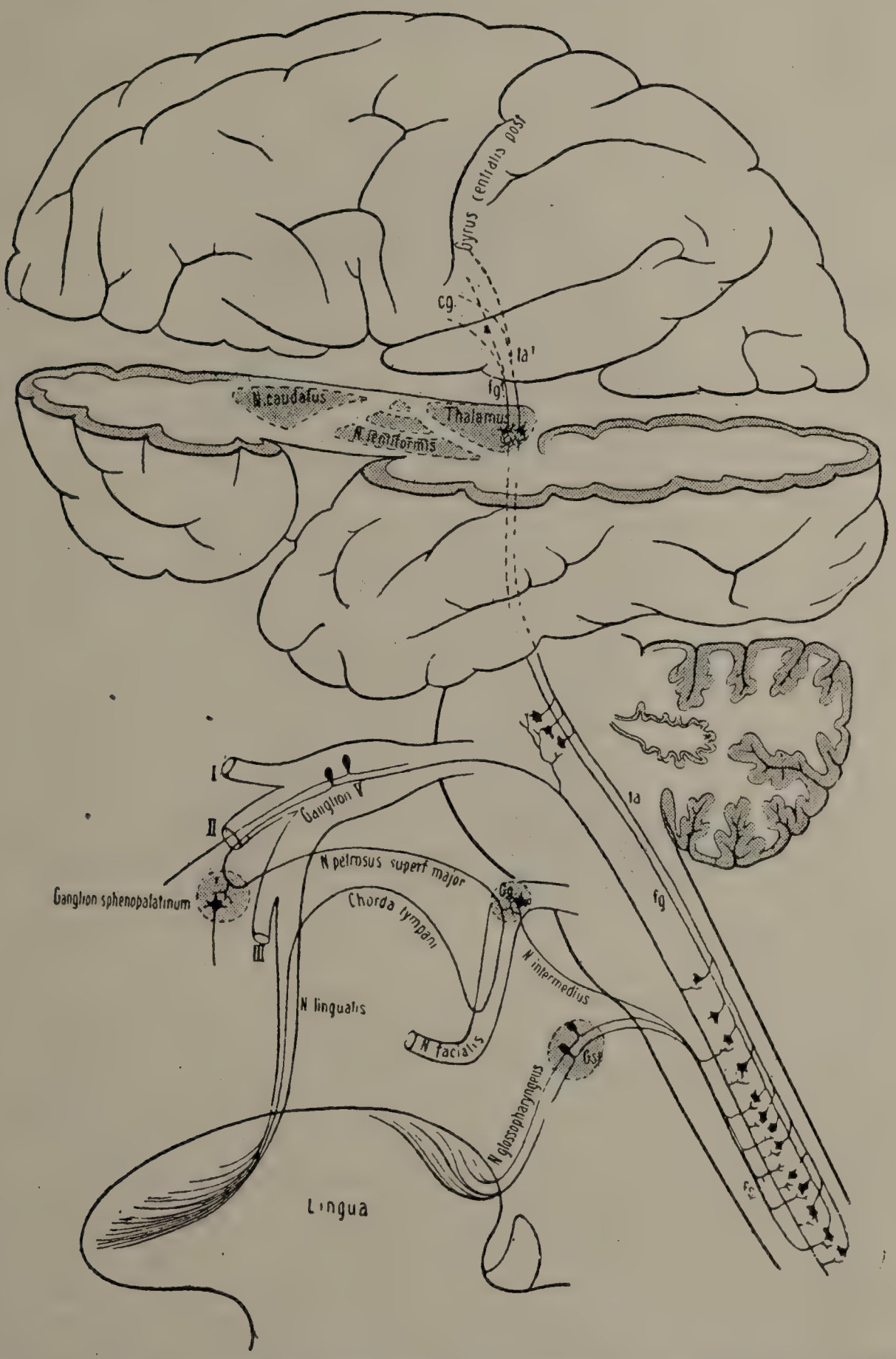


FIG. 210.—Pathways of the taste fibers. *I*, ophthalmic branch of *V*; *II*, maxillary branch; *III*, mandibular branch; *cg*, cortical taste area; *fg*, central ascending taste fibers in median lemniscus; *fg*, subcortical paths; *Gg*, geniculate; *Gsp*, jugular and petrosal ganglia of the glossopharyngeus; *ta*, central ascending fibers of trigeminus in median lemniscus; *ta*^{1}, subcortical connections of the thalamus with the inferior posterior central gyrus. (Bechterew.)

beneath the stylohyoid muscle and the posterior belly of the digastric, splits into its various branches, and innervates the muscles already mentioned.

Anastomoses with the ganglion nodosum, superior cervical ganglion, the lingual and first and second cervical nerve take place. A special branch, the ansa hypoglossi is formed by anastomoses of the descending branch of the hypoglossal, and branches from the second and third cervical nerves.

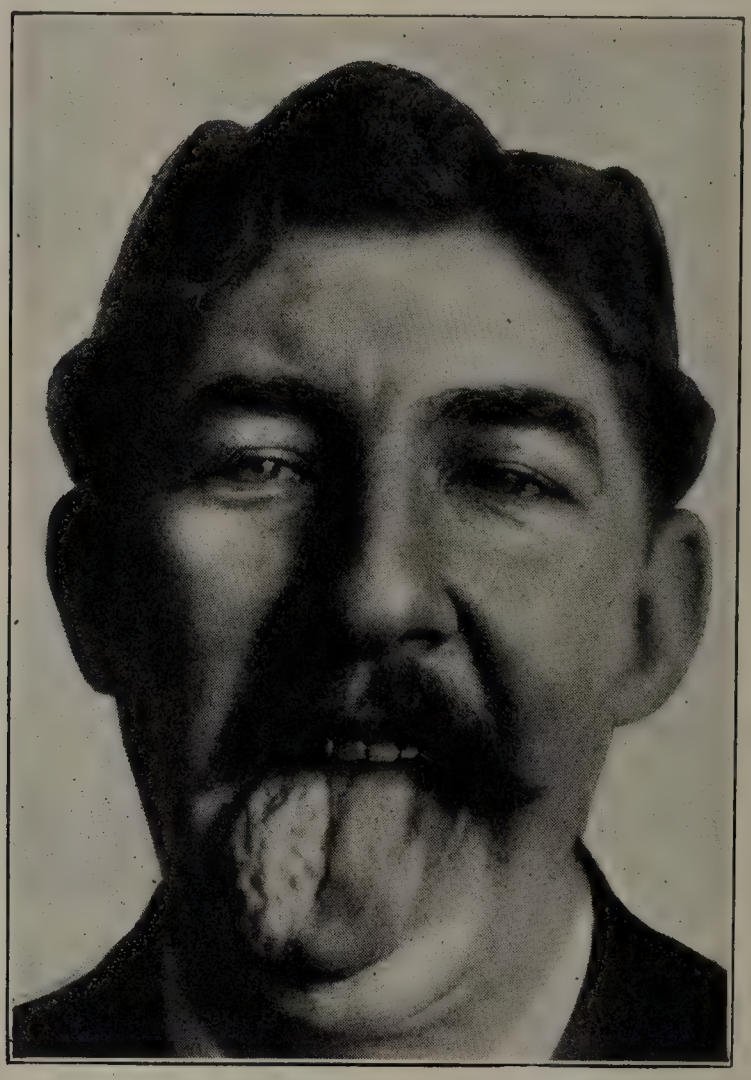


FIG. 211.—Atrophy of right half of tongue. Cerebral syphilis and injury.

The chief physiological functions by symmetrical innervation are as follows: The genioglossus muscle moves the tongue forward and down, the hypoglossus muscle moves the tongue back and up, the styloglossus moves the base of the tongue up and back. In asymmetrical innervation—that is, loss on one side—the combined action of these three muscles causes the tongue to deviate *in toto* to the paralyzed side. The form of the human jaw has been determined by these muscular attachments.[1]

Affection of the longitudinal muscle by symmetrical innervation causes shortening of the tongue, either pulling the top of the tongue up or down. By asymmetrical innervation the anterior portion of the tongue is pushed to the paralyzed side. Any loss of the function of the branch which innervates the transverse muscles brings about a narrowing of the tongue, whereas symmetrical affection of the vertical muscles produces a flattening of the tongue. The geniohyoid raises

[1] Robinson, L.: The Story of the Chin: Knowledge, 1913 (reprinted Smithsonian Ann. Rept., 1914, p. 599.

the hyoid bone, pulling it forward when the lower jaw is fixed, or pulls the lower jaw down. When the hyoid bone is fixed the sterno-hyoid and the thyreohyoid pull on the hyoid bone.

Peripheral lesions of the hypoglossal are the result, usually, of mechanical causes, resulting either from fracture of the base of the skull, from tumors, direct injury or tuberculosis, or dislocation of the upper cervical vertebræ. Cerebrospinal syphilis of long standing, is a frequent cause of peripheral palsies, while poisoning from lead, arsenic, alcohol, carbon monoxide may cause peripheral lesions.

Nuclear and supranuclear affections of the hypoglossal are due to hemorrhage within the medulla. Poliomyelitis, tumors, syphilis, and multiple sclerosis—these are the most frequent cause of nuclear or supranuclear lesions of these nerves.

Isolated cortical lesions cause anarthrias, dysarthrias, tongue ataxias. Psychogenic speech disturbances abound in various forms of stuttering, stammering and other compulsive disorders.

Furthermore, other than the hypoglossus, the tongue is innervated by the trigeminus, the facial and the glossopharyngeal, each mingling in the syndromies present.[1]

Clinical.—The most frequent lesion of the hypoglossal is unilateral. There is atony of the longitudinal muscles of the paralyzed side, and when the tongue lies quiet in the mouth its apex deviates slightly to the non-paralyzed side. The base of the tongue usually rises higher on the paralyzed side than on the sound side as a result of atony of the hypoglossal muscle.

Movements of the tongue are diminished; it becomes difficult to remove food which lies between the teeth and the cheek, and it becomes difficult for the patient to direct the tongue to the teeth on the paralyzed side. On thrusting the tongue out it deviates to the sound side. Speech disturbances are present, especially involving the labials and linguals.

In long-standing disease atrophies develop with fibrillary twitchings and tremors and pronounced motion irregularities. Electrical stimulation shows reaction of degeneration.

In bilateral paralyses speech disturbances are very marked. Chewing and swallowing are rendered difficult, and the movements of the tongue are markedly diminished in all directions.

In nuclear palsies, atrophy and fibrillary twitching are marked, the speech disturbances are pronounced, the chief characteristic being what is termed "hot-potato speech." The patient speaks as though he had a hot morsel in his mouth. Reaction of degeneration is also present. Supranuclear palsies, such as occur in hemiplegia, involve the muscles as a whole, cause deviation of the tongue to the paralyzed side, and other signs of hemiplegia are present. Isolated cortical spasm of the hypoglossal may be present.

[1] Langworthy, O. R.: Problems of Tongue Innervation, Johns Hop. Hosp. Bull., August, 1924. See discussion of Vulpian's Phenomenon. Vulpian. Comp. Royal Acad. Sci., 1863, **56**, 1009.

Psychogenic hypoglossal disturbances are by no means rare. These consist of tongue tics, lisping, stammering, stuttering, of constant tongue movements, such as are seen in hysterias, in patients with dementia precox, compulsion neurosis, and in various paranoid types of thinking.

In hysterical tongue paralyses, which are by no means uncommon, resistance to passive motion of the tongue is seen. There are no electrical changes and the speech disorder is apt to be obvious.

Treatment of hypoglossal disturbances varies according to the cause and is usually purely symptomatic.

Speech Disturbances.—Human expression, articulatory, mimetic, tactile, or by writing, is a highly complicated series of mechanisms. They include all of those movements resulting from optical, auditory or tactile contacts by which communication between individuals is brought about for social purposes. Language as it fully develops is therefore a tool with which one may cut into reality and utilize the facts of nature for purposes of adaptation.

All kinetic speech disturbances may be at first separated into those in which the receptive (sensory) side of the pathways are involved and into those in which the productive (or purely motor) part of the arc is implicated.

On the sensory side one finds the gradual accumulation of experience, chiefly through auditory stimuli with the gradual evolution of speech symbols (language), which stand in the developing psyche for the image of things, ideas or feeling values. Thought is symbolic action.

Optical stimuli—objects, signs, various glyphs, words, letters—unite to form complex types of expression in writing (psychically developed to conquer spatial limitations), while tactile stimuli are an integral part of language from the more restricted side of tactile reading of blind and tactile speech of the deaf and dumb to the sensory stimuli of the movements of the muscles and tongue and lips in speech. A complete analysis of the great complex of sensory factors which ultimately finds outlet in speech symbolism with its infinite psychical implication is not possible here. It would involve the entire problem of the evolution of civilization and culture.[1]

The productive side of the speech mechanism is also complicated. Originally showing itself in the child as a noisy symbol of crying, there is gradually differentiated more and more accurate sounds of expression to meet the needs of hunger and of love. Grunts, laughs, gurgles evolve into more precise formulations, until the rich symbolizations of speech are gained, with all their advantages of precision and efficiency. Here the muscles of the mouth, lips, larynx, chest, abdomen, arms, and pelvis all come into a gradually refining and orderly series of coördinated activities.

[1] Guntzmann: Sprach-Störungen. Various monographs on Aphasia. Schlesinger: Geschichte der Symbol, 1917. Jelliffe: The Symbol as an Energy Container, Jour. Nerv. and Ment. Dis., December, 1919.

As a celebrated French philosopher has phrased it:[1] "If the ants have a language, the signs which compose it must be limited in number, and each of them, once the species is formed, must remain attached to a certain object or a certain operation; the sign is adherent to the thing signified. In human society, on the contrary, manufacture and action are of variable form, and, moreover, each individual must learn his part, because he is not preordained to it by structure. So a language is required which makes it always passing from what is known to what is yet to be known. There must be a language whose signs which, cannot be infinite in number, are extensible to an infinity of things. This tendency of the sign to transfer itself from one object to another is characteristic of human language. It is observable in the little child as soon as he begins to speak. Immediately and naturally he extends the meaning of the words he learns, availing himself of the most accidental connection or the most distant analogy to detach and transfer elsewhere the sign that has been associated in his hearing with a particular object. 'Anything can designate anything' is the latent principle of infantile language." The truth of this is amply confirmed in the studies of symbolism in the psychoneurotic symptoms, the language of dreams and of delusional thinking. These are discussed in the chapters dealing with disorders of social adjustment, Part III.

Many schemes have been devised to set forth graphically some of the phases of these kinetic speech disturbances. One of Veraguth's is here utilized.

Here *1* represents the incoming auditory stimuli (tone, sound, words) with their more or less sharply defined sensorial perceptions gained gradually through experience. They constitute in their totality the various audible components of speech and are constituents of organic, auditory memories. The circle *I* represents such a psychophysiological combination, rather than an anatomical hearing area or zone, which latter is roughly outlined in the first and second temporal convolutions.

Pathway *2* represents the optic as well as the tactile, and kinesthetic neuron chain which conveys to the brain centrals graphic symbols (pictures, diagrams, graphs, letters, etc.). The general assembly place of these is represented by the circle *II*. It represents not an anatomically circumscribed area, but rather a functional capacity, but not unrelated to an optical sensory area in the cuneus and precuneus of the occipital lobes.

Outgoing pathways *3* and *4* represent the motor side of the arcs of expression by all those motions by which the act of articulation with infinite variation, shades and nuances, and those of graphic representation are carried out. Both pathways are related to cortical, bulbar, spinal localizations, which make functional unity possible as speech and writing (in widest sense). These are symbolized by

[1] Bergson: Creative Evolution, Henry Holt & Co., 1911.

circles *III* and *IV*. All of these are brought together in an enlarged concept (circle *V*), which symbolizes the heard, read, spoken, or written mode of expression (words, acts, representation, mimic, etc.).

The lines which bind these various centers represent, therefore, a series of possibilities. Whereas an anatomical substratum underlies these possibilities, no attempt will be made to represent them here.

1. Repetition of words without comprehension—Pathways *1, 5, 3.*
2. Reading aloud without comprehension—Pathways *2, 7, 3.*
3. Writing to dictation—Pathways *1, 6, 4.*
4. Writing to dictation without sense—*2, 8, 4.*
5. When heard word is comprehended—*1, 9.*
6. When read word is comprehended—*2, 12.*
7. Spontaneous speech of an idea—*10, 3.*
8. Spontaneous graphic expression of an idea—*11, 4.*

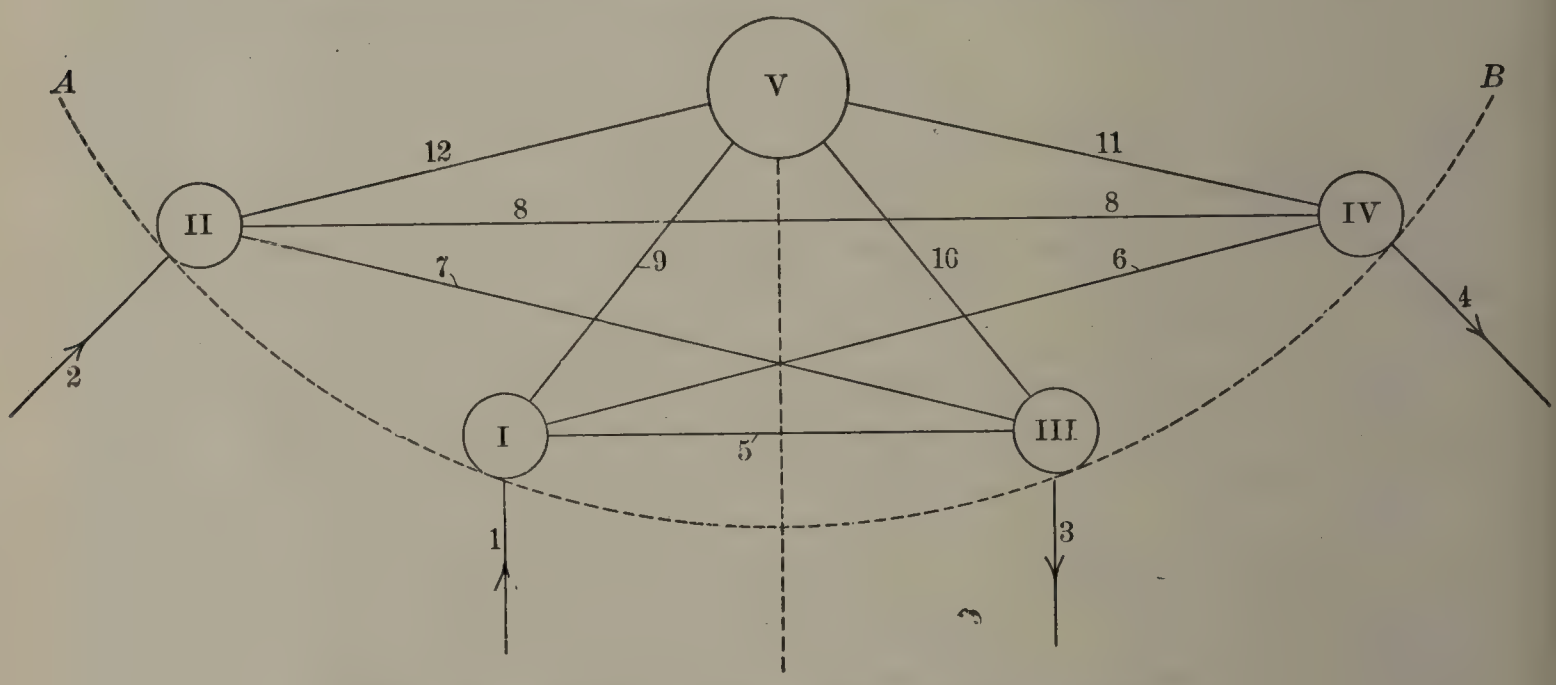


FIG. 212.—Scheme of speech connections. (Veraguth.)

9. When heard word is comprehended and reproduced by speech—*1, 9, 10, 3.*
10. When heard word is comprehended and reproduced graphically—*1, 9, 11, 4.*
11. When read word is comprehended and reproduced by speech—*2, 12, 10, 3.*
12. When read word is comprehended and reproduced graphically—*2, 12, 11, 4.*

The scheme also attempts to show an internal and external speech. At the present time exact correlation between all types of speech disturbance and definite pathways cannot be made. But in the main enough broad facts have accumulated to permit certain fairly exact generalizations. In the first place the general speech mechanisms are located predominantly in the left hemisphere in right-handed individuals. In the left-handed the localization is predominantly in the right hemisphere. Ambidextrous brains are known and educable opposite speech areas are known.

While in the discussion of the aphasias, one speaks of *speech* areas, auditory (temporal), optic (occipital), motor (Broca's convolution) and attempts to localize them, the fact is that the architecture of the brain is so complex, the pathways utilized in the speech mechanisms so widespread, that it is better to speak of *aphasia* areas. These are areas of special predilection for the occurrence of speech disturbances which are more or less stereotyped. These are areas which are supplied more particularly by the 1, 2, 3, 4 branches of the Sylvian artery, distributed to the third frontal convolution and operculum of the central convolution, Broca's area (motor speech asphasias), the insula (Heschl convolution), the posterior part of the first temporal auditory centers (amnesic aphasia) and the angular gyrus, cuneus (optic alexias).

These areas are well shown in v. Monakow's diagram here reproduced.

Clinical Forms.—These may be subdivided into *external* and *internal* speech disturbances.

I. *Deafness* brings about a special form of speech disturbance (deaf-mutism). Even though the speech apparatus be intact, it lacks the dynamic stimuli to be utilized. When speech is acquired, it has a peculiar monotonous quality. Certain forms of mutism from faulty hearing are to be distinguished.

II. *Dysarthrias.*—Used in a broad sense, these include disturbances in speech due to defect in the productive pathway.

(*a*) They may be of purely psychogenic origin, *i. e.*, compulsive ideas, hysterical conversions, psychotic symbol distortion, such as are evidenced by stammering, stuttering, hysterical speech, katatonic speech.

(*b*) Peripheral motor palsies; facial, palate (rhinolalia).

(*c*) Bulbar palsies (mouthful speech), as seen in progressive muscular atrophy (Aran-Duchenne type, often syphilitic) in amyotrophic lateral sclerosis, in acute poliomyelitis, in multiple sclerosis, syringomyelia, in tumors of the medulla and pons and in general paresis.

(*d*) *Pseudobulbar Palsy.*—The earliest clinical record of this syndrome seems to have been made by Magnus.[1] It had resulted from cysts in both paracentral gyri. In 1872 Joffroy and Jolly reported cases but Lepine in 1877 first named the syndrome. It is characterized by dysarthria, dysphagia, hemiparesis, with a short step and stiff gait, and usually some mental deterioration.

It is usually determined by multiple foci which are *mostly* bilateral and involving (*a*) bilateral symmetrical cortical zones of the tongue or lips (operculum of Arnold); (*b*) symmetrical lenticular zones; (*c*) association of right cortical and left lentricular lesions, or the reverse. Less often the lesion may be unilateral, as in capsular and lenticular softenings, centrum ovale lesions at juncture of corpus callosum and the superior extremity of the internal capsule.[2]

[1] Müller's Archiv., 1837, p. 258.

[2] Senise: Annali di Neurologia, 1920, **37**, 3. Jelgersma: Jour. f. Psych. u. Neur., 1918, **23**, 149.

(*e*) In disorders of the muscular tonus mechanism of the midbrain, cerebellar paths, corpora striata, as in acute choreas, Huntington's chorea, paralysis agitans, multiple sclerosis, one finds incoördination or scanning, jerky or monotonous speech.

The disturbances of inner speech are termed *aphasias*. They are here divided into (*A*) Receptor, and (*B*) Productive, which are practically synonymous with sensory and motor aphasia respectively.

Aphasias.—Although a separation of all aphasias into two groups, receptive and productive, is theoretically possible, practically this is not the case. The actual clinical pictures seen vary enormously.

In view of the fact of the millions of years of continuous growth in complexity and of effectiveness of this particular series of purposeful movements, it is no wonder that the many problems connected with speech and the mechanism of its production are still far from being clearly resolved, nor will this be undertaken here.

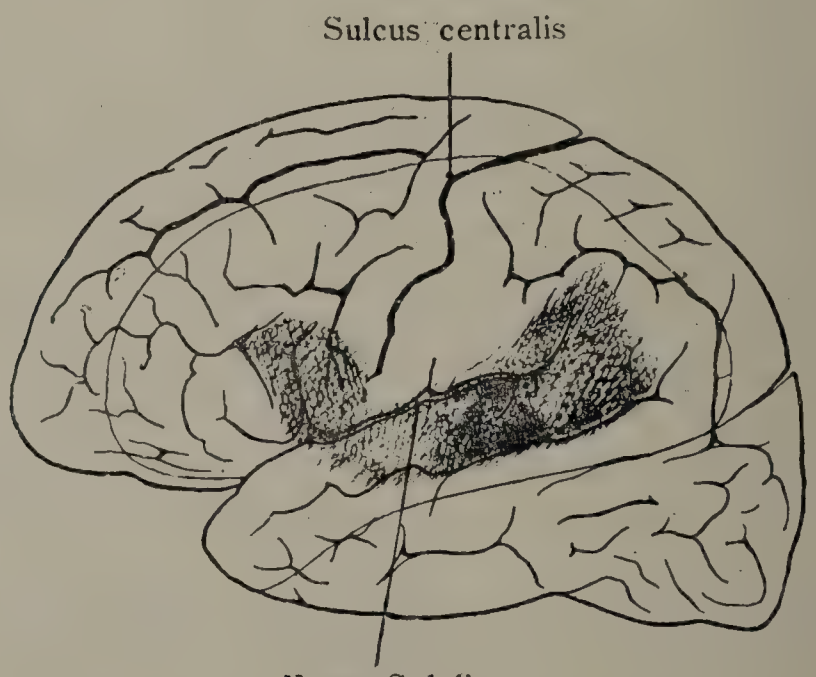


FIG. 213.—The aphasia regions in the left hemisphere. (Veraguth.)

What is here attempted is simply a general sketch of certain disturbances of speech which have for many years been called aphasia. No attempt will be made to detail all of the many interpretations that have been given to the term. Such may be found in large and valuable works upon the subject, notably in the monographs of Elder, Bastian, Head in English; of Kussmaul, Henschen, Wernicke, von Monakow, Niesl von Mayendorf in German; those of Dejerine and Miraille, Marie and Moutier in French and Mingazzini in Italian. The analysis of the problem has been found to be more and more complex, successively more time-consuming, money-costing and scientifically difficult. Each advance has cast into the scrap heap many of the classics of a generation past and it can be said that the problems now can be solved only by the most exacting and rigorous technical methods coupled with most penetrating and comprehensive clinical observations. To completely analyze a single case of aphasia according to present-day

requirements requires years of painstaking observation, two or three years of more or less continuous microscopic technical manipulations, and at the cost of thousands of dollars.

As Adolf Meyer once expressed it, "Now that the North Pole and the South Pole have been discovered, perhaps someone will take interest in that most important undiscovered country, the human brain." Certainly no better expedition could be fitted out than one to explore the uncharted seas of the human speech areas.

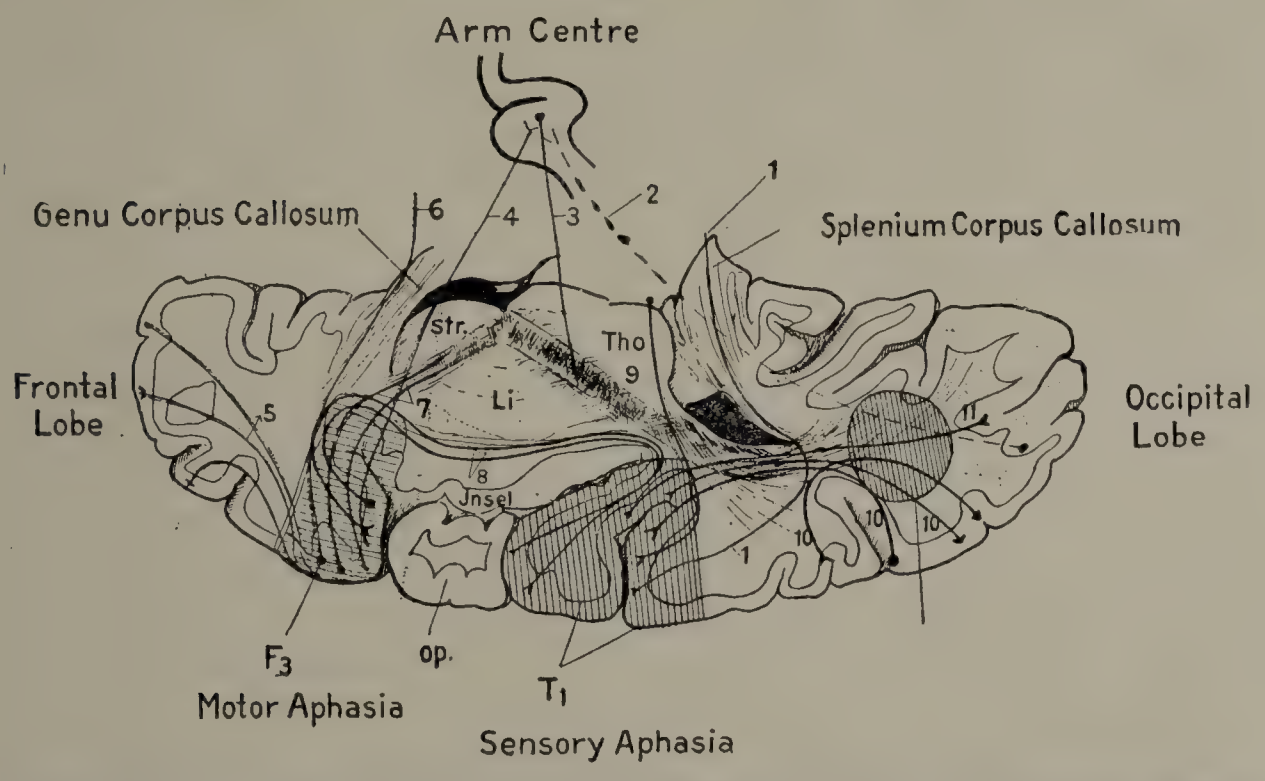


FIG. 214.—Scheme of the chief areas and pathways involved in aphasic disturbances. Nos. *3*, *4*, *5*, *6*, *7*, sensory pathways; Nos. *1*, *2*, *8*, *9*, *10*, *11*, motor pathways. *Str.*, corpus striatum; *Li*, lenticular nucleus; *Tho*, optic thalamus; *op.*, operculum; *1*, path from left temporal to right temporal by way of the corpus callosum; *2*, path from occipital lobe to the arm region; *3*, path from the arm region to the internal capsule and peripherally; *4*, path from temporal lobe to arm region; *5*, paths from the prefrontals to F_3; *6*, connecting path with the right hemisphere by means of the corpus callosum; *7*, paths from F_3 through the internal capsule downward; *8*, connecting paths from F_3 to T_1, operating both ways; *9*, path from the internal geniculate to T_1; *10*, connections between angular and supramarginal gyri and T_1; *11*, path between occipital and T_1. (Veraguth, after v. Monakow.)

History.—The work that the early explorers did is work that cannot be thrown away. It outlined the chief landmarks of the country which now must be left to the skilled pilots of the State institutions, endowed laboratories and university organizations with their corps of skilled technicians, neophytes to whom a year is but a day, and to trained interpreters learned in the difficult charts of brain anatomy. The old-time dabbling in these problems, though dabbling is but a relative term, has gone out of fashion.

To Bouillaud, in 1825, history turns for its first noteworthy find in this field. Tinctured with the notions of Gall, Bouillaud's work was worth while, in that he controverted the long-prevailing and orthodox doctrine of Flourens that the brain had no influence either direct or indirect upon the muscles. Bouillaud maintained that the brain was indispensable for movement and he very roughly localized the

organs of articulate speech in the anterior lobes. As Soury[1] well says, arguing from Bouillaud's own writings of 1847 and 1865, he was not really a seeing pioneer in this limited field, although his work was full of remarkably clear, valuable material. His work was too much colored by the conceptions of Gall, but he was a precursor of Broca who in 1865 really charted the first outlines of the aphasia sea. Bouillaud, however, noted that articulate speech could be abolished without paralysis of any of the muscles of phonation and separated completely motor aphasia from dysarthria. Bouillaud also apparently had an idea of what is spoken of as internal speech, for he wrote as early as 1825 that "the loss of speech depends at times upon the memory of words, at times upon that of the muscular movements of which speech is composed, or what is the same thing, at times upon a lesion of the gray matter and at times of the white substance of the anterior lobes."

Marc Dax, writing in 1836, however, made a series of interesting observations in which he concluded "that not all diseases of the left hemisphere can alter verbal memory but when this memory is altered by disease of the brain it is necessary to seek the cause of the disorder in the left hemisphere." Bouillaud contested the notion which Dax could not support by autopsy findings. Furthermore, Bouillaud, following a custom of the times, offered a prize of 500 francs to him who would show him a single case of the loss of speech described. This was in 1848, and the whole story of the times and Broca's presentation in 1861 is well told in an admirable critical essay by Marie.

The clinical pictures noted by Bouillaud, Gall, by Dax, father and son, Marcé, Jackson, Lelut, Leidat and others, received their anatomical explanation by Broca, in 1861, in his first autopsy case of Leborgne, a man fifty-one years of age who since the age of twenty-one had lost his use of language. He could pronounce only a single syllable which he repeated two or three times in succession, tau, tau, tau. He understood practically everything said to him. A second case, Lelong, soon followed, and Broca, then thirty-seven years of age, made the deduction that the seat of the lesions of motor aphasia (aphemia he called it at that time) was in the third left frontal convolution. These two brains were conserved in the Dupuytren museum at Paris and have been seen by many. In 1863 Broca reported 11 cases in which the left third frontal convolution was involved and in 1865 he presented a general thesis upon the subject. He noted the influence of right-sided lesions in left-handed persons.

Broca defined his aphemia in the following words: "There are individuals in whom the general faculty of language persists unaltered, in whom the auditory apparatus is intact, and where all the muscles, not excepting those of the voice and of articulation, obey the will, and in whom as a result of a cerebral lesion the articulate speech is abolished. This abolition of speech, in individuals who are not paralytics, nor idiots, constitutes a symptom sufficiently distinctive which it seems to

[1] Les Centres Nerveux. A work which every student of neurology should read.

me useful to designate under a special name. I shall call it aphemia (*á*, without, and *φημι*, I speak, I pronounce) for that which these patients lack is solely the faculty of articulating words. They hear and understand all that is said to them; they have their reason, they emit sounds with facility; they can move the tongue, and the lips much more energetically than is necessary to articulate sound and yet, notwithstanding, the response, while they understand perfectly what they would wish to say, is reduced to a few articulatory sounds, always the same and always uttered in the same manner. Their vocabulary, if one can call it such, is composed of a short series of syllables, often of a monosyllable which expresses everything, or rather which expresses nothing, for this unique word is most often a stranger to all vocabularies. Certain patients have not even a vestige of articulate speech; they make vain efforts without pronouncing a syllable."

Going into the anatomical correlations Broca says, "Aphemia, that is to say, the loss of speech without other intellectual disorder, and without any paralysis, has been the consequence of a lesion of one of the frontal lobes. In our patients the site of the lesion was in the second or third left frontal convolution, most probably in the latter. It is then possible that the faculty of articulate speech is located in one or the other of these convolutions." It is not necessary to proceed further with his very temperate statement and his careful wording, in which we can see his desire to support a superior in the Faculty, Bouillaud, and yet not offend him nor the most active believers in the localization of Gall. In 1862 he saw a patient with aphemia from a right-sided lesion which caused him to be cautious. In 1863 Parrol demonstrated a case of extensive destruction of the right third frontal convolution without any speech or intelligence defect, which also contributed much to a very active discussion going on in Paris. Finally in 1865 Broca came out flat-footed and said that the third left frontal convolution was the seat of his aphemia. Thus, to use Moutier's phrase, the "dogma" was created, although it was not by any means accepted by all. All of the acrimony of a great discussion raged but Trousseau gave it the weight of his great authority and it prevailed for many years and, gradually subjected to certain modifications, prevails at the present time.

Then began the great period of electrical stimulation of the cortex. Localization, a fantastic dream for Gall, became a scientific reality for the English physiologists and when in 1870 Fritsch and Hitzig published their studies upon electrical excitability of the cortex an entirely new method of localizing muscular movements and the speech mechanism became possible. Meanwhile the aphasia question was actively studied. Fleury (1865) distinguished Broca's aphemias from what he called aphrasics; they could pronounce but did not use the right meaning. Gardner (1866) separated internal speech trouble from intact ideation. Ogle (1867) fell upon the idea of agraphia and Bastian (1869) really discovered word-deafness. Paraphasia, jargon aphasia, were also described by Jackson and other English writers.

The honor has been given to Wernicke, however, for discovering that the incoming receptive side of the speech mechanism, *i. e.*, the auditory understanding of speech, separated from the hearing of sounds, was of immense importance in solving the problems of speech disturbances and he formulated that form of aphasia which is termed sensory aphasia. These patients heard sounds but they might as well have been Chinese or Choctaw. They had lost their meanings. This was in 1874, and the defect he then stated was due to a defect of the first left temporal convolution.

The aphasia problem, at first comparatively simple, and not yet a general problem, began to become complicated. Wernicke described his zone of language, and began the epoch of complicated hypotheses. There existed a motor aphasia, the aphasia of Broca and due to disturbance of the F_3 left, and a sensory aphasia due to disease of the first temporal of the left side and posterior, in which comprehension of spoken words was lost. This served as a control of the motor center and lesions here produced the various clinical pictures described particularly by the English as amnesic aphasia, paraphasia, agraphia, etc.

A most searching reanalysis of the entire question followed Marie in 1906.[1] The whole problem is extremely technical and involved, and further discussion cannot be entered into here. At all events the work of Marie caused several realignments of former attitudes which have not yet been completely settled.[2] The more recent psychological analysis of the aphasia problem by Head is critically received by Henschen and others,[3] and will be presented here but briefly later.

Clinical Forms.—Aphasia as here very simply outlined consists in a disturbance or loss of speech resulting from a failure to evoke or control the memory of those auditory or visual symbols used in social interchange of concepts or ideas. This loss of control or failure to evoke the proper symbols is due to structural changes involving the complex brain pathways. It has been seen that for speech as for any other volitional act, receptor and effector pathways must be open. The receptor pathways, *i. e.*, the sensory part of the process, include the hearing and the seeing of word symbols. The memories (engrammes) are stored in certain areas or zones which have been termed auditory and visual word areas. Centers is an older term but is somewhat objectionable. The auditory word zone or area is located about the upper surface of the temporal lobe in the anterior transverse gyrus of Heschl and extending also into the adjacent portions of the posterior and of the first temporal convolution.[4] The gyrus angularis

[1] Semaine Médicale, 1906, Nos. 21, 42, 48; see also F. Moutier's Thèse de Paris, L'Aphasie de Broca, 1908, for complete discussion of the whole problem.

[2] Marie: Questions Neurologiques d'actualité, 1922, Chapt. 20, 527.

[3] Head, Brain, 1920, **43**. Head, H.: Aphasia, 1926, 2 vols.

[4] See Henschen's more complete differentiation in a series of quarto monographs. Siebenter—Teil: Ueber motorische Aphasie und Agraphie, Stockholm, 1922, being the latest, with complete literature. Also see v. Monakow, Lokalisation in Grosshirn, 1914. Mingazzini: Anatomia Clinica, 1913. The most interesting recent work is that of Henry Head's, 2 vol., work on Aphasia, London, 1926, also small volume by Wilson, Psyche Monographs, 1926.

serves as a visual area for those who have learned to read. Lesions, which occupy these locations or are in such a position as to cut the pathways immediately related thereto give rise to word-deafness—auditory aphasia, or word-blindness, visual aphasia. These are the types of so-called sensory aphasia.

The productive or effector side of the speech reflex arc, *i. e.*, the motor side wherein motor images more or less functionate, is, as has been pointed out, in and about Broca's convolution and the adjacent areas of the precentral and insula convolutions. Lesions here result in motor aphasia, or, as Broca called it aphemia.

The chief lesions causing these aphasias are hemorrhages, abscess, encephalitis, tumors, thromboses, emboli and acute edemas. Functional losses are seen resulting from the epileptic discharge, from certain hysterical dissociations, in uremia or in severe angiospasms, as in migraine for example.

Motor Aphasias.—In this type of aphasia the patient has lost the capacity to express himself in speech. He cannot read or talk spontaneously. He may say a few words, is usually able to say, yes, yes, no, no, is irritated over his loss, frequently saying "damn" or other expletive in the fruitless and exasperating search for words. Typical expletives are frequent and are often repeated over and over again. The older students distinguished two types of motor aphasia.

Subcortical or So-called Pure Motor Aphasia.—Here the motor speech pathways on the way out to the medullary nuclei are cut off. The patient is usually quite able to understand spoken and written symbols, but cannot speak spontaneously, read aloud, or even repeat what is spoken to him. He usually is able to write. Hysterical dissociation may bring about this type, as well as the concrete lesions already mentioned.

Cortical Motor Aphasia.—This type, less common and theoretically more difficult of comprehension, suffers a greater loss of internal speech. The patient is usually unable to write and usually fails to bring together long sentences or complicated word relations. He fails to grasp them in their entirety. The more severe the speech defect, the more pronounced, as a rule, is the agraphia.

Auditory Aphasias (*Word-deafness*).—The patient hears without difficulty but heretofore known symbols are now as though foreign. He is usually able to repeat the phrases, imitate the sounds, but they might as well be Chinese for all his comprehension of them or his ability to use them properly is concerned. Here also two trends in the symptoms are capable of fairly sharp separations.

Subcortical or Pure Auditory Aphasia.—The pathways between the receptors and their central station are blocked just distal to the auditory area. Spontaneous speech does not suffer but cannot be counted upon unless through visual correction. By the guidance of the written word the patient may express himself perfectly. Repetition of sound symbols may be impaired, hence these patients cannot repeat spoken phrases.

Cortical Word-deafness.—Spontaneous speech is much more involved and the patient's talk is usually quite disturbed whether he attempts spontaneous speech or when reading aloud. Internal speech is seriously disturbed. The patient cannot repeat, nor copy to dictation. Mistakes in orthography are frequent. While he may be fluent in his speech it may be fairly clear or a mixed-up jumble. Furthermore, the patient is not aware of his mistakes or only partially aware of them.

A great variety of partial forms are met with. One patient will lose the value of names, of nouns, of objects, others are merely confused (paraphasia).

Auditory aphasics usually clear up but in those patients who are rich in auditory forms of memory, in contrast to those whose memory type is more apt to be visual, the disability is usually greater.

Visual Aphasias (*Word-blindness*).—This type is also spoken of as *alexia* at times. The patient sees but does not take it in. Previously recognized signs are now as though Egyptian or cuneiform. Shapes are recognized and may even be copied but are not comprehended. They have lost their acquired context. Two types are here to be recognized also. Many modern students doubt the existence of this type.

Subcortical or Pure Word-blindness.—The area or center being intact the lesion involves the incoming pathways adjacent to the cortex. The patient fails to understand written words, cannot read aloud but may write or may copy from dictation. Partial blindness (hemianopsia) usually accompanies this disturbance. These patients are unable to read their own writing even though that writing may convey sensible meanings and be a correct medium of intercommunication.

Cortical Word-blindness.—Spontaneous writing, writing from dictation, or writing from copy are lost. The patient is agraphic. He has lost all memories of written or printed symbols, words, designs, or what not. Minor grades of the defect cause "paragraphia" in which the patient writes with mistakes in misplacing letters, or syllables or words. Such paragraphias are very frequent in paresis, for instance.

In actual practice the more or less sharply cut forms here summarily described are less often met with than the numerous mixed forms. A frequent type is a complete or global aphasia in which the entire brain speech mechanisms are wiped out. These are the forms seen following severe hemorrhage with the middle cerebral syndrome, hemiplegia, etc., accompanying.

Newer Orientations.—The chief recent advances made in the aphasia problem have been along psychological lines. Wernicke was the forerunner of this aspect. Freud a second. In more recent times Storch and Goldstein have advocated psychological viewpoints. Goldstein's contributions must not be neglected.[1] As yet they defy systematization for textbook purposes. Head's system is deserving of record founded as it is upon Hughling Jackson's principles of evolution and dissolution of function of the nervous system.

[1] Goldstein: Ueber Aphasie Schw. Arch. f. N. P., 1927, No. 6.

Head divides the aphasias into four forms: *Verbal, syntactical, nominal* and *semantic*.

Verbal aphasia, or verbal "*defects*" as Head first spoke of them,[1] shows in general the type of reaction of the older motor aphasia of Broca (Henschen). In severe forms the patient is reduced to no words—mutism—or to "yes" and "no." As speech returns the vocabulary increases but the enunciation is halting and slow. Recalled words for objects are used correctly, even if badly pronounced. Repetition efforts bring out imperfect articulation, but more words are utterable thus, than spontaneously. Words are evoked with difficulty and are mutilated in their structure. After shock has passed, verbal comprehension is rapidly restored. The patient chooses objects in oral or printed requests and can even carry out complex orders. Reading is possible but the memory of past words is impaired, hence, much rereading is necessary. Writing behaves as the speech. Spelling is difficult. Transcribing print into script is difficult. Numbers are grossly defective but change and money values are not. Orientation in space is not disordered. Playing games is not interfered with.

Syntactical aphasia is characterized by a more or less gross disorder of rhythm and syntax. The patient talks rapidly, his speech is jargon, and prepositions, conjunctions and articles tend to be omitted: polysyllabic words are slurred or badly pronounced. These defects are apparent whenever the patient attempts to talk spontaneously, to repeat what is said to him, or to read aloud. Internal speech is also disturbed in a similar manner, though to a less degree. The patient can write the names of common objects, and, in the less severe cases, can compose a short letter; but he is liable to fail in writing down the gist of something he has been told or has read to himself. He writes badly to dictation, but can copy printed matter perfectly in cursive script. Orientation is not affected. Arithmetical foundations are not seriously disturbed.

Nominal aphasia is more particularly characterized by want of power to discover appropriate names, or to find categorical terms in which to express a situation. Except in the acutest stages the patient possesses plenty of words, but he cannot apply them exactly and verbal form may suffer in his efforts to discover the correct name. Internal speech is gravely affected and there is usually difficulty in understanding and in executing oral or printed commands. The patient cannot comprehend what he reads to himself. Spontaneous writing is grossly affected, to dictation he writes something better and he can usually copy printed matter in cursive script, interspersed with irrelevant capitals. Number use and arithmetic are involved. Many calculations are impossible, though memory of amounts is not implicated. In orientation he tends to be puzzled. Card games are impossible, but checkers and even chess may be played. He cannot read musical notation but can sing without words and recognize correct scoring.

[1] Brain, 1923, **46**, 355.

Semantic aphasia defects are characterized by lack of recognition of the full significance of words and phrases apart from their immediate verbal meaning. The patient fails to comprehend the final aim or goal of an action initiated spontaneously or imposed upon him from without. He cannot formulate accurately either to himself or to others a general conception of what he has been told, has read to himself, or has seen in a picture, although he is able to enumerate most of the details. Such patients understand what is said, can read and can write, but the result tends to be inaccurate and confused. Counting is possible and the relative value of coins may be recognized; but arithmetical operations are affected and the patient is commonly confused by the monetary transactions of daily life. Drawing even from a model is impossible. Orientation is definitely disturbed. He is rendered useless save for the simplest occupations, and yet his memory and intelligence may remain in a comparatively high general level.

Henschen (loc. cit.) has subjected Head's cases to critical study. Nominal defects he states are found in all the aphasias hence this is no definite form. Verbal aphasia corresponds to Broca's aphemia. The lesion is in the left F_3, at times the C^a is involved. Syntactical aphasia agrees best with the temporal word deafness of the localists. The localization is the dorsal part of T^1. Nominal aphasia in 3 of Heads cases Henschen[1] calls typical amnesic aphasias. Word-blindness with agraphia occurred in others.

The semantic group Henschen states do not belong to the aphasia group at all.

In an address upon aphasia given on his eightieth birthday Henschen opens as follows:

"Mingazzini the celebrated brain student in Rome has designated the problem of aphasia as the most difficult of difficult themes. And as a matter of fact a critical examination of the evolution and present-day position of the study of aphasia leads to such a judgment. More than one hundred years have gone by since the aphasia research began. More than four thousand contributions upon aphasia have seen the light of day without there being found as yet any basis, or norm or generally adopted method of research. The more the material accumulated so much more indefinite and remote and pushed further into the future seems the goal of solution of the problem of aphasia. Then one reads further a masterly résumé of the whole question which with Head's historical chapter puts one *en rapport* with the whole question.

In a textbook the complicated problems here touched upon cannot be summarily disposed of. The student is advised that the situation is still in flux. Modern conceptions indicate that there is no actual loss of word images (engrammes), but that the patient cannot adapt himself to the situation in question. Emotional aspects of speech are

[1] Henschen, S. A.: Aphasiesysteme. This lecture given by Henschen on his eightieth birthday is one of the best résumé's extant. In Flechsig Festschrift, Monat. f. Psych. u. Neurol., 1927, **65**, 87.

more fundamental than intellectual aspects of the same. The patient loses the power of the higher purposeful act (H. Jackson). This is true in motor aphasia and in auditory (word deafness) aphasia. In the auditory type verbal paraphasias predominate. Paraphasia means making the use of one word instead of another one which is the more correct one. Paraphasia should not be mistaken for "confusion." Pötzl has said that when a confused patient had paraphasia he was aphasic and not confused. Patients with motor aphasia usually feel very depressed, and they also have considerable lack of impulse to do anything. The patients with word deafness, on the other hand, especially in the beginning, have no knowledge of their defects. They overlook them, feel quite healthy and have much more impulse to speak than well individuals. Such patients are always in rather good humor. Pure auditory aphasia is certainly not due to an anatomical subcortical lesion.[1]

Treatment of the Aphasias.—Certain forms clear up spontaneously. No matter what the form, however, immediate retraining should be begun, as soon as the patient has recovered from the shock of the original insult. This retraining should be persistently followed according to special methods for the various types which cannot even be outlined here.[2]

Writing Disturbances.—A great variety of disturbances in writing, quite analogous to speech disturbances, are known. Thus, writer's cramp is analogous to stuttering and stammering. It is probably psychogenic in origin. In the psychoses, very characteristic writing features and failures are present. The paretic may write just as as he speaks, slurring, leaving out syllables or words, etc. The katatonic may show stilted writing just as he shows a stilted affected speech. The haste of the manic is seen in writing as in speech. In time a true science of chirography may be built up on a psychical basis, just as a true science of phonetics has been.

Paralysis agitans, multiple sclerosis, epidemic encephalitis show analogous features in writing and in speech.

[1] Goldstein: Topik ker Grosshirnrinde Deut. z. f. n., vol. **77**, and Ueber Aphasie, Schw. Arch. f. Nerv. u. Psych., 1927, No. 6. Die Lokalisation in der Grosshirnrinde, Handb. d. norm. u. path. Physiologie. Vol. 10, Springer, 1927.

[2] See Forel, Presse Med., loc. cit., 1927.

CHAPTER V.

AFFECTIONS OF THE PERIPHERAL NERVES: SENSORY AND MOTOR.

NEURALGIAS.

Pain is the most striking symptom of disturbance of the function of the peripheral sensory nerves.

If pain alone be present one speaks of *neuralgia;* pain with trophic disorders, tender nerve trunks and altered muscular function when peripheral is usually termed *neuritis; radiculitis* if due to root or plexus disorder; whereas when peripheral pain is associated with peculiar skin eruptions and due to posterior ganglion involvement *herpes zoster* or *zona* is the name given to the affection. The conversion symbolic pains of hysteria, the central pains of thalamic lesions, the pains in schizophrenia and the causalgia syndromes due to involvement of vegetative fibers are not considered here.[1]

The boundaries between neuralgia and neuritis are purely relative. Thus a very mild neuritis may present only neuralgic pains; and a zona may be so slight as to cause no eruption. From a clinical standpoint separation of these processes may be impossible. It is not always necessary. Pathologically speaking, one locates the lesion of zoster in the sensory ganglion, yet tic douloureux—or trigeminal neuralgia—is preëminently a disease of the sensory ganglion, the Gasserian, and yet there is rarely any zoster eruption. One evokes the etiological factor of an acute infection element in herpes zoster yet there are zoster cases due to other than bacterial causes. The differentiation between a radiculitis and a neuritis is chiefly a question of localization.

Too much weight, therefore, is not to be laid upon the classifications given. For practical purposes these affections are treated under three heads, but their fluctuating separations should not be forgotten. It is misleading to call neuralgia a functional disorder.

Like many other conditions in nature, these affections, when seen in an accentuated and pure form, for practical purposes, represent different entities, yet the partial and intermediary forms are so many that the description of the clear-cut, classic types does not do justice to the whole subject.[2]

Definition.—Neuralgia may be defined as a painful affection of a nerve trunk or its branches, characterized by remittent or intermittent flashes of acute pain, with free intervals, not usually accompanied by trophic disturbances of the muscles, unless its severity limits the activities of an organ, occasionally associated with painful nerve trunks and with changes in the skin structures.

Neuralgias are but the expression of many diverse factors which may involve the body in general, as toxic (including endocrine) and

[1] For a discussion of the large problem of pain, consult Behan (Pain), or O. Foerster's splendid monograph (Die Leitungsbahnen des Schmerzgefühls, etc., Berlin, 1927).

[2] Harris: The Neuralgias, London, 1926.

infectious states, a nerve trunk itself, the sensory ganglia, contiguous structures, or they may be the reflex expression of a disorder in a viscus remote from the site of the pain. They may be of purely psychogenic origin, mostly hysterical conversions, occasionally delusional projections. *Neuralgia* therefore may be analyzed either as a symptom, a syndrome, a painful somatic reflex, or a psychological symbol.

Etiology.—An extraordinarily wide range of causative factors may determine mild or severe neuralgias in very diverse regions of the body. The most frequent causative factors are:

(*a*) Anemias due to hemorrhages, chlorosis, pernicious anemia, kidney disease, endocrinopathies, malaria, syphilis, intestinal parasites, etc.

(*b*) Toxins of exogenous origin, inorganic, and organic or purely endogenous toxins: thus poisoning by lead, mercury, arsenic and copper. Alcohol and tobacco are frequent causes. Morphinism causes neuralgia as an abstinence symptom. The toxins of many infectious disorders are especially prone to bring about neuralgias. Tonsillitis and malaria are examples. Typhoid fever, measles, gonorrhea, influenza, syphilis and streptococcic infections are frequently accompanied by neuralgias. The endogenous toxemias of diabetes and latent nephritis are further examples.

(*c*) Inflammation of the sensory ganglia, which may be either of infectious or non-infectious nature, gives rise to some of the severest forms, as seen in herpes zoster. These zosters occur from involvement of any ganglion, from the uppermost to those farthest caudad. They are usually dealt with in books on dermatology, but they are essentially nervous disorders. Ganglion involvements of non-infectious types give rise to neuralgias, such as tic douloureux, while tumors of the sensory ganglia may condition persistent and obstinate neuralgias in the affected sensory nerves.

(*d*) Involvements of the nerve trunks themselves, either by mild neuritic processes, perineuritis, pressure from anatomical structures, pressure from lesions, cuts, bullets, wounds, tears, tumors, periostitis, osteitis (often infectious in type), aneurism, exostoses, fractures, or displacements may cause severe neuralgic pains. If the nerves degenerate, neuritis results.

(*e*) Reflex or associated neuralgias are numerous and puzzling. Pulmonary, cardiac, gastric, hepatic, renal, ureteric, intestinal, vesical, uterine, ovarian, prostatic, testicular and affections of other viscera may give rise to herpetic eruptions, with painful, sensitive skin areas and neuralgias; in many instances the neuralgia is not accompanied by herpes. Head's[1] complete analysis of this class of cases is of paramount importance. Thus a persistent sciatica may be the reflex of a prostatic disturbance. An anemic woman may not suffer from pain, but on menstruation her referred neuralgic pains may become very severe over the tenth dorsal nerve, and pain and tenderness are frequent over the areas of the sixth dorsal (heart), seventh dorsal (stomach), and there may be occipital and midorbital neuralgia (Head). Dental neuralgias may be direct or of reflex origin.

[1] Brain, **16,** 1; **17,** 339; **19,** 153.

(*f*) Somatic disease of the nervous system. General paresis, tabes dorsalis, spinal tumors, cerebral or thalamic disease, syphilitic meningomyelitis, etc., are often accompanied by neuralgic pains.

(*g*) Metabolic Factors: The arthritic, gouty, anaphylactic and scrofulous may be said to be predisposed to neuralgic disturbances. Unknown factors thought to be related to atmospheric pressure, humidity, high electrical tension, etc., play a role in many of these cases. Here emotional factors may release vegetative or endocrinopathic imbalances. Endocrinopathies themselves may be causative.

(*h*) Chronic vascular disease, and especially arteriosclerosis, is a frequent cause, particularly in the aged, the senile and the presenile.

(*i*) Exposure to cold is an important factor. It is not certain that all neuralgias caused by cold are not really mild types of neuritis or perineuritis; discussion of the distinction is fruitless. The older writers found colds a predisposing cause in from 25 to 40 per cent. of the cases. In damp, cold countries this is particularly noticeable.

(*j*) Psychogenic Factors: These play an extremely large part in practical medicine in determining neuralgic pains. (Topalgias of the hysteric.)

Symptoms.—Pain is the main feature in neuralgia. For the most part it is the only expression of the nerve disturbance. The character of the pain varies considerably, but in general it may be described as unilateral and paroxysmal. It is characteristic of most neuralgias that they are primarily localized in the periphery. The pain seems to begin beneath the surface, and may then shoot out to the periphery. It may be described as biting, boring, tearing, darting, cutting, like an electrical shock, like a hot iron, etc., each patient having his own pet expression. It may come and go in lightning-like flashes, or throbbing pulsations, persisting for a shorter or longer time, then stopping for minutes, hours, or days, then recurring. When continuous, the pain varies considerably in its intensity. Nausea, vomiting, sweating and great prostration accompany many severe neuralgias.

The painful area usually conforms to the peripheral distribution of the sensory nerves. In the herpetic and referred neuralgias the root zone area is involved. Many neuralgias are but precursors of more serious central disease. Symbolic neuralgic pains rarely correspond to anatomical areas.

Certain tender *points* seem to be foci from which the pains start. These are usually situated along the nerve trunks, and pressure upon them is often sufficient to cause an exacerbation of a mild attack, or to provoke an attack in a period of intermission. Valleix attached considerable importance to these points. They are found, according to him: (1) at the point of emergence of the nerve trunks from bony foramina; (2) at such situations where a nerve trunk traverses a muscle to reach the skin; (3) at points where the nerve fiber breaks up into branches; (4) at points where the nerve becomes very superficial; (5) at Trousseau's apophysial points. Valleix's points are of diagnostic importance particularly in separating the neuritic from the neuralgic types.

Accompanying phenomena are frequent. In some patients a sense of apprehension may precede the coming on of an attack; vague sensations of discomfort often antedate the neuralgic outbreak. Ripples of pain, like pin-pricks, short twinges, etc., announce the advent of a more serious attack, or may be the sole evidence of an abortive one. Such mild phenomena are extremely frequent in certain of the so-called predisposed or neuralgic individuals; some feel that they cannot live at high altitudes; others fear rain, or an east wind; a thunder storm causes others to have twinges; while, again, certain dietary indiscretions make others complain of painful twinges for days. Just what conditions are at the basis of these features may be difficult to run down. They are none the less real.

Skin hypersensitiveness is frequent. It may precede or accompany an attack, and persist after the pain has ceased. Epicritic sensibility is mostly implicated. Light touch, a pin-prick or slight degrees of heat or cold are magnified. Deep pressure and extremes of heat and cold are usually palliative.

Anesthesia is not infrequent following an attack of pain, and the exact topographical distribution of the sensory modifications on the skin throw considerable light on the possible etiology of the neuralgic pains (Head).

Paresthesiæ are very frequent, and certain distributions seem to show them more than others. Thus, in the cutaneous branches of the femoral they are not infrequent. Here they take on the character of a "meralgia paresthetica."

Motor disturbances, either as cramp-like contractions or as paralyses, are not infrequent accompanying conditions. The painful contractions of tic

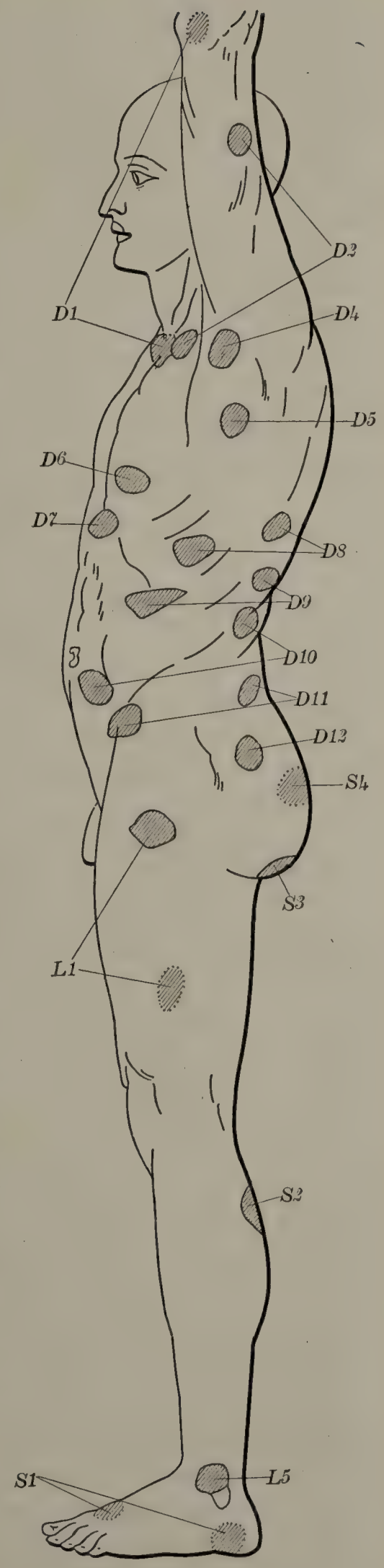


FIG. 215.—Cutaneous reflex zones of hyperalgesia, showing their relations with the spinal root segments and their vegetative nervous-system connections. The dotted areas are to be referred to the internal surfaces. (After Dejerine.)

douloureux and the oculomotor paresis of ophthalmoplegic migraine are familiar examples of this.

Vasomotor and secretory symptoms are frequent. The blood-vessels are frequently contracted in the early stages of a neuralgic attack, with resulting blanching and cooling of the skin. Following this a period of warmth, of redness, of free perspiration may result from the secondary dilatation of the vessels. In many cases of trigeminal neuralgia other secretions may be modified. Crying, coryza or salivation are not infrequent, while in widespread neuralgic attacks an increase in the amount of urine and of milk secreted is frequently found.

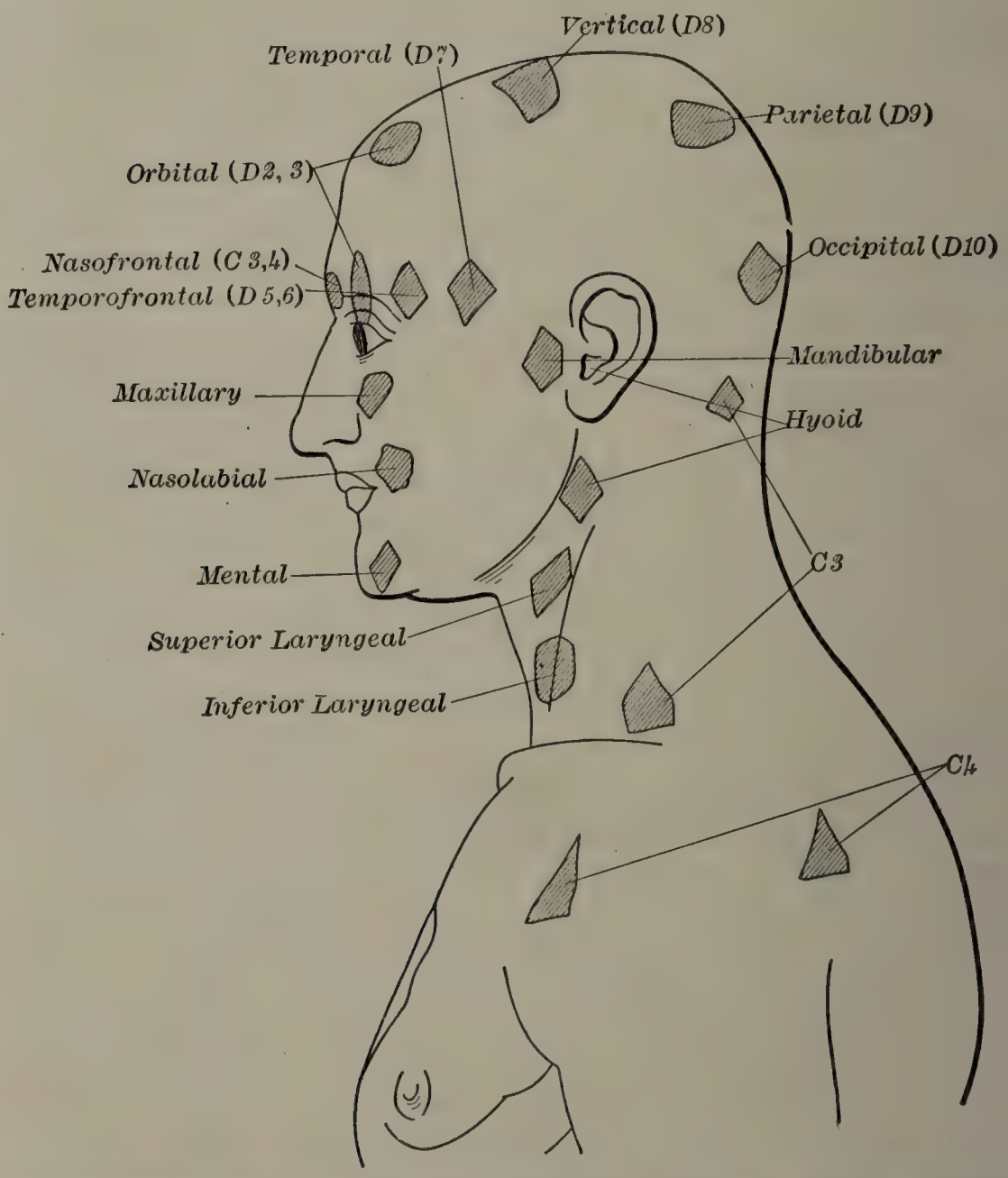


FIG. 216.—Cutaneous reflex zones of hyperalgesia of the head, neck and shoulders in their relations to vegetative nerve (somatic) disturbances. (After Dejerine.)

Premature graying of the hair, loss of hair, thickening of the skin, erythemata, eczema, pemphigus, herpes, thickening of the bones and occasionally, muscle atrophy are among the rarer trophic by-products.

During an attack, irregularities of the pulse are not unusual; slowing is the rule. The pupils are frequently dilated.

The general physical and psychical reactions are extremely important. Loss of sleep and anorexia cause the patient to lose strength

and flesh; and anxiety, irritability and petulance are almost inevitable. Mental depression, sufficient to lead the patient to make suicidal attempts, is not infrequent, especially in severe cases of trigeminal and sciatica neuralgia. The contracting of a drug habit is not unusual.

Course.—This depends naturally upon the underlying conditions. Many are acute and transitory, persist three or four days and never reappear. Such are the herpetic types. Many reflex neuralgias run an acute recoverable course, but show a marked tendency to recurrence. The neuralgias which accompany the chronic cachexias of nephritis, carcinoma, brain or spinal-cord disease, usually progress in a markedly chronic manner. In those hereditarily disposed individuals the tendency to chronicity with longer and shorter periods is proverbial.

Earlier French writers attempted to distinguish benign and severe forms. Most neuralgias in which the causative factor is undiscoverable (the so-called idiopathic or primary neuralgias) run a benign course, while the neuritic types are less amenable to treatment.

The subdivision of neuralgic neuroses, subacute neuritic neuralgia, and chronic neuritic neuralgia offer a grouping referable to a course which has only clinical convenience to warrant it.

In the first type one finds the disorder more or less limited to the neuropath. The attacks come without appreciable cause, or follow a nervous shock. Exposure to cold, or dietary indiscretions are frequently claimed as causes, but are not. The pain comes on with great suddenness and usually goes without great violence; it comes and goes apparently without rhyme or reason, and is not accompanied by painful nerve trunk nor trophic disturbances. It recovers at times, to recur at intervals of a year or years.

In the subacute, neuritic neuralgic type, exposure to cold or pressure, especially in arthritic patients, determines an attack. The attack develops gradually; the pain, at first mild and intermittent, gets worse and worse and more continuous. Finally, after a day or more, the paroxysms become extreme, the intervals being marked by dull pain; hypersensitive Valleix's points are characteristic findings.

When a mixed nerve is involved, muscular atrophy or other trophic signs appear, signalizing the occurrence of a neuritic process. Local edema and herpes zoster are frequent accompaniments. This type usually commences to recover in from two to three weeks, and an ultimate recovery is to be expected. Recurrences occur, however, and a leap to the third type of chronic neuritic neuralgia is made. This form is frequent in the aged. The history is usually that of several subacute attacks with increasing tendency to chronicity. Here the trophic disturbances in muscle and in skin are more marked. The paroxysms run a remittent course.

Diagnosis.—Enough has been said to emphasize the need for a searching analysis of the causative factors of every neuralgia. They are many, and presumably the most widespread diagnostic error is the overlooking of an early tabes dorsalis in young to middle-aged adults.

Children are not prone to neuralgias—in the narrower sense—and

a neuralgic affection in childhood calls for close scrutiny. It is usually somatic, but may be psychogenic.

Since unilateral pain of special localized type, occurring in irregular attacks, is almost the sole criterion of neuralgia, it is very frequent that organic disease of a viscus will show precisely similar accompanying features. In the majority of cases the underlying somatic lesion may be detected—occasionally it remains difficult to locate. Not infrequently the diagnosis of a persistent neuralgia may be cleared up by the finding of malarial organisms in the blood, or more rarely the presence of a marked eosinophilia will call attention to trichina as the cause of an obstinate neuralgia; or the eggs of an intestinal parasite in the feces (uncinaria) may direct attention to an anemia which underlies a severe neuralgia. Syphilitic neuralgias, either toxic or vascular, are by no means infrequent.

The diagnosis of myalgia from true neuralgia is not often difficult, but occasionally, especially in the intercostal and lumbar regions, the diagnosis becomes uncertain. These neuralgic-like myalgias are usually isolated in their location, are not, as a rule, accompanied by acute exacerbations, nor are the regions usually painful on pressure. Motion, on the contrary, usually aggravates myalgias.

Neuritis of a mild grade offers an especially difficult problem. As already stated, mild neuritis shows itself as a neuralgia. The question to be solved concerns the likelihood of a more severe degree of neuritis. In this case the usual signs of neuritis are painful, swollen nerve trunks, trophic disturbances, more continuous pain, Lasègue's phenomenon, weak, flabby muscle fibers and electrical changes. New growths pressing upon or involving the nerve trunks within or without the spinal canal, in the early stages particularly, begin as pure neuralgic syndromes. Minute analysis of the sensory phenomena will usually clear up the diagnosis early, although at times it may be impossible in the earliest stages.

A neuralgic affection may be one of the earliest signs of a multiple sclerosis. Oppenheim has found a severe tic douloureux to have been the earliest sign of this disorder. Syringomyelia may begin as a localized neuralgia. Minute hemorrhagic lesions of the spinal cord of traumatic origin give rise to neuralgias.

In the diagnosis of hysterical neuralgia, great caution should be exercised. Hysterical neuralgias partaking of the nature of a pseudoneuralgia are extremely diffuse, and react very rapidly and markedly to suggestive influences. Hysterical neuralgias are almost invariably accompanied by other conversion signs. (See chapters on Psychoneuroses.)

Neurasthenic pains need to be differentiated, if not almost entirely ruled out, as a common diagnostic pitfall. The many mixed forms of neurasthenic, hypochondriacal and hysterical neuralgic pains should bear their characteristic sidelights. The diagnosis of these types of neuralgia should not be lightly made, for it is not to be forgotten that these syndromes of themselves may be the reaction on the part of the

nervous system to some more fundamental somatic lesion. Thus patients suffering from severe so-called neurasthenia with cachexia, and severe intercostal neuralgic pains may have an undiscovered carcinoma of the stomach, mediastinum, etc.

In *tabes dorsalis* the neuralgic pains have a wide range, are rarely localized in a peripheral nerve distribution and are apt to be radicular in their distribution. Pain on pressure of the nerve trunk is usually absent. The objective findings in the pupils and cerebrospinal fluid establishes the diagnosis.

The pains, cramps and muscular weakness of *intermittent claudication* sometimes give rise to severe neuralgias. Aortic aneurism gives rise to reflex neuralgic pains, which are usually very severe, burning or boring in character. Aneurisms in other regions are to be carefully excluded.

In reflex neuralgias the use of cocain or other local anesthetic may determine, by exclusion, the site of the original lesion. An orthoform suppository pressed well against the prostate has been known to relieve a severe sciatic neuralgia. Tumors of the pelvis frequently give rise to sciatic and crural neuralgias, and persistent neuralgic pains of the knee are often a reflex from hip-joint disorder.

Neuralgic pains are a frequent indication of disturbed nerve fiber metabolism, with a hyper- or a hypothyroid affection behind it.

Periostitis and osteitis, often resulting from members of the less virulent streptococcus groups, M. viridans, etc., are not infrequent causes for neuralgic pains which are often localized.

For the precise localization of the areas involved consult Figs. 27, 28, 29, 30, 31 and 32, where both the peripheral and radicular nerve distribution are figured.

Prognosis.—This is conditioned by the pathological process that is responsible. The more chronic of the neuralgias, which in years gone by tended to bring about chronic invalidism or inveterate drug habits, have ceased to have such a sinister import by means of a better understanding of the underlying conditions, and by a much more resourceful therapy. The younger and stronger the individual, and the less the tendency to hereditary disposition, the better the prognosis in those neuralgias which apparently are idiopathic, as well as those due to alcohol, lead, or other toxic agent. In the more chronic forms which are not due to removable condition the prognosis is bad. With increasing insight, however, into the many intricate disturbances of nerve metabolism many of the intractable forms may be conquered.

Treatment.—The chief indications are to quiet the pain and ascertain the cause. A painstaking study of the history and an exhaustive physical examination are necessary in all cases. The therapy will therefore vary widely if the cause be ascertained; quinine will cure one patient, a surgical operation may be called for in another. General rules, therefore, are largely illusory. One should never treat a *neuralgia per se,* as it is a general result of varying causation.

Taking up the general therapeutic indications, the analgesics which

have proved useful may be discussed first. Phenacetin, acetanilid, antipyrin, aspirin, pyramidon, lactophenin and phenocoll are among those that have been valuable. New ones are constantly being added, and among them some are certain to be of value.[1] The salicylic acid group combinations are at times useful, especially in the milder cases and in patients with arthritic tendencies. In influenza and tonsillitis neuralgias the salicylates are valuable. Combinations of these with soporifics, such as chloral, paraldehyde, sulphonal, trional or veronal, are helpful in procuring sleep, and thus prevent the reduction of the patient's resistance.

If any of the opium group be necessary it is better to give such in sufficient doses. Usually smaller doses may be given when combined with the analgesics mentioned. Aspirin, gr. vij (0.5 gram), codeine, gr. $\frac{1}{3}$ (0.02 gram), and trional, gr. vij (0.5 gram), for instance, is a useful combination to be taken at night. Other combinations are equally effective. For the chronic neuralgic pains morphin is to be avoided as long as possible. This does not apply to a very old patient, or one in whom the neuralgia is simply the expression of some chronic incurable disorder—carcinoma for example. The gradually acquired immunity, with the need for larger doses, and the pernicious effects of a habit apply to all the members of the opium group.

All analgesics should be used only provisionally. They give the patient ease while looking for the real cause of the pains.

If one's inquiry is satisfied by the relief of pain the use of analgesics alone is bad therapy. Other drugs are quinine, which in combination with the salicylates is specially valuable; arsenic which is serviceable in the neuralgias due to anemia, especially in combination with iron. Atropin and aconitin were used widely before the days of the antipyretic analgesics. Their definitely poisonous qualities have driven them into the background. The unreliability of cannabis indica has done the same for this otherwise useful analgesic. The iodides are called for in the syphilitic neuralgias and are useful in many neuritic neuralgias.

Counter-irritation is of great service in many cases of severe neuralgia, especially after the acute onset is over. The Paquelin cautery is the best means; mustard paste, cantharides, turpentine, chloroform, ether and acupuncture all have their place. Local freezing may be carried out by ethyl chloride, methyl chloride, ether or other volatile substances. Menthol, or other similar derivatives may be used to advantage for mild neuralgic pains.

Direct applications of local analgesics, either to the nerve trunk or within the spinal canal, are valuable in many deep-seated neuralgias, especially of medullary origin. Cocaine, tropococaine, eucaine are all useful given by the Corning or Quincke method.

Local applications of heat are grateful and valuable. Hot-water bags, hot sand, electrical pads, etc., may be utilized. General or local hot-water baths or hot-air baths (baking) are at times desirable.

[1] Consult S. Fraenkel (Die Arzneimittelsynthese, 6th ed., 1927) for a full consideration of the newer synthetic narcotics.

General hygienic treatment is imperative. A generous diet, full sleep, healthful occupation and freedom from mental worry are essential. Cod-liver oil, nitrogenous diet, with iron, arsenic, strychnin, calcium salts, are indicated. Faddy dietaries should be avoided. Even in arthritic neuralgias it is doubtful if meat does any particular harm when not taken to excess. Alcoholic beverages are to be restricted.

An alkaline therapy often helps many fugacious, persistent neuralgic pains. Fruits containing the citrates seem to give relief.

Preparations of the internal secretions, particularly thyroid and pituitary clear up some intractable neuralgias of unknown origin. They may be given in doses of from $\frac{1}{10}$ to $\frac{1}{2}$ grains twice or thrice daily.

Climatic changes are rarely advisable. Low-lying, damp and humid atmospheric conditions seem least desirable for certain patients. The general stimulus that comes from a dryer, higher atmosphere, even if colder, works to the general advantage, even if not directly valuable for the relief of pain.

Electrotherapy when well managed and properly selected is of great value in some neuralgias. It cannot be said that it is clearly recognized just what forms of current are best utilized in what types of neuralgia, hence most efforts must follow the method of trial and error. In general, however, Leduc's modifications of d'Arsonval's rapidly interrupted current offer the readiest and most widely applicable form of electrical current for the relief of neuralgic pain. It is doubtful if any other form of electrical application is known at the present time that is as valuable as this. It is, in fact, a type of electrical anesthesia, solely palliative, but very grateful. Newer applications are being brought out, and other forms may replace the Leduc currents, but at present these seem to give the most reliable results.

Faradic currents, as heretofore employed, act for the most part simply as counter-irritants, and seem to possess little superiority over the actual cautery. Galvanism with mild currents is useful for many topalgias.

Psychotherapy is the only rational treatment for the psychogenic neuralgias. These make up at least 25 per cent. of the neuralgias.

Surgical intervention is called for in all cases in which pressure is demonstrable and the cause removable. Tumors and new growths, involving or pressing upon nerve structures, if removable should be taken away. Surgical interference may be of radical service in many of the reflex neuralgias of obscure origin, probably related to visceral ptoses. Such surgical interference is justifiable only in chronic cases where these visceral ptoses have resulted from long-continued psychical causes. Psychotherapy is preferable in the early stages. Nerve stretching needs mention mostly to be condemned.

SPECIAL LOCALIZED FORMS OF NEURALGIAS.

While any sensory nerve in the body may become painful, there are certain regions which show a greater tendency to involvement

than others. Bernhardt has collected the statistics of localized distribution in some 685 cases with the following results: sciatic, 303; trigeminal, 124; brachial, 108; intercostal, 45; occipital, 42; crural, 25; obturator, 2; lumbo-abdominal, 12; anterior femoral, 11; tarsalgia, 4; metatarsalgia, 4; Achilles, 3; and coccygeal, 2. In 616 cases collected by one of us (J.) during four years (1902 to 1906) the distribution was as follows: trigeminal, 315; sciatic, 194; brachial, 31; occipital, 28; intercostal, 19; lumbo-abdominal, 19; peroneal, 2; crural, 2; ulnar, 1; coccygeal, 1; and plantar, 1.[1]

Trigeminal Neuralgia.—Simple neuralgias of the branches of the fifth nerve are among the commonest of all the neuralgias. Fothergill's studies on *A Painful Affection of the Face*, published in 1773, is a classic. The inferior and superior branches preponderate in frequency of involvement. Most frequently these neuralgic pains are due to some affection of one of the branches. Inflamed teeth play a predominant role. Affections of the ears; the eyes, iritis, cyclitis, iridocyclitis; the skin of the face or head; inflammation within the accessory sinuses of the nose, forehead, antrum, mastoid, all of these may produce diffuse neuralgic pains, at times clearly separable from a neuritic neuralgia of the fifth, at other times not.[2]

Cold and wet are important agents in facial neuralgia. In certain countries, notably England and the north of Germany, trigeminal neuralgias from this cause are extremely common; they seem to be much less frequent in the United States, and notably so in southern countries.

Neuralgia of the superior branch is seen more commonly by physicians, although the dental branches are involved much more frequently. These patients go to dentists and therefore rarely enter into medical statistics. This is a reason why it is incorrectly stated by most writers that the superior branches of the fifth are most often involved. For the most part the milder types of neuralgia are induced by irritation of some of the terminal filaments, while in the neuritic form, tic douloureux, which is the more classic, a lesion of the Gasserian ganglion is usually present. Mild cases of tic douloureux may be indistinguishable clinically from other types of neuralgic pain.

Tic Douloureux.—Enough has been said on neuralgia in general to indicate the character of the simple forms of fifth nerve neuralgia. One type, however, by reason of its severity and its fairly definite pathological anatomy, needs more extended consideration. Avicenna knew tic douloureux and described it with great accuracy. It would be desirable to restrict the term tic douloureux to a definite and, if possible, limited type of neuritis of the fifth nerve, particularly to the form due to changes of a chronic degenerative nature occurring in the Gasserian ganglion. This is not yet possible, and clinically the neuritic, and peripheral neuralgic cases are either not at all separable from the ganglion cases, or with considerable difficulty.

[1] See Reports of Vanderbilt Clinic, Jour. Nerv. and Ment. Dis., 1902, *et seq.*

[2] See recent monograph of Sluder on the Sinus Pains, St. Louis, 1927.

Tic douloureux usually affects one side of the face. In the majority of cases some selection occurs among the branches, one or two being involved, rarely all three; the ophthalmic branch the oftenest, the inferior maxillary the least often implicated.

The more classical tic douloureux neuralgias are characterized by the extreme severity of the pain, usually preceded by paresthetic phenomena, and frequently accompanied by sympathetic or irradiating pains in other branches than the one chiefly involved, or in other nerves. The pain may be paroxysmal or continuous, with marked exacerbations. Patients compare them to the piercing pains of a sharp knife or the burning of a red-hot wire. The patient remains for a shorter or longer period, a few minutes to several hours, under the grip of the pain, unable to move a muscle of the face or fearful of stirring, lest a spasm more fearful than the others should occur; even the air-pressure of a suddenly closed door may bring on an exacerbation. The longer attacks are rarely as vicious as the shorter ones.

Hypersensitive Valleix's points are relatively constant. In ophthalmic involvement the sore points are found above the supraorbital notch, at the external angle of the upper lid, the upper, outer aspect of the nose and the globe of the eye; in the superior maxillary branch the inferior orbital notch is the chief point of pain; the malar bone, and opposite the last upper molar are other less frequently found points, while the outer angle of the mouth, and the roof of the mouth are rarely their site. In the inferior maxillary distribution the points are chiefly just in front of the auditory canal, the side of the tongue, the border of the chin, and Trousseau's points over the first and second cervical vertebræ.

Vasomotor and secretory disturbances are usual. The skin is, as a rule, hot and swollen, occasionally pale and frigid; tears, nasal secretions, and saliva flow in abundance. The eyelids may be swollen, the conjunctiva reddened to the point of ulceration at times; within the nose and mouth extravasations occur, and ulcers are not uncommon. Herpetic attacks are also not infrequent, and in some of the attacks grave injury to the eye structures may take place. Glaucoma is one of the severe complications. Other trophic disturbances are skin eruptions, acne, erysipelatous reddening, graying of hair, and blackening of the tongue. In long-continued cases hemiatrophy may occur. Changes in the sense of taste, of touch, of hearing, are at times present. Photophobia is frequent, while diminution in the visual fields and accommodation cramps have been noted.

Severe mental disturbance, amounting at times to hallucinatory confusion may be present. Suicidal attempts are to be guarded against in these excruciating cases.

The motor disturbances consist of convulsive movements of the facial muscles (convulsive tics, spasmodic epileptiform neuralgia, Trousseau), sudden forced closing of the eyelids, drawing of the mouth to one side, or sudden turning of the head. At times the convulsive movements extend to the arms. Paralytic phenomena in the third

nerve have been noted. The general psychical disturbances noted are prone to occur in this type.

Course.—In the majority of cases the attacks appear in series and attain a periodicity which comes to be dreaded by the sufferer. The free intervals usually become shorter and shorter; but many patients may have only one attack a year, especially in cold weather, or even at longer intervals. A single attack may last a few days, or in the severe forms several weeks, the patient not being free from pain day or night, save under the influence of morphine. Some patients have a few attacks in a lifetime, others are not free from the disease for years. The severer convulsive forms are prone to occur late in life.

Diagnosis.—Ordinarily the classical form of tic douloureux is recognized without difficulty. Patients have all their teeth extracted, however, under the mistaken diagnosis of a dental disease, while some intractable trigeminal neuralgias have been cured by proper attention to diseased teeth. Aneurism of the carotid, tumors pressing upon the nerve or upon the Gasserian ganglion, may be difficult to determine as the exciting cause. These, however, are usually accompanied by accessory symptoms, palsies, eye-ground changes, aneurismal murmurs, pain within the head, cerebellar syndromes, ear pains, etc. The otalgias (tympanic neuralgias) usually considered in this connection are probably due to cephalic ganglion disorder, although frequently referred to by some authors as neuralgias of the seventh nerve.[1]

Multiple sclerosis has startedas a trigeminal neuralgia.

Treatment.—It is as essential to endeavor to find and treat the cause for a facial neuralgia as for neuralgia in general. The various remedies given under the heading of neuralgia may be tried, and as malarial neuralgias are very frequently trigeminal, energetic quinine therapy may be given; the absence of blood findings is not contraindicative, especially in malarial countries. Gelsemium, the tincture in 10-minim doses, gradually ascending, aconite in doses of $\frac{1}{100}$ grain, cannabis indica (fresh), in doses of from $\frac{1}{4}$ to $\frac{1}{2}$ grain, are reputed as especially valuable in the facial cases. Any of the analgesic antipyretics may suit individual cases, and avoid the use of morphine, which alone is reliable in many severe cases. Local applications of cocaine to the conjunctiva, nasal mucous membranes, buccal surfaces are sufficient to repress some mild attacks. Cathartics of the magnesium sulphate group are often of signal service, combined, at times, with aconite to the point of causing peripheral tingling.

Injection methods have been tried for years. Pitres and Vaillard, in 1887, and Schlösser,[2] in 1900, took up a series of experiments with alcohol, and the latter has perfected a method of injecting alcohol within the substance of the Gasserian ganglion, which has given excellent results. The chief features in the deep alcohol injection method is the introduction of a long, dull, cutting needle into the foramen

[1] Kidd: Rev. of Neur. and Psych., 1913. Mills: Jour. Nerv. and Ment. Dis., 1911.
[2] München. med. Wchnschr., April 30, 1897.

ovale and there injecting *in situ* the branches of the trigeminus. Special methods have been devised. Narcosis is not necessary; 80 per cent. alcohol is used. In three or four hours following the injection the pain is relieved, and two or three more injections are given within a week to complete the treatment. Immediately following the injections, which should be done only after extended practice on the cadaver, there is a marked anesthesia on one side of the anterior part of the head, including the nostril, palate, and one-half of the tongue; a slight paralysis of the muscles of mastication, which may persist for some time, but usually disappears in a few hours; a degenerative process is set up in the nerve trunk, which is recoverable, and general sensibility usually returns, but the pain returns. Relief extending over a year in a number of cases is reported by numerous observers. Our own experience is distinctly against this procedure. We consider it a barbaric method and prefer the surgical procedures.

Three surgical procedures have been seriously advocated. The first and earliest consisted of peripheral section, first said to have been done by J. C. Warren of Boston. Section of the fifth may be employed to advantage in those cases in which the disease is undoubtedly peripheral. As modified by more recent procedures, the older objection that regeneration takes place is partly done away with.

Rose, MacEwen, Horsley, Hartley and Krause perfected the operation of excision of the ganglion, and the modified Hartley-Krause operation by the temporal route has been largely the method of hoice. Cushing's more recent modifications are of lasting value. The operation still remains one of much difficulty and seriousness.

Van Gehuchten, in 1903, suggested tearing. Spiller, in 1898, had suggested the surgical expedient of cutting the sensory root, which he claims is safer than the operation of tearing, a procedure tried in 1881. The method of division of the sensory root, as reported by Frazier and Spiller, and more recently Stookey's[1] and Frazier's methods of fascicular differentiation promise the most valuable results. Foester's experience is against the sympathetic (Jaboulay *et al.*) and arterial sympathectomy (Leriches *et al.*) procedures.[2]

Psychogenic cases, not infrequently, are curable by psychoanalysis.

Cervico-occipital Neuralgia.—This occurs in the distribution of the sensory nerves of the cervical plexus, consisting chiefly of the occipitalis major, the occipitalis minor, auricularis magnus, cervicalis superior, supraclavicularis and phrenic. Neuralgia in this general region seems to be rare. In Remak's summary of 15,000 cases only 50 were in the cervico-occipital regions. Valleix has given one of the most complete monographs on neuralgia in this area and little has been added to his description, save in the finding of divers etiological factors.

[1] Stookey: Jour. Nerv. and Ment. Dis. Trans., New York Neur. Soc., November, 1927.

[2] Foerster, O.: Loc. cit.

Etiology.—The several causes of neuralgia are operative here and need not be repeated. Special determining features seem to be the carrying of heavy weights on the shoulders (a more frequent cause for brachial neuralgias), arthritis deformans of the upper cervical vertebræ, caries, syphilis, tuberculosis, tumors, cervical pachymeningitis, falls and blows wrenching the cervical vertebræ, enlargement of the cervical lymphatics and aneurisms of the vertebral artery. Oppenheim refers to the great frequency of hysterical neuralgia in this region and psychogenic neuralgias of the back of the neck and occiput are extremely common. They are often found in individuals who are either under great strain or those who are constantly forcing themselves.

The pains occupy the regions mentioned, being particularly localized in the neck, below the occiput, and running up to the vertex, occasionally behind the ears. The Valleix points found most frequently are the occipital point between the mastoid apophysis and the first cervical vertebra; points between the sternocleidomastoid and trapezius (cervical); the anterior border of the mastoid and the middle of the ear are of less frequent occurrence.

The pain is frequently bilateral. Dull pain on pressure, with tender skin, is usual as a paroxysmal occurrence. This tends to make the sufferer hold his head in a stiff position, which in time may cause a characteristic attitude. This tenderness may be so acute that ruffling of the hair will start a paroxysm. Graying of the hair, loss of hair, with other trophic signs may be present. Sudden pulling back of the head, or other muscular involvement, is an occasional symptom.

Diaphragmatic Neuralgia.—This form of neuralgia, also known as phrenic neuralgia, is of rare occurrence. Falot and Peter have written upon it. The pain is usually present near the free border of the ribs, occasionally as high as the chin and in the neck, beneath the clavicle, and in the scalenus anticus muscle. Trousseau's points are located over the second to the fifth cervical vertebra. The pain frequently runs down the arm, especially in certain complex cases of mixed brachial neuralgia.

Breathing may be seriously interfered with, the breath coming fast and short; longer excursions of the diaphragm are impossible. It is a common experience to have a short, sharp stitch in the side, with inability to breathe for fear of pain. This is the type of distress encountered in phrenic neuralgia. In the majority of cases the pain is in the left side.

Anemia, affections of the mediastinum, heart and pericardium and aneurism of the aorta are the most frequent attending features. An intractable phrenic neuralgia may complicate an exophthalmic goiter, or be present in carcinoma of the neck region.

Idiopathic or pure phrenic neuralgias seem to be unusual, whereas temporary or more permanent types are seen as symptoms of the affections named. In the latter case the prognosis depends on the initial difficulty.

Brachial Neuralgia.—In this general form the components of the brachial plexus, from the four lower cervical, or some of its filaments, and first dorsal roots, are those involved. The chief nerves carrying sensations from the skin area of the arms and shoulders are the circumflex, radial, internal cutaneous and musculocutaneous. These enter, for the most part, the upper and middle cords of the plexus. In the majority of cases the pains of brachial neuralgia are located in the upper arm and about the shoulder, *i. e.*, in the area of the circumflex, radial, musculocutaneous and internal cutaneous nerves.

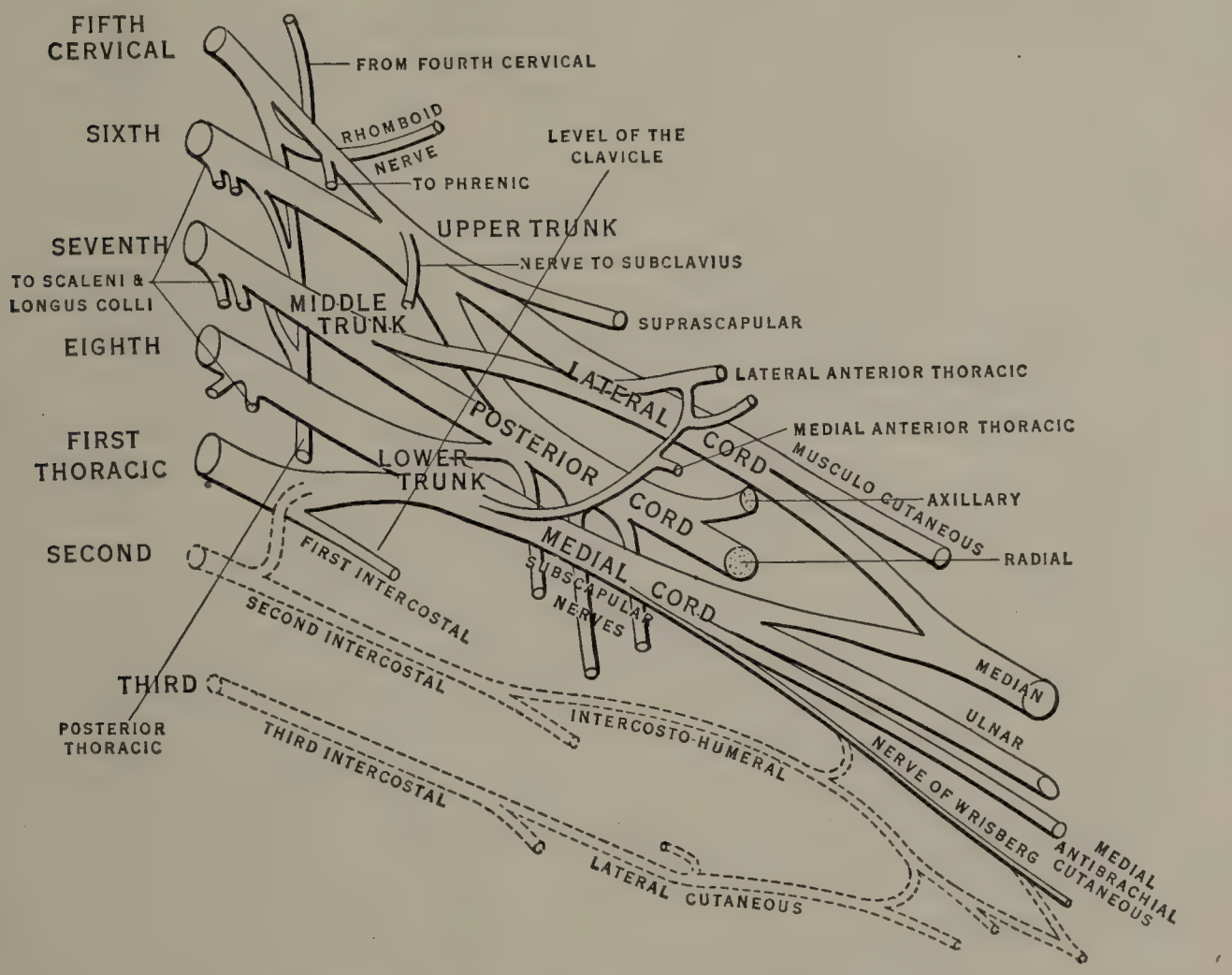


FIG. 217.—Plan of the brachial plexus. (Gerrish.)

Bernhardt's statistics show that men are more frequently affected than women, but the reverse is shown in the figures of other observers (Romberg, Erb). More women have brachial neuralgia than men and in most instances it seems that excessive sweeping is the attributed cause. In piano-players, neuralgias in this area are frequent. Perhaps these should be relegated to the occupation neuroses with the pains of hair-dressing, skirt-carrying, telegraphy, writing, etc. At any rate, arm and shoulder pains are frequent, in their mild grades at least, and very variable. (See Fig. 224 and Figs. 23, 24, 25, 26, 30 and 31.)

The usual causative factors come into play here. The neuropathic constitution is put in the foreground by Oppenheim; Bernhardt lays considerable stress upon the importance of bone injuries with callus formation in the causation of many arm neuralgias. Small punctured wounds about the forearm, wrist and arm are responsible for many

symptomatic neuralgias, as Weir Mitchell has so well shown. More remote causes are found in vertebral disease, tumor formation, aneurisms, syringomyelia, multiple sclerosis and tabes. The frank neuritic processes in their beginnings must be borne in mind, and cervical rib should not be overlooked.

Symptoms.—Cervico-brachial neuralgias are extremely variable in distribution, extent and severity. The onset is usually sudden, especially in those patients in whom an antecedent history of exposure to cold and to wet is obtainable (motormen, policemen, etc.); at times the beginning is preceded by twinges and slight distress. On awaken-

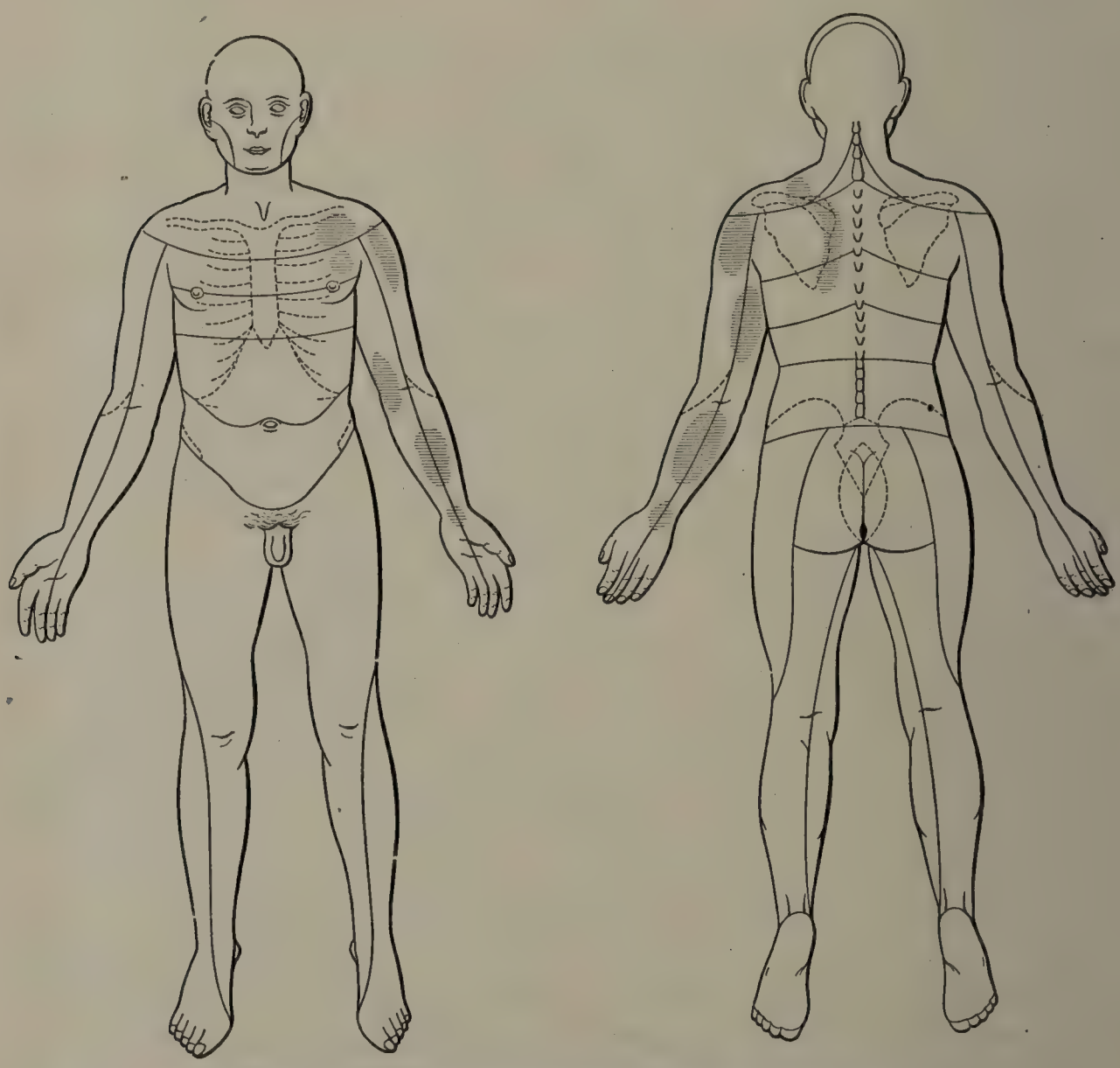


FIG. 218.—Painful points in brachial neuralgia.

ing in the morning sharp pain is felt in the shoulder and arm. The pains in brachial neuralgia are less apt to be the sharp, shooting variety so dreaded in tic douloureux, but sudden accession of sharp pains, ivaryng in their intensity, are frequent. As with most neuralgic pains, movement increases them. Toward evening the pains are apt to increase and the patient, although obtaining relief by lying down rarely sleeps well. Soreness of the skin, slight swelling and general reduction in tone are the usual accompaniments. With increasing disuse slight atrophy is common, and swelling is usual. The tendon reflexes are frequently more irritable and active. More atrophy, paresis

with vasomotor-trophic symptoms and altered tendon reflexes indicate a definite neuritic process. Herpetic eruptions occur with non-infectious as well as with infectious involvements of the sensory ganglia.

Tender points are very variable. They are most frequently in the middle of the back; about the level of the second or third dorsal there is usually a sore Trousseau point. Gowers notes that the inferior ulnar point in front of the wrist is the commonest sore point. Babinski has called particular attention to a radial neuralgia due to a mild or severe neuritis of the radial. The pains occupy the posterior portion of the arm and are unusually severe. Neuritic changes are not infrequent. The chief causes seem to be exposure to cold and disturbances of the reciprocal action of the ovaries or testicles and thyroids and other endocrinous glands. The menopause is a frequent period of onset.

Diagnosis.—In the diagnosis particular care is needed in excluding affections of the spinal cord, meninges, and vertebræ, as well as angina pectoris and pseudoangina. Disease of the joints and bones should be excluded at the outset, although it may be very difficult in some cases of periostitis. In tumors and other somatic affections of the cord the painful points are usually absent, but the earliest and only symptom of spinal-cord tumor, intramedullary or extramedullary, may be a brachial neuralgia. In tabes the pains are apt to be bilateral. The exhaustion neuralgias are also apt to be bilateral. The general indefinite features of a myalgia, plus the muscular, rather than the nerve soreness, are usually sufficient to exclude it.

The occupation neuroses involving the arm and shoulder are many. The history of protracted exercise of certain groups of muscles is usually sufficient to identify the proper cause for the neuralgic pains. Occupation neuralgias, like neuritic neuralgias, are neuralgias none the less, the sole diagnostic question arising as to the cause, and through this the proper mode of therapeutic attack and the probable outcome. Alcoholic neuritis in its mild grade offers particular embarrassments. Lead-poisoning neuralgias are to be borne especially in mind, while diabetes is of prime importance. Brachial psychalgia is a possibility, but the diagnosis must be made with extreme caution after a rigid exclusion particularly of organic factors. Psychogenic and neurasthenic neuralgias occur in this distribution as well.

Treatment.—Rest is a necessity, and is primarily insured by means of a sling. The diagnosis of a cause being assured, treatment should be begun to remove it, either by medical or surgical means. Nerve sutures for injury has come to occupy an important place, and is usually attended with good results, even after long periods of loss of function. In the early stages active mechanical treatment is to be avoided. Hot applications are useful in most acute neuralgias. In the later phases massage, particularly the Nägeli movements, are valuable. Galvanism, 3 to 6 milliampères, is well adapted to these neuralgias, but usually much better results are obtained by the Leduc rapidly alternating currents. Salicylates (especially in analgesic combina-

tions), iodides, quinine, arsenic and large doses of strychnine are of value at times. The internal secretions are curative for some. Psychoanalysis is to be used in psychogenic cases.

Intercostal Neuralgia.—The twelve dorsal nerves constitute the plexus involved, although the upper series, especially of the left side, are most frequently concerned. Bernhardt says that the site of election is mostly from the fifth to the ninth. Since the dorsal nerves divide into internal and external branches the site of the neuralgia may be on the surface or within (pleurodynia, etc.). The two upper nerves send branches to the internal surface of the arm, and pain is occasionally felt there. The abdominal involvements are rarer and may extend down to the genitals. (See Figs. 27 to 32.)

Women more often show this form of neuralgia than men, and the disorder is much more common in cold weather.

The pains are usually less severe than in other regions, although their sharp, sticking character impairs chest movements, especially since all movement tends to aggravate them. Tender points are found at the site of the nerve exits near the spine. Skin hyperesthesia is extreme at times. Herpetic neuritic neuralgias are relatively common in this distribution.

Among the causes to be diagnosed may be costal caries, affections of the spinal cord and meninges, disorders of the pleura, particularly carcinoma and tuberculosis, aortic aneurism, dilatation of the stomach, carcinoma of the liver, angina pectoris, pericarditis, local trauma, fractures, etc.

Mammary neuralgia or *mastodynia*, which is frequent in the later stages of nursing, and in some women at the menstrual epoch, is a special form. The pain is usually deep within the gland, and may be accompanied by a slightly increased secretion. The whole skin may be sensitive, especially the nipple, when the superficial nerves are mostly involved. Local glandular induration occasionally occurs. This has led to the mistaken diagnosis of carcinoma, but a neuralgia may occur due to a carcinoma of the breast. Tabes may give rise to an intercostal neuralgia.

Treatment.—Local applications are useful, especially the ethyl chloride spray. Blisters are efficacious. Support by bandaging affords marked relief. General measures already described call for no further mention.

Lumbar Plexus Neuralgias.—These are most conveniently arranged as (1) lumbo-abdominal; (2) ilioscrotal or testicular, (3) crural (sciatica), (4) femoral, and (5) obturator, involving in each case certain of the branches of this plexus. Mixed and indeterminate forms are not infrequent.

The Sciaticas (Sciatic Neuralgias, Sciatic Neuritides).—It has already been indicated that it is largely indifferent whether one regards this as a neuralgia or a neuritis, since transition forms are very frequent. It consists of pain in the distribution of the nerves of the sacral plexus, the sciatic and its branches.

History.—Cotugni, in 1764, gave so clear a description of sciatica that the malady is often given his name. Valleix, in 1841, described the painful points with great minuteness. Lasègue, in 1864,[1] described his well-known symptoms of neuritis of the sciatic, since which time many monographs have appeared, the most important of which are those of Brühl, Lago, Vulpian, Bernhardt and Roger and Aymès (1923).[2]

Etiology.—Similar causes are at work here as in the other neuralgias and neuritides. It is unnecessary to amplify these causative factors. Any of the general causes found on previous pages may produce a sciatica, but special emphasis should be laid upon two or three. Trauma is responsible in many cases for the development of sciatica. Syphilitic osteoarthritis and syphilitic meningitis of mild grade are

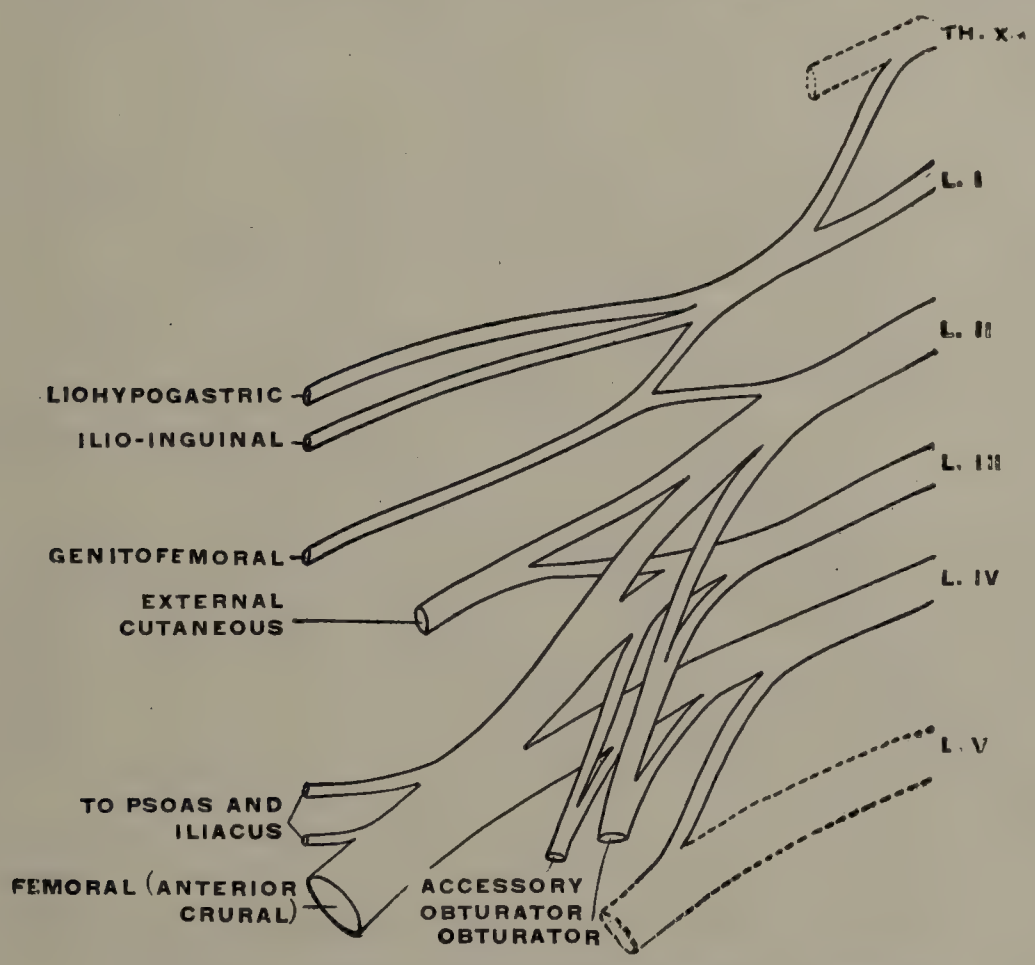


FIG. 219.—Diagram of the lumbar plexus. (Gerrish).

responsible for the development of intractable sciaticas. Certain French authors claim as high as 90 per cent. of all sciaticas to be due to this syphilitic factor. In this respect then sciatica stands in sharp contrast to the neuralgic neuritides of the upper extremities.

Gout is an infrequent causative factor, while diabetes is more common, especially for double-sided sciaticas. Double sciaticas may also be the expression of a tumor of the pelvis, of pressure due to a gravid uterus, to venous stasis, spinal-cord tumor, or new growths of the cauda. Occasionally prostatic enlargement of tuberculous or gonorrheal origin gives rise to sciatic pains. Exposure to cold, with prolonged standing is frequently met with in the histories, and occasionally in those who sit a great deal. Prolonged walking or

[1] Lasègue: Arch. gén. d. méd., 1864.

[2] Roger and Aymès, Paris, 1923.

marching may occasion an attack, and bicycling predisposes to mechanical injury of the nerve.

It is a comparatively common affection, particularly in men, being one of the most frequent neuralgias met with in dispensary practice. Chronic constipation as a cause should not be overlooked. The etiological factor in some cases is impossible to find. This is largely a result of inadequate methods of examination.

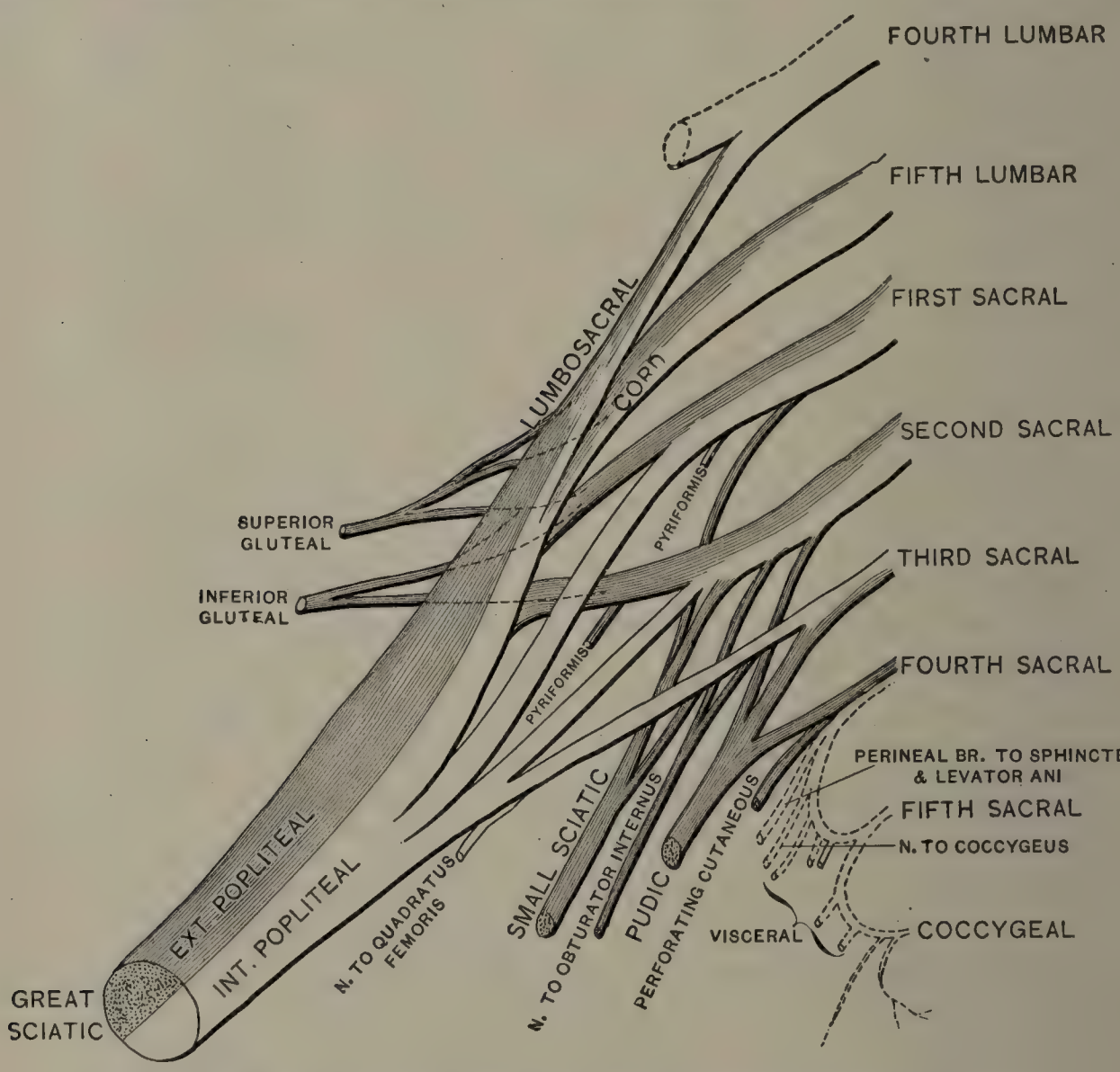


FIG. 220.—Plan of sacral plexus with the pudendal plexus. (Gerrish.)

Symptoms.—There is no one sciatica, there are many, and it is advisable at the outset to separate those cases in which the principal symptoms are pain and inability to use the limb from those in which there is added nerve tenderness, with motor, sensory and trophic phenomena.

The pains rarely commence abruptly, but beginning more or less gradually from a sense of soreness to uneasiness with occasional twinges, gradually develop into well-marked severe pains, usually at first more intense just beneath the sciatic notch, gradually extending from above downward to the entire distribution of the sciatic and some of the branches. There is usually considerable variation in the character of the pain during an attack. Some patients suffer for some time simply from boring, dull pains, while others have excruciatingly sharp, stabbing twinges that make the slightest movements impossible.

Rarely continuous, the pains come in attacks, sparing almost no region of the distribution of the plexus either *en masse* or picking out special branches.

The pain in the proximal portions of the leg is usually deep-seated, but becomes more superficial distally. In some patients an extensive series of involuntary adaptive positions take place in order to seek the most comfortable position, not only of the thigh and leg, but of the pelvis, or even the vertebral column.[1] (Scolioses.)

Valleix's painful points are fairly constant. In some cases, often mild, pressure points are lacking. Lasègue's phenomenon is of considerable diagnostic significance. It is brought out either in the lying or sitting position. The patient's foot is grasped with one hand, the other placed upon the knee, and keeping the leg stiffly extended the thigh is flexed upon the pelvis, when a severe pain develops beneath the knee or higher up in the course of the nerve. In the sitting position the pain is more apt to be beneath the knee, since the extension on the pelvis cannot be made so extreme.

With the general extension of the neuritic process—as in many alcoholic cases—the entire nerve may be sensitive to pressure.

Minor[2] has described some interesting findings on having the patients arise from a prone posture. Patients with well-marked sciatica can rarely get up—without excessive pain—with the arms crossed. They put their two hands behind them, push the hips backward between the arms, flex the knees slowly beneath the buttocks, then gradually with one hand on the hip, and then on the knee, the other balancing in the air, gradually come to a standing position. The procedure is not invariable, but is useful in gaining some idea as to simulation, and as to the diagnosis of a lumbago. The untrained simulator gets up in a variety of ways, the lumbago patient usually rises on all fours, the arms in front, somewhat as does the pseudo-hypertrophic dystrophy patient. Neri, Bonnet, Sicard, Roussy and Cornil have all described special maneuvers for testing the sciatic.[3]

In those groups of patients with more serious neuritic involvements there are added the signs of muscular atrophy, of circumscribed paralyses, of fibrillary contractions, of contractures, of sensory disorders, and of trophic disturbances.

The atrophies may be true atrophies, but are usually position or disuse atrophies. They are demonstrated by palpation, or by measurements.

Moreover, the atrophies may be segmentary, or radicular, in either of which instances the localizing diagnosis is aided. The electrical examination of the muscles in sciatic neuritis is usually contradictory. In positive cases with partial R. D. the nature of the process becomes clearer.

[1] See Roger and Aymès for literature regarding these antipain positions and resulting scolioses.

[2] Deutsch. med. Wchnschr., 1898

[3] See Roger and Aymès (loc. cit.).

Reflexes.—These show fairly constant alterations especially after the disorder has progressed several days. The *Achilles* reflex is usually normal early, then diminished or lost, rarely exaggerated. After five to eight days it may be lost and remains absent even after the pain has ceased. Alternate areflexia has been observed (André Thomas; Accornero). Loss of the retromalleolar reflex may precede that of loss of the Achilles reflex (Barré), as may also the medio-plantar reflex (Barré and Guillain[1]). The posterior tibial reflex and the posterior femoro-peroneal reflex (L_5, S_1, S_2) may be lost or remain normal. Dissociation of these reflexes may occur. The knee-jerk is more frequently preserved or may be exaggerated.[2] The periosteal and bony reflexes give inconstant results. Of the cutaneous reflexes, the gluteal has shown an exaggeration. In cases of diffuse sacral root involvement exaggerated anal and sacro-lumbar cutaneous reflexes are found. The plantar cutaneous reflex is often abolished. The cremasteric and abdominal reflexes are usually normal or exaggerated. Muscular hyperexcitability is frequent and may induce a true or false Babinski. Foix has stated that the postural reflex of the leg is conserved even if the Achilles reflex is lost.

Sensibility is often unaffected, but careful testing, following Head's methods, may show a hypesthesia to touch, pain and temperature. Occasionally the disturbance of sensibility shows a marked radicular distribution, which speaks for a more or less localized process in the plexus, frequently of a syphilitic, meningomyelitic, or osteoarthritic nature.

Trophic and vasomotor phenomena are not infrequent, consisting either of acroparesthesiæ, erythemas, local cyanoses, diminished or increased perspiration, changes in the growth and character of the hair or even the nails. True herpes is rare. Glycosuria, polyuria, azoturia are rare complications. Buscaino has reported irregular pupils, tachycardia, abolition or inversion of the oculo-cardiac reflex.[3] The cerebrospinal fluid in 50 per cent has shown mild lymphocytosis (Leri and Schaeffer), also hyperalbumosis (André Thomas). Petrén has found mild increase in tension.

Course.—In the early attacks in healthy adults, the prognosis is good. The patients usually recover in from six to eight weeks under proper treatment. A failure to respond should excite suspicion as to the diagnosis, especially with reference to tabes, to a spinal growth or a radiculitis. Recurrent cases usually develop a tormenting chronicity, which exhausts one's therapeutic resources, and occasionally drives the patient into a mental state which suicide or chronic morphinism alone terminates. Fortunately such cases are becoming rarer with better methods of diagnosis and enlarged therapeutic resources.

[1] Soc. méd. hôp., February 7, 1916.
[2] Fromental: Thèse Montpellier, 1916.
[3] Riv. di patol. nerv. e ment., June, 1918.

Clinical Forms.—Certain variants—largely based on etiological concepts—may be met with. Fournier's gonorrheal sciatica with an acute onset, slight temperature, with prostatic and articular complications is one. Brissaud has described a spasmodic type with increased tendon reflexes, contractures in the periarticular muscles of the hip, and trepidation or pseudoclonus. Quenu's varicose sciatica, which has certain analogies to the intermittent claudication of arteriosclerotic type, is characterized by deep-seated pain, and a very protracted onset and chronic course. Psychogenic sciaticas may always be expected, but they are extremely rare, except under war conditions.

Diagnosis.—The increased knowledge afforded by lumbar puncture, *x*-rays, and finer modes of testing for sensory disturbances is dismembering the old sciatica group fairly rapidly. Roger and Aymès would divide the sciaticas as follows: (1) Simulated forms; (2) false sciaticas —extraneural from articular, osseus, muscular and vascular factors; (3) parasciaticas from intraspinal or extraspinal compressions; (4) symptomatic sciaticas, as from infections, intoxications, external agents, varicosities, neoplasms; (5) essential or arthritic sciaticas—trunk, plexus or radicular.

Of the more common diagnostic errors, tabes dorsalis and lumbago call for special mention.

Tabes lacks the pressure points, the Lasègue phenomenon, and usually shows the lost knee-jerks, lost Achilles-jerk, and possibly the Argyll-Robertson phenomenon. The pains are usually bilateral.

Lumbago is usually much relieved by the recumbent posture, and is increased by the movements of the trunk; the site of the pain is, as a rule, higher.

Lumbo-sacral radiculitis calls for special scrutiny since many of the classical chronic sciaticas fall under this disorder.

Muscular rheumatism (myositis) lacks the pressure points, Lasègue's sign, and the pains are more diffuse.

Spinal-cord tumors in their initial symptomatology cause sciatic pains, usually bilateral, occasionally unilateral, but careful sensory examination soon shows anomalies; special localizing signs, pareses and trophic symptoms point to a severe lesion of the cord.

Hip-joint disease lacks the classical situation of the pains. Arthritis deformans cases with sciatic pains show diminished power of abduction and adduction and the joints are painful.

Intermittent claudication occasionally gives rise to diagnostic difficulties. Its arteriosclerotic nature is revealed by palpation of the bloodvessels, and by roentgen-ray examinations which show the tortuous modified vessels.

Achillodynia, Morton's tarsalgia, relaxation of the sacro-iliac joint, and flat-foot occasionally cause sciatica-like syndromes.

Therapy.—Rest and quiet are the first essentials. Then an etiological therapy becomes imperative. Mercury for syphilitic cases, quinine for malarial cases, surgical intervention for pressure cases, diet for

diabetic cases, rest for sacro-iliac cases, orthopedic measures for flat-foot, etc.

While one is waiting to obtain a clear notion as to etiology, general treatment may be necessary. Such treatment should always be regarded as provisional, not final.

For the treatment of the pain, analgesics are imperative. These are numerous, and different patients will respond to different members of the group. Even during an attack it may be found that one analgesic has lost its value, and another must be substituted. It is of value to bear the chemical structure of the various analgesics in mind in one's therapeutic endeavors. Antipyrin, or its related products aspirin, salipyrin, pyramidon; the amido-phenol series, with phenacetin, lactophenin, exalgin, apolysin, citrophen, phenosal, phenocol, and salocol, as representatives are often of signal service, but require considerable testing. Acetanilid itself, with its compounds, is in wide use. These analgesics can be used in combination, when smaller doses of the two or three in use are more efficient than large doses which often have marked toxic action, either on the blood cells (acetanilid, amido-phenol series) or on the vasomotors (antipyrin derivatives).[1]

In the presence of anemia, iron and arsenic should be added.

Counter-irritation is especially useful. It is best practised by means of the actual cautery (Paquelin) but mustard plasters, cantharides plaster, deep but careful massage, etc., are useful adjuvants. Applications should be made along the nerve trunks.

Hydrotherapy is often extremely valuable but must be employed with reason. A too energetic hydrotherapy with massage often aggravates a sciatic pain, especially in the initial stages when rest is so imperative. Later hot packs, mud baths, spray douches, with mild massage are indicated. In many patients the treatments carried out in bath resorts is especially indicated. Hot-air treatment is not well borne in the initial stages, but later is grateful and of therapeutic value. Artificial diarrheas are frequently helpful (Carlsbad, magnesium sulphate, etc).

Direct nerve injections of substances having a degenerative action on nerve fibers, osmic acid, carbolic acid, etc., are to be condemned. Infiltration methods, using water or cocaine, or allied substances, or various mixtures have more to recommend them. Schlosser has reported excellent results but has also had permanent palsies follow his injections. Our experiences is against these methods.

At times it may be deemed necessary, by reason of the severe pain, to practise injections of stovaine, cocaine or allied substances into the region of the cauda or into the spinal cord (Corning). Such injections are useful, but their action is temporary as a rule. Nerve stretching is to be condemned.

The opium derivatives should be used only as a last resort in senile cases.

[1] See Fraenkel, Die Arzneimittesynthese, 1927, for newer synthetic remedies.

Electrotherapy.—The older methods of galvanization and faradization are useful in a few cases, but on the whole are unsatisfactory. Sinusoidal currents are more valuable, while the Leduc rapidly alternating currents are almost always of some service in relieving pain but not in curing. High-frequency currents with the use of the ultraviolet rays at times give extremely satisfactory results from the same standpoint. Diathermy sometimes relieves.

Surgical Treatment.—Decompressive laminectomy or posterior radicotomy have been of service in arthritic compression cases. Cauda equina lesions, spinabifida occulta, sacro-iliac injuries, disease within the pelvis, varicosities of the nerves and other situations may call for surgical intervention.

Lumbo-abdominal.—These occupy the lower half of the trunk, and are extremely variable. The chief nerves involved are the iliohypogastric and its branches, the inguinal, and genitocrural. Strict localization to one trunk is rare, and men are more frequently affected than women. The chief causes, in addition to those of general moment, are local inflammatory conditions or new growths involving the plexus or some of its branches. The pains are usually unilateral, occasionally bilateral, involve the region of the back below the ribs, the gluteal region, the abdominal and inguinal areas, the scrotum, or the labia. The chief Valleix's points are over the lumbar vertebræ, the hip or iliac point, hypogastric point, and the scrotal point. Lumbo-abdominal pains are usually accompanied by intercostal pains above or thigh pains below.

Testicular Neuralgia.—Astley Cooper termed this neuralgia the "irritable testicle." The pains are usually unilateral and pass into the testicle which may be swollen and tender to the touch. The pain not infrequently passes into the leg and back, and the patient may have an attack of vomiting. Bernhardt notes that the pain may be so intense as to cause the patient to seek castration. The affection is an obstinate one, and is not helped, as a rule, by removal of the testicle. Diagnosis involves a rigid exclusion of somatic disorder of the testicle although many affections (gonorrhea, tuberculosis, chronic prostatitis etc.) are not infrequently accompanied by persistent neuralgic pains.

Crural Neuralgia.—The crural or femoral nerve is here implicated. The pain extends in the upper front and inner side of the thigh, to the knee, and farther through the saphenous distribution to the ankle and inner aspect of foot, extending as far as the big toe. It is almost entirely confined to men, and shows considerable variability as to the branch involved. It not infrequently accompanies a sciatica. Special etiological features are found in fecal impaction, or even chronic constipation, disease of the hip or knee bones, enlargement of the inguinal glands, aneurism of the iliac artery. Charcot called attention to the frequent association of crural neuralgia and diabetes. Spinal arthritis is an obscure cause.

Movements of the thigh usually are painful and the patient comes to bend his body forward in a strained position. The painful points of

greatest frequency are just below Poupart's ligament, just within the inner condyle, over the malleolus, inner side of the instep, and one over the great toe. Neuro-atrophic changes usually occur in the quadriceps, but the patellar reflex is rarely affected, save when a definite neuritis is present. Herpes, reddening, hyperesthesia are not infrequent. In the diagnosis, disease of the inguinal vessels is to be looked for, as well as intrapelvic disorders, new growths, etc. Crural neuralgias have a fairly good prognosis.

Femoral Neuralgia.—Here the cutaneous femoris lateralis, arising higher up in the pelvis, is involved. The pain is felt in the upper and outer aspects of the thigh, extending to the knee. A painful point over the anterior spinous process of the ilium is usual. Paresthesia in the distribution of this nerve has been extensively studied (meralgia paresthetica). The relation of the pressure of corsets in the causation of this type of neuralgia has been pointed out by Freud, and much sitting in adipose individuals is frequently associated with this neuralgia. The prognosis is favorable.

Obturator Neuralgia.—Lesions of this nerve are fairly constant as a result of the pressure of the intestinal loops of a hernia. The pain is located in the inner side of the thigh, and is accompanied by a feeling of stiffness, creepy, crawly feelings of the skin, and inability to bring the thigh toward the middle line of the body.

Neuralgias of the Pudendal Plexus.—A large number of neuralgias of the genital plexus are recorded. The median hemorrhoidal branches, distributed to the rectum, bladder, and vagina, the inferior branches to the anus, and the pudendal nerve supplying the testicular sac, the labia, penis, urethra, and clitoris, are the chief nerves involved. The general terms, spermatic neuralgia, anal neuralgia, perineal neuralgia, rectal neuralgia, vesical neuralgia or cystalgia, urethralgia, prostalgia, penis neuralgia, irritable uterus, ovarian neuralgia, are utilized to describe these different affections. These neuralgias are very rare, but often very obstinate. Spermatic neuralgias are among the most frequent, and are not infrequently accompanied by painful priapism, perhaps ejaculation.

Since the advent of bicycle riding neuralgias of this general region have been on the increase. The ovarian neuralgias are complex, and more often come within the domain of the gynecologist, as structural defects are often the underlying causes. Localized herpetic eruptions accompany neuralgias of this plexus. Lesions of the cauda equina are to be carefully excluded in neuralgias of this region.

Neuralgias of Coccygeal Plexus.—Coccygodynia, painful coccyx, is a not infrequent disorder in women, especially in multiparæ and in the badly constipated. Trauma and caries are frequent causes. The psychogenic coccyx is not unusual and referred coccygeal pains are common. The pain is so intense at times that defecation is rendered impossible; the patient cannot sit, and a grave neurasthenic condition supervenes. The medico-legal significance of coccygodynia is real, appearing frequently as a local symptom of a general traumatic

neurosis. Surgeons frequently lay considerable stress on a freely movable coccyx in accident litigation. A just estimate of the true bearing of an injury to the coccyx can only be arrived at by a careful survey of all of the factors of the particular case.

Local treatment is seldom efficacious save in the truly neuralgic types. Resection is rarely a justifiable procedure.

HERPES ZOSTER: SHINGLES: ZONA. RADICULO-GANGLIONIC SYNDROME (ACUTE POSTERIOR POLIOMYELITIS).

In a broad sense zoster consists of a special type of painful erythematous eruption with formation of vesicles occupying the radicular distribution of the segment involved, due to disease of the posterior roots and the sensory ganglion. In this sense it may be due to an acute or chronic meningitis, tabes, Pott's disease, carcinoma of the vertebræ, acute infectious diseases, intoxications or other lesions implicating the posterior roots and the ganglion (symptomatic zoster).

In a narrower sense it may be conceived of as a specific infectious disease affecting the ganglion cells in the posterior spinal ganglia and the adjacent fibrillary structures (essential zoster or posterior poliomyelitis).

History.—Zona was first well described by Rayer in 1835, although notes on its occurrence date from Hippocratic times. Baerensprung, in 1861, gave the first important monograph, and pointed out the implication of the ganglia as an essential feature of the disease, while Landouzy (1883) and Head and Campbell (1900) called particular attention to a specific infectious type. Rosenow and Oftedal[1] have isolated streptococci from the ganglia.[2] Levaditi,[3] Achard,[4] Bruni and Doerr[5] have contributed recent important monographs.

Etiology.—Nearly all of the general causes which give rise to a neuralgia or a neuritis may by an extension or an intensification of the pathological process involve the posterior ganglia and thus develop a herpes. In poisoning by arsenic and carbon monoxide these ganglia seem to be specially affected, and the acute gastro-intestinal affections, pneumonia, and tuberculosis are not infrequently contributory factors. Trousseau (1865) first called attention to the zoster, which was a specific infection, which type has been so extensively studied by Head and Campbell.[6] Epidemics of zoster point to the truth of this position.

Symptoms.—Neuralgic pains and a skin eruption constitute the main symptoms. The disorder shows a slightly different order of development according to the etiological factors. In the pure or

[1] Jour. Am. Med. Assn., June 12, 1915.

[2] E. Salomon: Ztschr. f. d. ges. Neurol. u. Psych. Ref., **7**, 345, for recent work on this syndrome.

[3] Levaditi: Herpes et le zona, Paris, 1926.

[4] Achard: Herpes et le zona, Paris, 1925. Bruni: Eziologia dell Herpes, Paris, 1923.

[5] Doerr: Zentralbl. f. Haut. u. Geschlk., 1925, **13**, 417.

[6] Brain, 1900, **23**, 353.

essential zosters (acute posterior poliomyelitis) there is usually a feeling of malaise, a slight temperature, and gastro-intestinal disturbances, then the patient has neuralgic pains which may be mild and superficial, burning or pricking, or deep and extremely severe, and in from three to four days an eruption develops. There is marked hyperesthesia of the skin along the affected segment, with redness, and suddenly or gradually there appears a group of vesicles varying in size from a few millimeters to a few centimeters. These vesicles are rarely confluent, and the fluid, which is at first serous, sometimes tinged with blood, may later become purulent. The vesicles gradually dry, leaving a scaly, yellowish-brown stained scar which persists for a long period. Ulceration or gangrene occasionally occurs, especially in diabetes.



FIG. 221.—Herpes zoster. Typical thoracic location. (Knowles.)

The cycle occupies about four to eight days. There is usually some anesthesia to both epicritic and protopathic sensibility after the acute stage has passed. One attack seems to confer immunity.

In the symptomatic zosters of the infectious type there is rarely fever or gastro-intestinal disturbance, the development of the eruption is usually irregular, and it often shows a chronic character. Symptomatic zona may involve both sides, whereas the infectious (influenza, etc.) type is nearly always one-sided.

In the dorsal types only is the girdle distribution maintained (intercostal herpes zoster), whereas involvement of the Gasserian, cervical, lumbar or sacral roots gives rise to irregular eruption appearances by reason of the segmental complexities of these regions.

Ophthalmic zoster is an especially severe type, occurring in individuals above middle life, and often accompanied by alcoholic and arteriosclerotic factors. It may develop apparently like an erysipelas of the face, with severe neuralgia, and then a widespread vesicular eruption, even involving the mucous surface, develops. Ocular complications, conjunctivitis, keratitis, iritis, of a severe nature, are not infrequent. Facial palsy may be present in zoster of the cephalic ganglia; thus the geniculate. A symptomatic ophthalmic zoster due to lesions in the region of the pons occurs.

Pathology.—In essential zona there is an acute, often hemorrhagic inflammation in the sensory ganglia. These are swollen, the capsule notably thickened, with marked infiltration of leukocytes. The ganglion cells are in part destroyed or damaged, and the contiguous fibrillary structures, both central and peripheral, are also involved in the inflammatory reaction. The inflammation is usually limited to a few ganglia. In the spinal cord secondary degenerations have been observed, and occasionally there is an extension of the general process to the cord. In certain cases of what appears to be essential zona the ganglia have been free, the only lesions found being those of a neuritis. There is an inconstant lymphocytosis of the cerebrospinal fluid in the infectious zones.[1]

Treatment.—For the symptomatic cases, the cause must be found. Otherwise the treatment is purely symptomatic. Local applications of zinc oxide ointment for protection and the use of a mild antiseptic to prevent suppuration are advisable. For the pains the analgesic remedies, already spoken of in the treatment of neuralgia, are useful. Gastro-intestinal therapy relieves the discomfort and itching somewhat, and may possibly limit the accumulation of a possible secondary irritant.

RADICULITIS.

The radicular syndrome, often confused with neuralgia and neuritis, is due to an inflammatory or traumatic lesion of the spinal nerve roots, usually of the brachial, and of the lumbosacral plexuses.

Attention has been given to it chiefly by French neurologists, notably Dejerine (1905) and his pupils.[2]

Symptoms.—These are neuralgic pains, which are usually severe, yet very variable. They usually occur in crises, and are sticking and lancinating in character, at times extremely intense; not infrequently resembling the pains of tabes. The pains have a tendency to remit and then to recur at shorter intervals, leaving a certain soreness behind. The nerve trunks are usually not markedly painful. There is usually a marked hyperesthesia over the radicular segment.

[1] Schaller: The Cerebrospinal Fluid in Herpes Zoster, Calif. State Jour. Med., 1918. See Baerensprung (Charité Ann., vols. **9**, **10**, **11**) for earlier pathology of posterior ganglia See Levaditi (loc. cit.) for complete reports on pathology. Wohlwill: Ztschr. f. d. ges Neurol. u. Psychiat., 1924, **89**, 171.

[2] Dejerine and Thomas: Maladies de la moelle epiniére, 1909; Revue neurol., 1917. Mayer, E.: Jour. Am. Med. Assn., August 3, 1918. Camus: Les Radiculites, 1910.

It does not follows the peripheral distribution as in a non-radicular neuralgia. This hyperesthesia is usually followed by an anesthesia to both epicritic and protopathic sensibility, and occasionally bony sensibility is involved as well if the inflammatory reaction is intense. Deep sensibility may be so involved as to cause astereognosis. Paresthesiæ and acroparesthesiæ are common.[1]

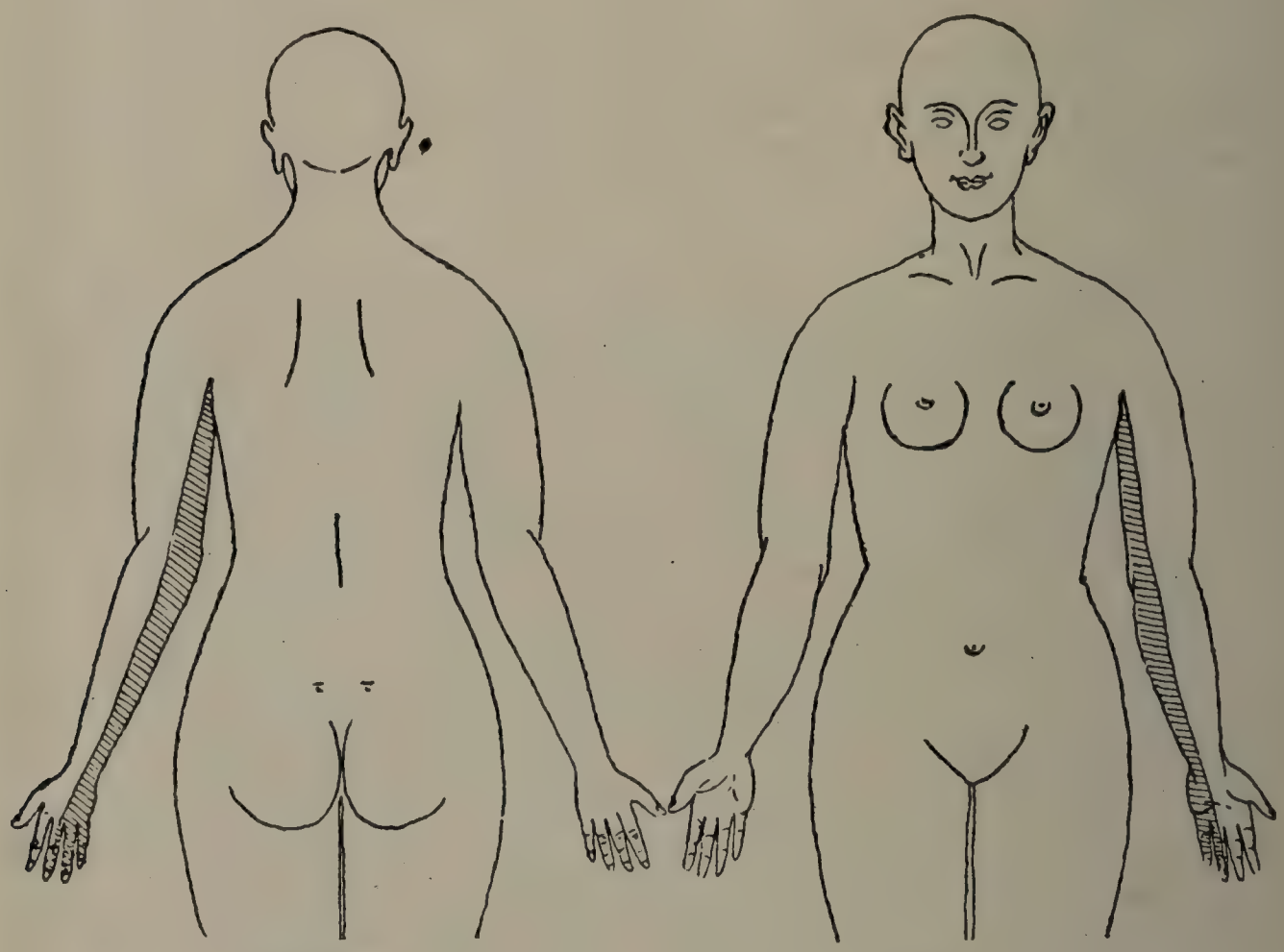


FIG. 222 FIG. 223

FIGS. 222 and 223.—Topography of the sensory disturbance in a syphilitic radiculitis (type Klumpke). The C_8 and D_1, distribution are involved. (Dejerine.)

It is essential that these features, which may be found in other affections, be radicular in their distribution. They are not segmentary, *i. e.*, involving the hand, the forearm, or the arm; nor do they follow the peripheral nerve distribution. They are distributed in long bands down the arm or the leg, corresponding to the root segments involved. (See Figs. 230, 234, and also Figs. 27 to 32, and Plates XII and XIII.)

It is a striking fact that sneezing or coughing may bring on a paroxysm of pain in the cervicodorsal plexus, and coughing and straining at stool may bring on pain in a sciatic radiculitis.

In the upper extremity it is rare to find a pure radiculitis, *i. e.*, one without some motor involvement, while for the lower limb the great majority of the classical cases of sciatica are due to a radiculitis.

In the mixed cases, involvement of the anterior roots causes muscular atrophies, likewise radicular and not peripheral in their distribution.

[1] Rousellier, Paris Thése, 1907.

Explanation of Plate XII

The First Sensory Neuron and the Origin of the Spinal Part of the Secondary Sensory Paths.

1. First Sensory Neuron.—Its origin in the posterior spinal ganglia; the cutaneous root zones of the head, the trunk, the extremities; tributary of the peripheral sensory nerves. The constitution of the posterior columns of the cord due to the posterior root fibers; their arrangement at different levels of the spinal cord and the mixed zones of endogenous and root fibers.

In Violet.—The first neuron of the trigeminus, its root field—frontocranial, nasociliary (V_1), nasojugal (V_2), and maxillar (V_3), the long, descending spinal root (*Vsd*) which surmounts the gelatinous substance of the posterior roots not descending beyond the upper border of the fourth cervical segment.

In Pink.—The superior cervical neurons (C_2, C_4), the radicular zones of the neck (C_2, C_8) and of the head (C_2) and of a portion of the shoulder (C_4); tributary of the superficial cervical plexus and of the posterior occipital. The area of the posterior root fibers (C_4 to C_1), in the posterior root zone of the cervical cord, and in the anterolateral column (dotted rose), the crossed (*Pyc*) and direct (*Pyd*) pyramidal tracts.

In Red.—The inferior cervical neuron (D_1); the cutaneous root zones of the upper extremities (C_5, D_1); tributary of the brachial plexus and the area of the posterior root fibers (D_1, C_8) in the posterior columns of the cord.

In Yellow.—The dorsal neurons (D_5 and D_{12}). The cutaneous root zones of the trunk (D_2–D_{12}), territory of posterior (dark yellow) and anterior (light yellow) dorsal nerves (D_2–D_{12}). The field of the posterior root fibers (D_{12}–D_2) in the posterior dorsal and cervical cord.

In Pale Blue.—The lumbar neuron (L_2). The cutaneous root zones of the buttocks and thighs; territory of the posterior branches of the lumbospinal nerves (L_1, L_3) and of the nerves of the lumbar plexus (L_1–L_4). The field of the posterior root fibers in the (L_4–L_1) in the posterior columns. This field area reaches the surface of the cord in the upper lumbar and upper dorsal segments. It occupies a deep position in the cervical cord, where it is arranged on each side of the median line in front of the area of the lumbosacral root fibers.

In Dark Blue.—The sacral neuron (S_2). The root zones (L_5–S_3); territory of the ischiatic plexus and, in the posterior column, the field of the posterior root fibers (S_3, L_4) at different levels of the cord, lumbar, dorsal and cervical.

In Black.—The sacral neuron (S_3). The cutaneous root zone (S_3) territory of the genital plexus. In the posterior column, the area of the posterior root fibers (S_6–S_3) at different levels of the spinal cord. In the lower sacral segments (S_4 and S_3). This area occupies the entire area of the posterior columns, above, little by little it is contracted by the incoming lumbosacral (S_3, L_4), lumbar (L_4–L_1), dorsal (D_{12}–D_2), and cervical (D_1–C_6, C_4–C_1) fibers. It diminishes rapidly in its ascending path, a few long root fibers, of S_3 alone reaching the medulla where they terminate in the nucleus of Goll.

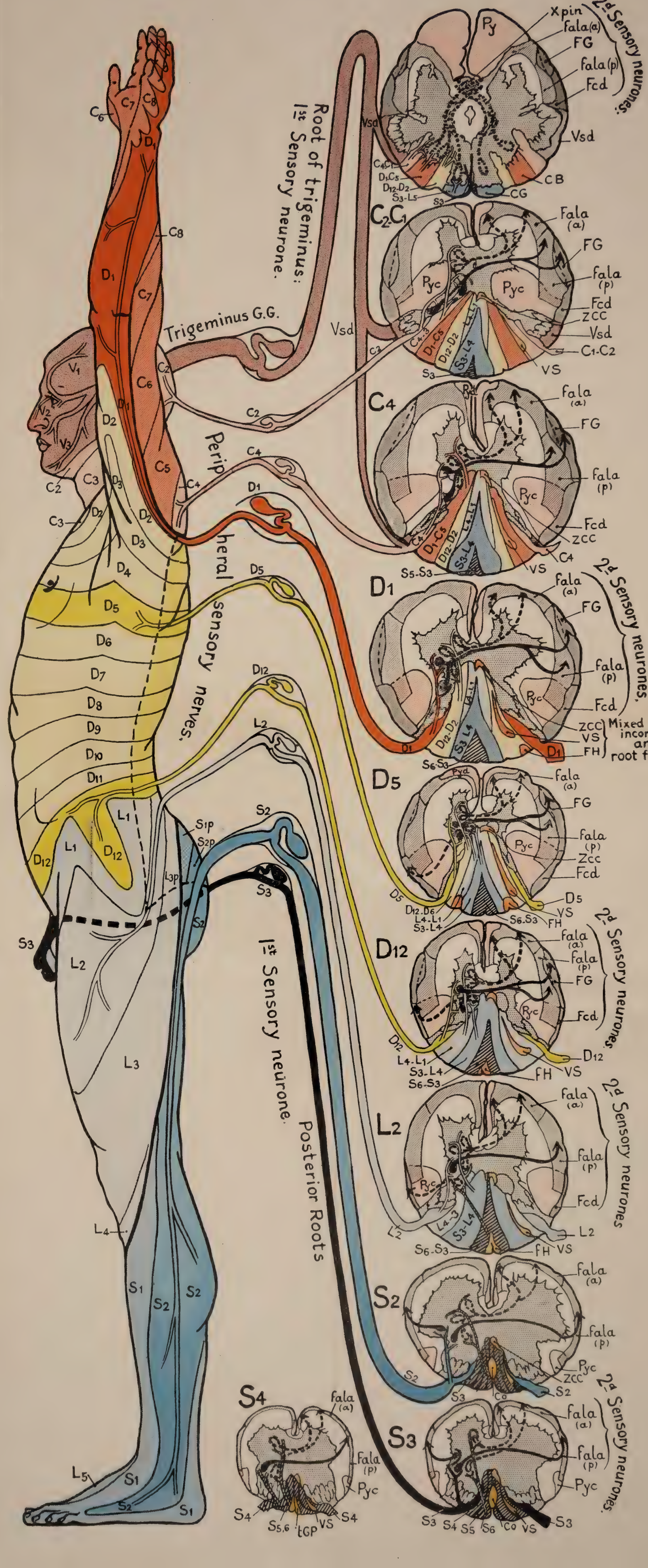
The arrangement of the long root fibers of the different spinal segments is such in the cervical region of the cord that the column of Goll (*CG*) is made up of the long root fibers of the inferior sacral (black), lumbosacral (dark blue), lumbar (pale blue), inferior dorsal (yellow), and the column of Burdach (*CB*) is made up of the long root fibers of the upper dorsal (yellow) and the long and median root fibers of the lower cervical (red) and upper cervical (pink).

In the right posterior column are indicated in orange those zones rich in endogenous fibers, thus: the *cornu commissural zone* (*Zcc*) and the *comma tract* of Schultze (*vS*) which occur at all levels of the cord. The peripheral band of Hoche (*fH*) appearing in the upper dorsal region migrates inward, reaching the middle line in the upper lumbar segment. The *central oval* of Flechsig (*Co*) which follows the former in the lumbosacral cord (L_3, S_3) and the *median triangle* of Gombault and Philippe (*tGP*) which is found in the lower sacral cord (S_3 S_6). These are mixed zones containing endogenous and root fibers; the cornu commissural zone containing short root fibers ascending and descending, the comma tract of Schultze short root fibers and long descending fibers from different spinal segments, Hoche's tract long, dorsal descending root fibers; the central oval and medial triangle, descending long lumbar, lumbosacral and sacral descending root fibers.

2. The Origin of the Spinal Portion of the Secondary Sensory Neurones.—(*a*) The secondary sensory pathways for the encephalon. *Anterolateral ascending* fibers (light gray oblique lines), posterior segment (*fala,p*) and anterior (*fala,a*); (*b*) secondary sensory paths for the cerebellum (dark gray, oblique lines): *direct cerebellar tract* (*Fcd*) (Flechsig) and the *tract of Gowers* (*F.G.*). Respective situations of these tracts at different levels.

In the gray substance are indicated in plain black lines (———) the synapses of the secondary sensory neuron with the short root fibers and which give rise to the posterior segment of the ascending anterolateral tract (*fala,p*) and Gowers' tract (*F.G.*) broken lines (— — —) those synapses with the short root fibers and which give rise to the direct cerebellar tract (*Fcd*) and the anterior segment of the ascending anterolateral tract (*fala,a*); in dots (. . . .) the synapses of the nuclei of Goll and Burdach and the long root fibers of the posterior columns, and which, entering into the piniform decussation (*xpin*), give birth to the rubrothalamic tract.

The ascending anterolateral fibers occur at all levels of the spinal cord; located at the periphery of the anterolateral column in the sacral and lumbar segments and in the dorsal and cervical segments inside of the cerebellar tracts. They are sparsely distributed fibers, not collected into a compact bundle, the largest fibers lying more to the periphery. The long fibers of the posterior segment (spinospinal, spinoreticular, spino-olivary, spinotectal, spinothalamic) are intermingled with the cerebellar fibers and with the descending fibers of the crossed pyramidal tract and the prepyramidal (rubrospinal) fibers. The long fibers of the anterior segment displace themselves little by little toward the external peripheral of the column, and are there intermingled with the descending fibers of the cerebellovestibular system, with the tectospinal and reticulospinal fibers, and also with the superficial aberrant medullary fibers of the peduncular pathways when present. They are reinforced by the short spinospinal, spinoreticular fibers, and in the medulla by the fibers coming from the nucleus of Burdach and which participate in the piniform (sensory) decussation. (Dejerine.)



Explanation of Plate XIII

The First Sensory Neuron and the Origin of the Secondary Sensory Pathways of the Anterolateral Column of the Spinal Cord.

Mode of entrance of the posterior roots S_3, L_4, D_{12}, D_5, C_8 into the spinal cord. Ascending and descending branches; short, medium and long root fibers. Composition of the posterior columns by the posterior root fibers of the lower sacral, lumbosacral, lumbar, dorsal and lower cervical. The mixed fiber tracts of the root and endogenous fibers of the posterior columns, the cornu commissural zone, comma tract of Schultze, Hoche's tract, and median triangle of Gombault and Philippe. In this figure *A* represents in a way the synthesis of the sections shown in Plate XI. The colors correspond to those in Plate XI. They indicate the location occupied in the posterior columns by the sacral root fibers (S_5S_3) (colored black), the lumbosacral fibers (S_3L_4) (dark blue), the lumbar fibers (L_4–L_1) (pale blue), the dorsal fibers (D_{12}–D_2) (yellow) and the cervical fibers (D_1–C_8) (red).

Figure *B* shows the course of the pathways devoted to the different types of superficial and deep sensation in the peripheral nerve (somatic and sympathetic fibers), in the posterior root fibers and in the posterior and anterolateral columns of the spinal cord.

The colors indicate the *functions* of the different sensory fibers. Pain and temperature sense travel in those colored yellow; tactile sense in those colored pale blue; in the dark blue fibers stereognostic sense, tactile discrimination of two points, the idea of weight, of bony sensibility to tuning-fork vibrations, deep conscious sensibility of position in space, of the attitude of the members, and of active and passive movements; in green the fibers conveying deep unconscious sensibility underlying muscular tonus, coördination, direction of movement, and of the maintenance of static and dynamic equilibrium.

The specific sensations of pain, temperature, touch, sense of localization, of tactile discrimination, and of stereognostic perception are *dissociated* at the periphery and following the course of the superficial and deep nerves, while the perception of weights, of bony vibration, deep conscious and unconscious sensibility are functions of the deep sensory nerves (articular, muscular, tendinous, osseous, periosteal, etc.). Pain and temperature impressions follow the course of the sympathetic fibers; the two categories of fibers, sympathetic and somatic, enter into the composition of the nerves of superficial and deep sensibility and participate in the terminal skin, hypodermal and muscular organs—the Pacini, Meissner, Golgi-Mazzoni, Dogiel, Ruffini, Timofejew, etc., bodies.

In the spinal cord a qualitative grouping of sensations takes place. Pain and temperature sensibility (yellow) enter the spinal cord in short root fibers and are transmitted to the optic thalamus by the fibers of the posterior segment of the ascending anterolateral column (*fala,p*) on the opposite side of the cord. Tactile impressions (*pale blue*) follow the course of the median root fibers and are transmitted to the optic thalamus by the fibers of the anterior segment of the ascending anterolateral column (*fala,a*) which are concentrated near the site of emergence of the anterior roots. Deep unconscious sensibility (*green*) is transmitted to the cord by the short and median root fibers, receive collateral stimuli from long root fibers, and pass to the cerebellum by the cerebellar contingent of the secondary sensory pathway of the cord—direct cerebellar tract and Gowers's tract; this latter is made up of fibers of crossed origin. As to deep conscious sensibility (*dark blue*) it follows the course of the long root fibers of the posterior columns of the cord and enters its secondary pathway only at the level of the neck of the medulla.

A horizontal line (*ab*), which sections the base of the posterior horns, divides the cord in a posterior portion which contains above all the direct pathways arising or terminating in the homolateral side of the spinal cord and lesions of which determine homolateral symptoms, and an anterior portion which contains for the most part the crossed pathways which take their origin or which terminate in the gray substance of the opposite half of the cord and lesions of which give rise to contralateral symptoms. (Dejerine.)

The atrophy is rarely accompanied by fibrillary twitchings, or by spasmodic contractions.

Ataxias may be met with. Lasègue's sign is commonly found.

The knee-jerks are primarily exaggerated, later diminished or lost in sacral forms.

Trophic disturbances, particularly in the joints, and vasomotor signs (cyanosis) are found in long-standing cases. In rare instances the anterior roots are involved primarily.

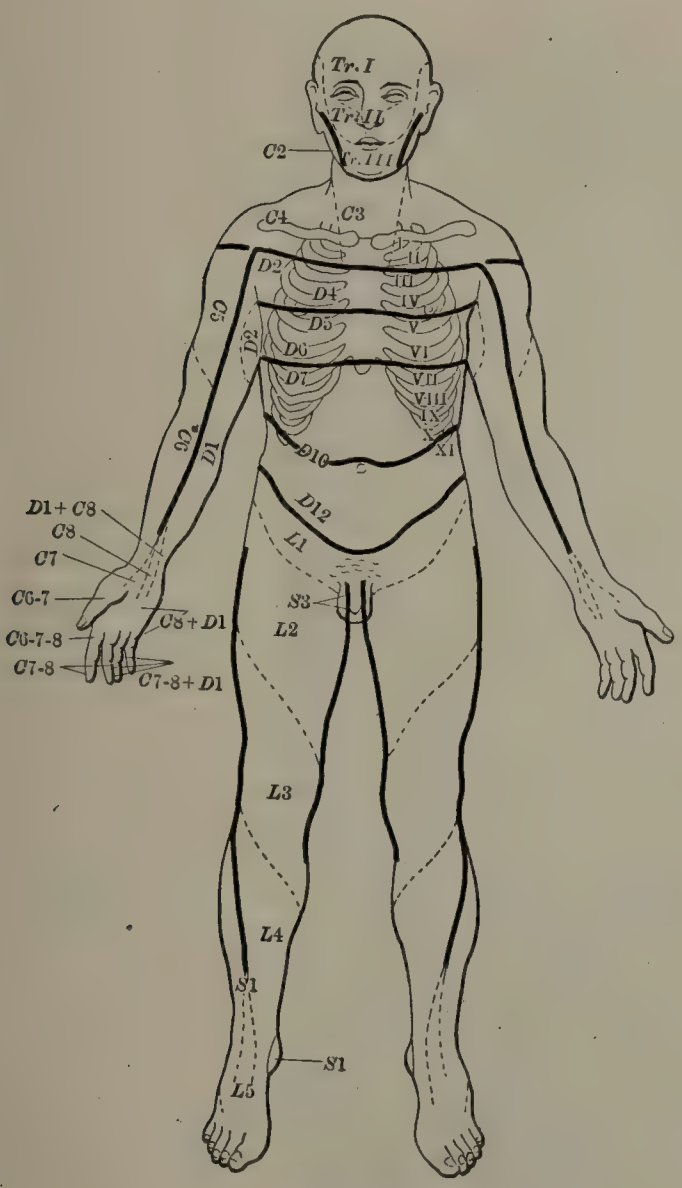


FIG. 224

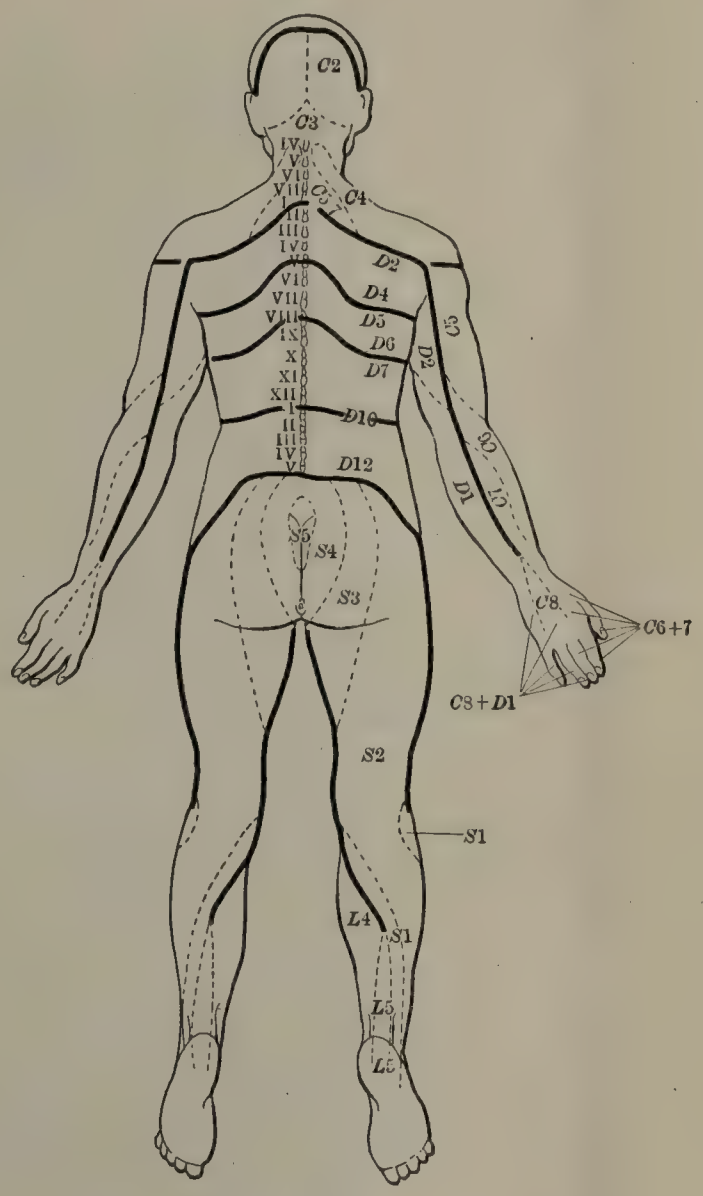


FIG. 225

FIGS. 224 and 225.—Scheme of root (radicular) segment distribution. (Flatau.)

Diagnosis.—Lumbar puncture may reveal a lymphocytosis. As noted, radiculitis of the lumbo-sacral plexus has for the most part been grouped with the sciatic neuralgias; both under the so-called true and the symptomatic neuralgias.

The presence of sensory changes, using the procedures of Head and Dejerine, the radicular distribution of the hyperesthesiæ, the anesthesiæ and the atrophies are sufficient to determine a diagnostic picture. The crural nerve is more often involved in the radicular process than in the peripheral sciaticas.[1]

[1] Dejerine: Semiologié du systeme nerveux, 1926.

The presence of ataxia in the lower limbs, occasionally in the upper, Romberg's sign, and the frequent loss of the knee-jerks often leads to the mistaken diagnosis of tabes. From one point of view tabes begins as a radiculitis, and the diagnostic difficulty centers about the etiological element. Since so much of radiculitis of the lower extremity is due to syphilis, the ordinary lumbar puncture and Wassermann tests are essential to make the diagnosis positive. In tabes one must also take into consideration the involvements of the cranial nerves, Argyll-Robertson pupil, etc.

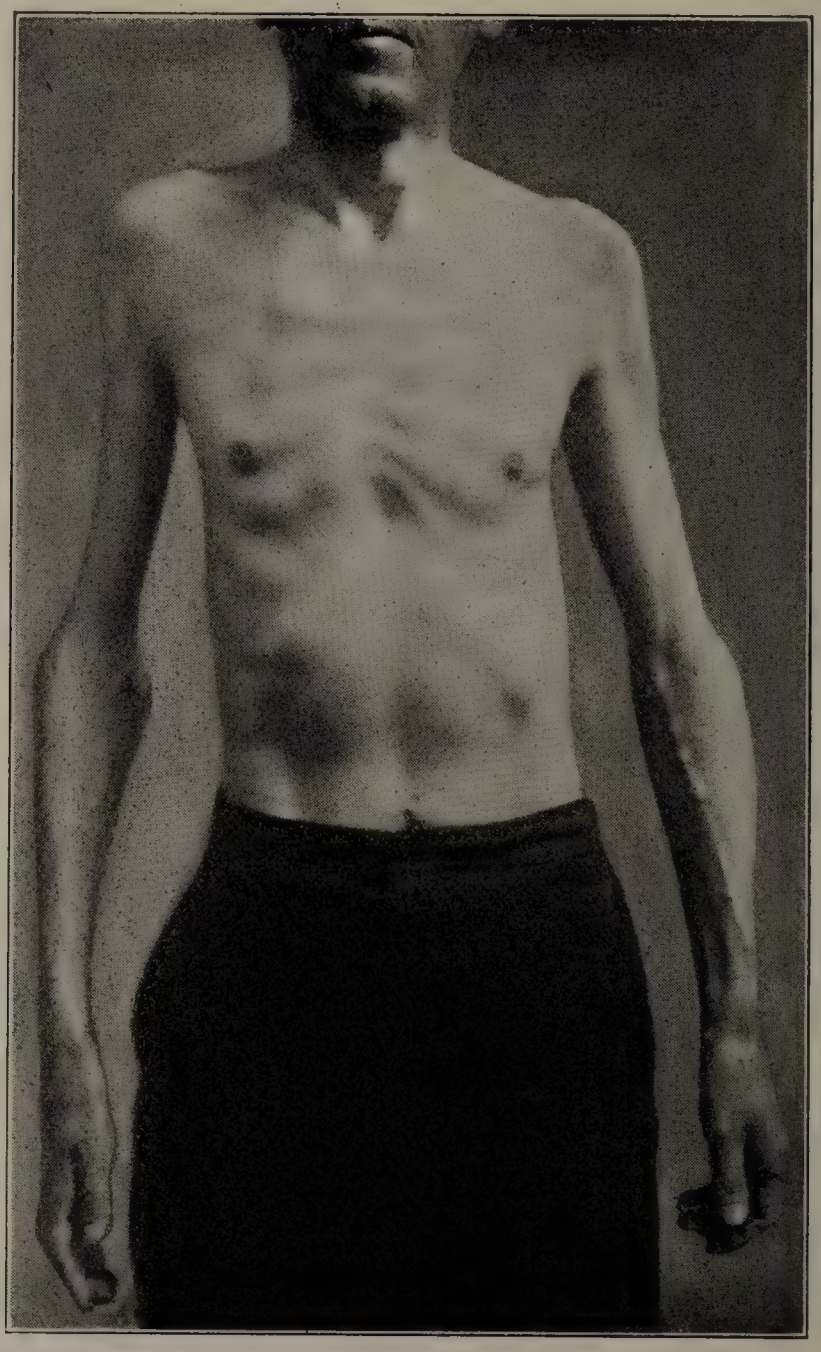


FIG. 226.—Syphilitic meningomyelitis with cervico-brachial radiculitis. (Jelliffe.)

Cervical radiculitis naturally travels under the guise of a cervico-brachial neuralgia. Here the greater implication of the sensory system is enough to make a diagnosis. In mild early cases the diagnosis of a radiculitis is impossible. Muscular atrophy is a frequent complication of cervical radiculitis. Furthermore, pupillary phenomena (Klumpke) are more common in this type.

Brachial radiculitis gives rise at times to a typical Aran-Duchenne atrophy—the biceps, anterior brachial, supinator longus and deltoid—the fifth and sixth cervical roots being chiefly involved. The Klumpke

oculo-pupillary phenomena, *i. e.*, diminution in size of the palpebral fissure, slight retrogression of the eyeball and miosis are present if the last cervical and first dorsal roots are involved.

Disseminated types are described by Dejerine[1] with practically all the signs of a tabes. Cervical rib may cause a radiculitis.

Occasionally cerebral tumors cause the symptoms of a radiculitis with alterations in the posterior columns and radicular lesions (Collier, 1899; Nageotte, 1909; Raymond, 1907). Both limbs may be involved.

Acroparesthesia was described originally by Gamberini in 1844, later by Nothnagel (1881), by Putnam (1882), named by Schultze, and then shown by Pick to be due, so far as the so-called chronic organic cases were concerned, to a radicular or intraspinal involvement, principally distributed in the lower half of the cervical region. Other areas may be involved and vegetative level acroparesthesiæ (hypothyroidisms) as well as psychogenic acroparesthesiæ also are frequent.

Its symptoms are, in the periodic cases, pains or burning, or cold sensations, usually in the fingers, accompanied by blanching and coldness of the skin with the sensation of engorgement and extreme heaviness of the hand or the fingers.[2]

Treatment.—Spontaneous radiculitis seems to be preëminently syphilitic. Hence an antisyphilitic treatment is indicated in every case of suspected radiculitis. Other forms of meningitis may produce it, however. The prognosis is good in the syphilitic forms, but less so for the others. Many patients with cervical and brachial radiculitis recover spontaneously after from six to ten months. Treatment seems to alleviate but not cure. The general treatment for a neuritic neuralgia is indicated. Violet-ray exposure should be tried in the intractable cases.

NEURITIS.

Neuritis is a generalized inflammation of the peripheral nerves, involving in varying degrees of completeness the motor, sensory and vegetative fibers. In old-standing cases the spinal portions of the neurons are implicated.

The inflammatory changes may proceed either from the perineurium, or from the endoneurium and involve the axis-cylinders. These may be poisoned and degenerate (parenchymatous inflammation). The pathological nature of the lesion bears little relation to the general symptomatology, and pathogenically considered there is considerable interplay of the various processes.

Etiology.—A vast variety of causes may bring about a neuritis, seen either as the result of acute toxic parenchymatous changes, acute degeneration due to actual pressure or injury, or some acute or chronic inflammatory changes following a variety of noxa. The most important of these causes are as follows:

[1] Rev. Neurol., 1904, p. 524.
[2] Putnam: Jour. Nerv. and Ment. Dis., September, 1916.

Infections.—Neuritis may result from the toxins of microörganisms, as those of diphtheria, tuberculosis, syphilis, influenza,[1] smallpox, dysentery, typhoid fever, pneumonia, streptococcus, occasionally measles, scarlet fever, parotitis, whooping-cough, etc. Practically there is no infectious disease that has not produced a toxic, usually parenchymatous, neuritis. A second group of infections, such as leprosy, beriberi, malaria, rabies, bring about neuritis. Neuritic types of poliomyelitis and epidemic encephalitis are known.

Intoxications.—Next in general importance are the intoxications, either exogenous or endogenous. Alcohol plays the chief role, while arsenic, lead, carbon monoxide, sulphur, and anilin compounds are rarer exciting causes. Of the autointoxications diabetes is perhaps the most important. Leukemia and also anemia cause neuritis.

Trauma.—A third important cause for the neuritic process is trauma to the nerve, either direct injury, or from the pressure of new growths, dislocations, false positions or other anomalies of structure combined with the prolonged fixed positions required in certain occupations (professional neuritis).

Less frequent causes are extensions of inflammatory processes (ascending neuritis), inflammations about joints, inflammations of adjacent organs, etc. The war neuritides form an immense group for which special monographs, which are numerous, should be consulted.[2]

Symptoms.—The symptomatology of neuritis varies enormously, not only so far as the etiological factors which determine general trends of reaction are concerned, but also as to location, onset, etc.

Only the symptoms of the generalized process will be considered here, reserving for the sections on Pareses or Paralyses, both of the plexuses and of the peripheral nerves, a more detailed description of the various isolated types.

It has already been indicated that the conceptions neuralgia, radiculitis and neuritis are very flexible—it is only for the sake of description that one draws more or less arbitrary lines between them.

There is a generalized type of neuritis which of and by itself constitutes a fairly definite syndrome. This is so-called multiple neuritis, or polyneuritis. It is largely due to toxemias, organic or inorganic.

Under the general caption of peripheral neuritis, one considers a large number of peripheral palsies, while as localized neuritis one has to consider a number of the professional neuritides.

Polyneuritis, Multiple Neuritis.—This is a general, widely distributed, diffuse, parenchymatous neuritis in which the entire peripheral neuron is involved. Primary or secondary degenerations of the cerebral neurons take place.

Etiology.—Multiple neuritis is almost invariably due to some toxemia. Such toxemias may be due to (1) alcohol, lead, arsenic

[1] Jelliffe: Loc. cit.; New York Med. Jour., November, 1918. See Crookshank, Influenza, 1922.

[2] Tinél: Nerve Wounds, 1918. Athanassio-Benisty, 1918: Revue Neurologique, Neurologie de Guerre, 1915, 1916, 1917, 1918. Stewart: Nerve Wounds of War, 1918.

zinc, carbon monoxide, bisulphide of carbon, sulphuric acid, and some of the rarer metals: mercury, copper, phosphorus, etc., (2) or to the toxins of acute or chronic infectious diseases, such as smallpox, typhoid fever, grippe, measles, scarlet fever, diphtheria, pneumonia, dysenteries, streptococcemias, leprosy, malaria, tuberculosis, parasitic worms, syphilis, poliomyelitis and epidemic encephalitis; (3) auto-intoxications such as diabetes, leukemias, anemias, beriberi, etc. Acute chilling of the body is held to be responsible for certain cases, particularly in the presence of some infectious diseases, or acute toxemias, notably in alcoholic cases, cases of rabies, influenza, etc.

Occurrence.—No general laws can be stated with reference to occurrence because of the wide range of etiological factors.

Symptoms.—Clinically considered, one meets with subacute and acute cases. In the subacute cases, which are in general milder, the patient usually begins to have a rapid progressive enfeeblement of the muscles, as a rule, of the lower extremities. There is rarely any fever at the onset, and the loss of power gradually extends from the peripheral segments toward the trunk. Thus, the extensors of the leg and of the foot first show weakness, and later those of the thigh and hip. At the same time, or shortly following, the upper extremities may be involved, in accordance with the same general law, the muscles of the hand, wrist and forearm usually being primarily involved. There are occasional exceptions to this general law of progression, but they are comparatively rare.

It is further characteristic that the weakness and paralysis are more or less symmetrically distributed. Although one leg or one arm may show a greater amount of weakness than the other there is almost invariably quadrilateral involvement. In the milder cases quantitative variations in the severity occur, and in the mild subacute cases the cranial nerves are less often diseased. Still the muscles of the abdomen, the diaphragm, the face, eyeballs or tongue may all suffer.

In the more severe cases, the implication of the pneumogastric is shown by tachycardia, dyspnea and feebleness of the pulse.

The superficial reflexes may first be exaggerated, but later become lost, as a rule, and the tendon reflexes usually exhibit the same phenomena.[1] Disturbances of sensibility are usually more marked. Initial pain is more or less universal, but the sensations of actual pain are often preceded by tingling or creeping sensations, and the skin, muscles, nerve trunks and joints may all show hyperesthesiæ. The Lasègue phenomenon is universally present.

Careful testing of epicritic sensibility may show no loss, although, as a rule, the sense of localization to light touch and the ability to distinguish between two points of a compass soon become somewhat diminished. A certain amount of loss of epicritic temperature sense may also be met with. In the milder cases the atrophy gradually disappears, and there is no tendency to the development of contrac-

[1] See Tetzner, H. L.: Berl. klin. Wchnschr., 1921, No. 51, 1494, for preserved tendon reflexes in rare cases.

tures, but in other cases contractures may follow, and the limbs become fixed and immobile.

In acute generalized polyneuritis the attack begins very abruptly, usually with high temperature and chill, headache, malaise, suppression of urine, albuminuria, and the general signs of an acute illness. Paralyses develop very rapidly, usually involving the lower extremities first, and gradually ascending the trunk and the arms, closely resembling the ascending type of acute anterior poliomyelitis (Landry). There is great tenderness on pressure over the nerve trunk, sharp, shooting pains, marked hyperesthesia of the muscles and the Lasègue phenomenon. The reflexes are rapidly abolished, the patient loses epicritic sensibility, is unable to localize touch, cannot distinguish points of the compass, but rarely loses sense of pain or of deep pressure. The sphincters are not usually involved, except *in extremis*. Atrophies, contractures, trophic disturbances of the skin, such as glossy skin, pemphigus-like eruptions, perforating ulcers, fragile nails, thick and fragile hairs, etc., develop. Then ocular palsies are met with, occasionally facial palsy, tinnitus frequently results from cochlear disturbance, and neuritic vertigo from vestibular disorder is found. Pupillary inequalities are frequent in the severe cases. Sluggish light and accommodation reflexes are fairly constant and occasionally a true Argyll-Robertson pupil is found. Loss of accommodation with retained light reflexes is met with occasionally. Amaurosis, complete or partial, is not infrequent.

The cranial nerve nuclei are not infrequently involved. In the fatal cases the implication of the pneumogastric causes death. The symptoms are those of asphyxiation, or with cardiac irregularity and paralysis of that organ.

Course.—Diagnosis.—Treatment.—Inasmuch as multiple neuritis varies not only with reference to its symptomatology and course, but offers special diagnostic problems according to the etiological factors, and since the treatment must depend upon a due consideration of the etiological factors, it is best to discuss these problems under special heads.

Alcoholic Multiple Neuritis.—This is the most common of all the types of multiple neuritis. Any form of alcohol-containing drink can cause it, including heavy beers. Cologne, ether and other substances of the marsh-gas series are among the etiological curiosities. It is not without a certain amount of interest to record that James Jackson, an American observer, called attention to this syndrome first in 1822.

It is usually subacute in development, although occasionally, especially if the patient has been subjected to severe cold, the disease may begin in a very acute manner, and present the picture of a Landry syndrome. In a few instances an apoplectiform onset has been noted. Intercurrent infectious disease in an alcoholic may constitute the point of departure for a polyneuritis; this is especially true of influenza.

These patients usually complain of tingling sensations; of formication over the hands or down the legs, with occasional twinges of

pain, particularly in the legs. The skin is usually hyperesthetic throughout the entire body, and the muscles become very sore. Such symptoms may be present for weeks or even months. The patient commences to notice difficulty in walking, inability to go up and down stairs and occasional falling when called upon for certain muscular exertions.

Examination of the muscular power at this stage shows marked weakness, particularly in the extensors of the feet, and of the hands. In walking there is a tendency to "foot-drop," and the patients lift the legs a little higher, and may show a characteristic flopping step superficially resembling that of the tabetic.

In this stage, before patients are confined to bed by reason of the muscular weakness, an exaggeration of the tendon reflexes is occasionally found, although diminution and loss become the rule. Atrophy, flabbiness of the muscles, and the trophic signs may then appear.

A number of these patients are able to be about, and are often mistaken for cases of beginning tabes, especially as there is very frequently a certain amount of ataxia, definitely marked in the lower extremities, less so in the upper. The sphincters are, as a rule, intact.

In some severe cases, in addition to the physical signs, a very definite psychosis develops. This is treated under the head of polyneuritic psychosis—chronic alcoholic delirium. Korsakoff's psychosis. (See chapter on Toxic Psychoses.)



FIG. 227.—Alcoholic neuritis in chronic stage with contractures.

Course.—Alcoholic polyneuritis shows an innumerable number of variants, but in the main it runs a subacute course. The patients go through the usual symptoms of chronic alcoholism, with tremor, sleeplessness, gastric disturbance and malnutrition. In the severe cases which develop marked mental signs—Korsakoff's syndrome—they usually go through one or more periods of acute delirium (delirium tremens).

Then the neuritic symptoms commence to appear, usually with formication or other paresthesiæ in the arms and legs. Sudden twinges of pain, particularly in the lower limbs, are frequent, and an unusual sense of muscular fatigue develops. Dynamometer readings show this loss of power and excessive fatigability early in the disorder. The average case, if drinking continues, and the malnutrition and insomnia are not overcome, takes three or four to eight weeks for the development of definite palsies. The patients note great difficulty in going up and down stairs, with much palpitation and shortness of breath. Then on some sudden exertion they fall, or their legs shut up like a jack-knife under them. They totter in their walk, or show a steppage or ataxic gait. The disease may be arrested at this stage, and recovery takes place with proper care. In advancing cases, locomotion becomes impossible. Foot-drop, wrist-drop, and ptosis may develop, and the atrophies, contractures, and trophic disturbances come on rapidly. The pains are of increased severity and frequency, and are apt to be excruciating. The hyperesthesia, which has been excessive, may now be gradually and irregularly supplanted by hypesthesia or anesthesia, hypalgesia, or analgesia, and extension to the cranial nerve nuclei may be looked for. Sensitive nerve trunks and Lasègue's phenomenon are invariably present.

The patient who has advanced to the stage of paralysis, atrophies, and trophic changes is usually confined to bed for several months, and then commences to make a slow and irregular recovery. It may be complete, but there is apt to be some local, persistent impairment which may require treatment for years, especially if fibrotendinous contractures have developed. In the Korsakoff cases certain grades of residual mental impairment are extremely common.

No two cases of alcoholic polyneuritis are alike. There is a general tendency for the disorder to involve all four extremities in the pronounced cases, but the lower extremities are more severely implicated. Hyperesthesiæ and paresthesiæ are frequent. Special predilection is shown for the extensors of the foot and wrist. Pains are extremely severe and are universal. Blindness (amaurosis) is frequent; with methyl (wood) alcohol it usually comes on in advance of any other neuritic symptoms.

Treatment.—Treatment of alcoholic neuritis involves the absolute withdrawal of alcohol in any form, complete rest, forced feeding, particularly with foods rich in fat—milk, eggs, butter. Pain is best relieved by hot applications; continuous warm baths are very grateful if there is marked hyperesthesia of skin, muscles, and nerve trunks; the temperature should not exceed 96° to 97° F., if the bath is to be continued for any great length of time. Active catharsis and diaphoresis are essential in the early stages. After the stage of acute hyperesthesia is passed the forced feeding should be continued and the use of strychnine and electricity commenced. Great care should be taken in the selection of the hypnotics used to give sleep, since so many of them contain alcohol, and a few are directly poisonous to the nerve

trunks. Bromids, hyoscine, or occasionally the opium derivatives may be employed. If an alcohol hypnotic seems necessary paraldehyde and chloral are indicated.

Glycerophosphites with calcium are valuable, preferably not given in an alcoholic medium. Massage and muscular movements of various types are indicated in the chronic stages, and most cases will repay constant working upon them. In the presence of contractures, surgical intervention may be necessary, but should be deferred until persistent massage and muscular therapy have been exhausted.

Lead Neuritis.—*Lead Palsy.*—In neuritis from lead poisoning which occurs from the gradual intake of lead in some form—drugs, hair dyes, cosmetics, contaminated water, certain occupations (plumbers, workers in type foundries, printers, glaze workers, potters, etc.) the picture is very different from that seen in alcoholism, although the terminal stages may be similar. The histological alterations are practically identical. Many cases are complicated with alcoholism.

Symptoms.—There are the initial gastro-intestinal symptoms of lead poisoning, furred tongue, constipation, attacks of colic, headaches, anemia, painful joints, and perhaps the signs of a nephritis. The gum lead line is frequently present. After a few months, or even a year or so of exposure, the neuritis develops, often after an attack of colic. It usually attacks the upper extremities, by preference, although there is always some slight involvement of the lower limbs. (Sciatica.[1]) Lower limb palsies are rare, and usually occur only in children. The pareses predominate particularly in the extensors of the index finger and thumb —the sensory symptoms, hyperesthesiæ, pains, nerve tenderness, Lasègue's phenomenon, paresthesiæ, are usually much less than in alcoholic neuritis. The supinator longus is very frequently spared. The paralyses are usually symmetrical, but may be quite irregular; the proximal trunk muscles may be involved—the distal ones free. This occasionally happens in alcoholic neuritis as well. Reaction of degeneration appears in the paretic muscles. Anesthesia, atrophies, trophic disturbances, and contractures are met with, but may be considered exceptional. Oculomotor palsies also occur, and optic nerve atrophy is not infrequent. Other cranial nerves, those of the larynx, pharynx, and face are also implicated, though rarely. Lead excephalopathies somewhat resembling those of alcohol are known.[2]

Course and Treatment.—The course of lead polyneuritis is essentially chronic, lasting from several months to a year. The prognosis is usually favorable. The treatment is the same as for neuritis in general, with the addition of excessive diuresis, and the use of such remedies, as may hasten lead elimination, such as potassium iodid.

Arsenical Neuritis.—Dejerine,[3] in 1883, first insisted on the essential similarity of neuritis of alcoholic and arsenical origin, which view-

[1] Tedeschi: Sciatica from Lead, Riforma med., January 16, 1922.

[2] Lead Studies: Hunter and Aub: Quart. Jour. Med., 1927, **20**, 123. Reznikoff and Aub: Arch. Neurol. and Psychiat., 1927, **17**, 444. Schmidt: Recent Researches, Klin. Wchnschr., 1927, **6**, 367.

[3] Comptes rend., October, 1883, **98**, 17.

point has been amply verified in the extensive studies following a severe epidemic of arsenical polyneuritis in England, in 1899–1900.[1] The most frequent source of arsenical poisoning has been shown to come from impure glucose products; the sulphuric acid used to convert the starch containing arsenic. Other sources are wall-papers, certain manufactures (dyestuffs), artificial flowers, cosmetics, beauty pastes and powders, hair dyes, and arsenic used in medicines. The pathological alterations (a parenchymatous neuritis) differ but little from those seen in alcoholic neuritis.

Symptoms.—Acute polyneuritis from arsenical poisoning is rare. It sets in shortly after the gastro-intestinal symptoms have ceased.

In the chronic cases the general symptoms of chronic arsenic intoxication are first observed. These are the anorexias, congestions of the upper respiratory tract (nasal catarrh, cough) or more frank diarrheas of gastro-intestinal irritation. Cerebrospinal lymphocytosis may be present. The neuritis develops simultaneously with the symptoms of chronic intoxication.

As with alcoholic neuritis, sensory symptoms, paresthesiæ, hyperesthesiæ, numbness, shooting pains, sweating, develop first. A pigmented condition of the skin, most marked about normally pigmented areas, is found in the majority of the cases. The pigmentation may become very general and very dark, and is associated with herpetic, eczematous, or scaly, papular eruptions. Certain of the newer preparations of arsenic which have been extensively advocated for the treatment of syphilis, notably atoxyl and arsacetin, are reported to have caused optic nerve atrophy with blindness.

The signs of sore nerve trunks, Lasègue's phenomenon, loss of motor power in both extremities, are present in arsenical cases. Ataxias occur, and cases of arsenical neuritis have been confounded with tabes. The prognosis is usually good, but the blindness has been permanent.

Other Intoxications.[2]—Carbon monoxide, diabetes, and illuminating gas poisoning, if severe and not lethal, frequently develop a severe grade of multiple neuritis not differing in any marked degree from alcoholic polyneuritis. The gases found in natural gas, and in many artificial gases contain the same chemical radicals as alcohol, and the toxic action is identical. In the very severe cases, polioencephalitis develops with multiple softenings, not entirely confined to the thalamus or corpora striata.

Carbon bisulphide, which is extensively used in rubber industries, may give rise to a multiple neuritis. The toxic ion is not definitely known. Similar poisoning results from sulphonal and trional, two sulphuric acid-alcohol hypnotics. A number of the nitrobenzol series can produce localized or general neuritis.

Phosphorus, mercury, copper, and silver can produce poisoning with the development of multiple neuritis.

[1] Lancet, 1900, **1**, 1610. See Petrén: 1926, Arsenicisme; a psychical epidemic in Sweden.

[2] Industrial Intoxications, see A. Hamilton, De Lamar lecture, John's Hopkins, Bull., 1927.

Phosphorus metabolism defect (vitamines) seems to underlie the beriberi cases.[1]

Infectious Disease Types.—Mild or severe general neuritis has occasionally followed nearly every infectious disease.

Diphtheria.—Here the neuritis is rarely generalized, and the cranial nerves bear the chief brunt of the poison. In mild forms the soft palate and pharyngeal muscles are first or alone implicated. Oculomotor palsies are also frequent. Some degree of facial palsy is also met with. In the severe types the larynx, tongue and the pneumogastric innervations become invaded.[2]

Generalized forms occur, differing in no essential particular from the types already described.

Diphtheric palsies may come on soon in the disease or they may follow a month or six weeks after the subsidence of the disease proper. For those patients who develop polyneuritis the diagnosis is usually grave. The cranial nerve types are usually less severe, although occasionally one finds pneumogastric palsies which are fatal.

Influenza.—The toxins of the influenza organism seem to have a special attraction for sensory nerve structures. Neuralgias, localized neuritides, are extremely common, and polyneuritis not a rarity. The polyneuritis is of a parenchymatous type, is usually mild, quite irregular, and differs in no essential manner from other types described. Its course is rarely over a few months and the prognosis is good.

Polyneuritis of a mild parenchymatous type occurs as a sequel of typhoid fever, smallpox, erysipelas, pneumonia, pleurisy, acute articular rheumatism, parotitis, gonorrhea, dysentery, measles, Pasteur rabies treatment, whooping-cough, and puerperal septicemia. *Acrodynia* is a form occurring chiefly in infants of dietetic or infectious nature.[3] Trichiniasis is a rare but definite causative factor.

In chronic tuberculosis mild grades of neuritis are frequent, and severe polyneuritis is occasionally met with in the marantic type. Syphilis rarely causes a polyneuritis, but it is known.[4] Malaria is also a rare cause. Leprosy causes a specialized form.

PLEXUS PALSIES.

In peace times plexus palsies were rare. They occurred more often in the upper extremity. Brachial plexus palsy, as Erb's birth palsy, is the type. Lumbar plexus palsies rarely occur alone uncomplicated by cord lesions, since they are usually produced by compression, resulting from tumor, fracture, Pott's disease, etc. Sacral plexus palsies, however, are not infrequent. They make up the classical cauda equina lesions, arising from the pressure of a tumor, from hemor-

[1] Jelliffe: Jour. Nerv. and Ment. Dis., June, 1919.

[2] Archambault: Ciliary Ganglion Involvements, Jour. Nerv. and Ment. Dis., 1917. Walshe, F. R. M.: Brain, 1920, **43**, 74.

[3] Rodda: Am. Jour. Dis. Child., 1925, **30**, 224. Weston: South. Med. Jour., 1926, **19**, 655.

[4] Hoffmann, J.: Ueber syphilitische Polyneuritis, Neur. Ztbl., 1914, 1075.

rhage, fracture of the sacrum, bony disease of the lumbar vertebræ, pelvic tumor, abscess, etc. War injuries have caused a vast variety of complex plexus palsies.

Brachial Plexus Palsies.—These most frequently arise from pulling or wrenching of the arms from accidents. Dislocation of the shoulder-joint can cause a plexus injury. Cervical rib is a rare cause, aneurism of the subclavian also. Gunshot wounds occasion them. (See Fig. 228.)

The plexus is made up of the lower four cervical (fifth, sixth, seventh and eighth) and the upper thoracic roots. Extraspinal as well as intraspinal lesions go to make up the symptom picture. This picture is rarely complete, but as the fibers making up the plexus have a fairly definite arrangement so far as muscular distribution is concerned, a study of the muscles involved gives a key to the roots injured.

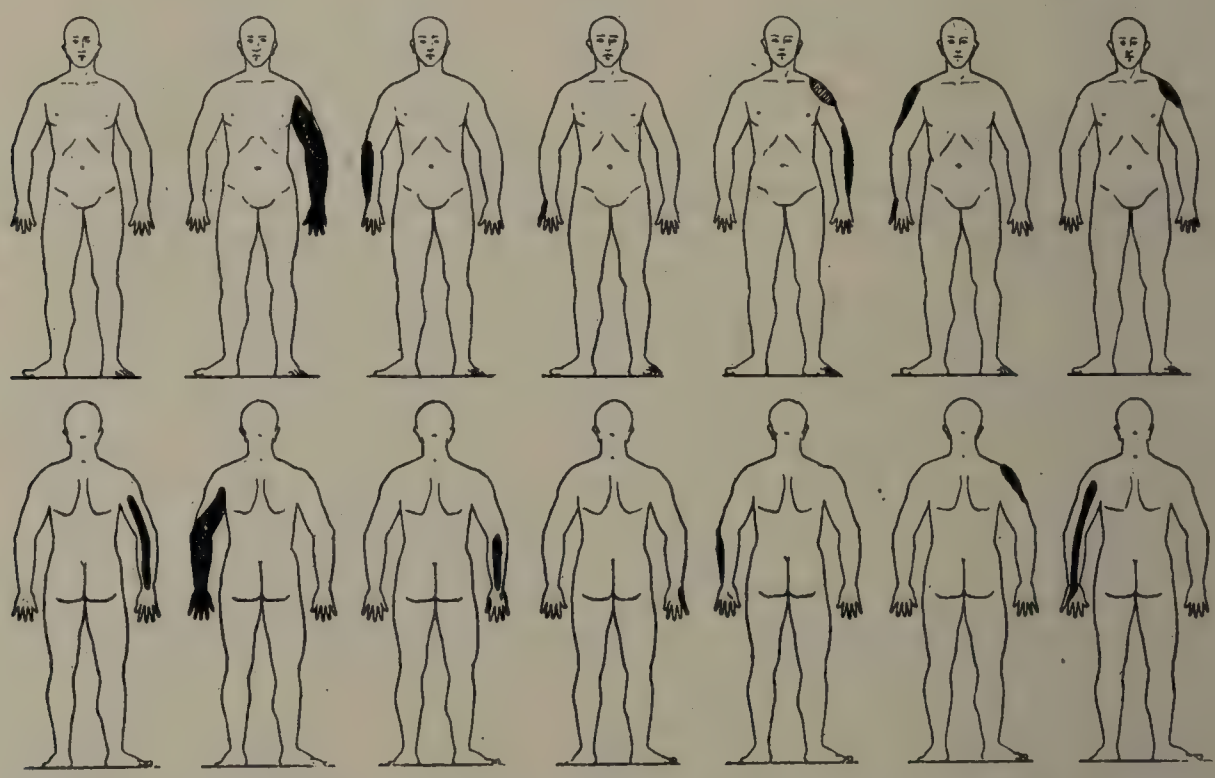


FIG. 228.—Sensory disturbances in seven cases of gunshot wounds of the brachial plexus in the "World War." (Edinger.)

The fifth and sixth cervical roots contain the fibers going to the deltoid, biceps, brachialis, brachioradialis, supraspinatus and infraspinatus, the clavicular fascia of the pectoralis major and the serratus anterior. The seventh cervical root contains the fibers distributed to the triceps, the sternal portion of the pectoralis major, to the dorsalis, to the extensors of the wrist and also some filaments to the median and ulnar nerves.

The eighth cervical and the first thoracic form the brachial, medial antibrachial cutaneous, ulnar, median and parts of the radial nerves with their muscular innervations, as seen in the illustrations. (See Figs. 22 to 30, also Fig. 218.)

The clinical pictures seen are those of a total brachial plexus palsy, a superior and an inferior type.



FIG. 229

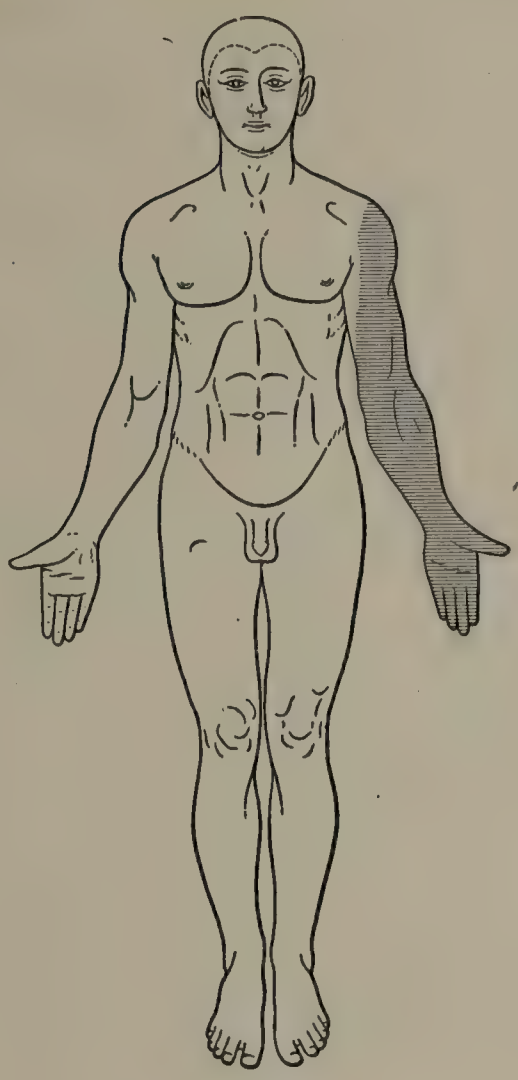


FIG. 230

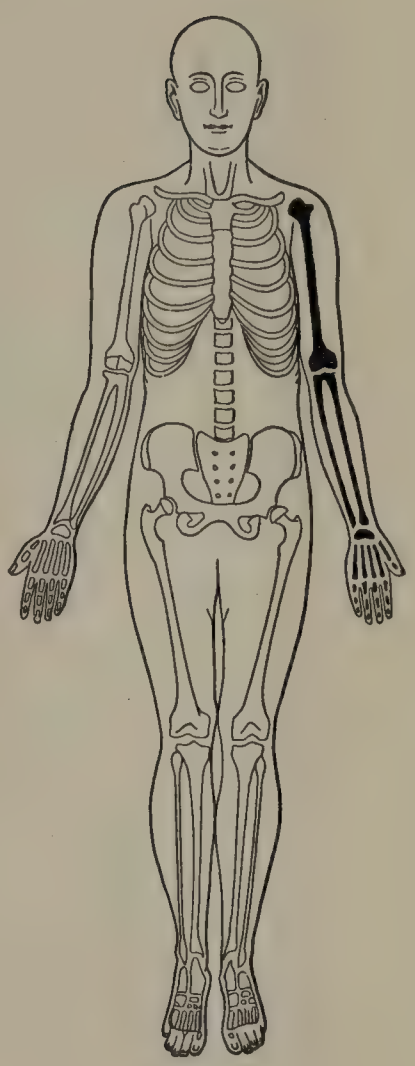


FIG. 231

FIGS. 229, 230 and 231.—Distribution of radicular sensory disturbances in case of traumatic brachial palsy of the left side. There was total paralysis, atrophy and loss of tendon reflexes, edema and cyanosis of left arm. Abolition of all types of sensibility of the distribution of the C_8 to T_1. Myosis and enophthalmus in left eye. (Dejerine-Klumpke.)



FIG. 232.—Erb's birth palsy. (Frauenthal.)

Total Brachial Palsy.—Here all of the muscles of the hand are paralyzed, those of the forearm, the arm and the shoulder. The arm hangs limp like a flail. In the early stages the skin is cyanosed, there are severe pains (in the accident cases), and a suppression of the secretion of sweat. Atrophy comes on quickly and is extreme, with loss of electrical responses. Trophic disturbances are usually present.

Sensory disturbances are present. There is loss of all forms of sensibility, including the sense of position in the hands and the forearm, excepting the upper part of the shoulder, Civ, and a triangular zone in the inner side of the arm Tii Tiii.

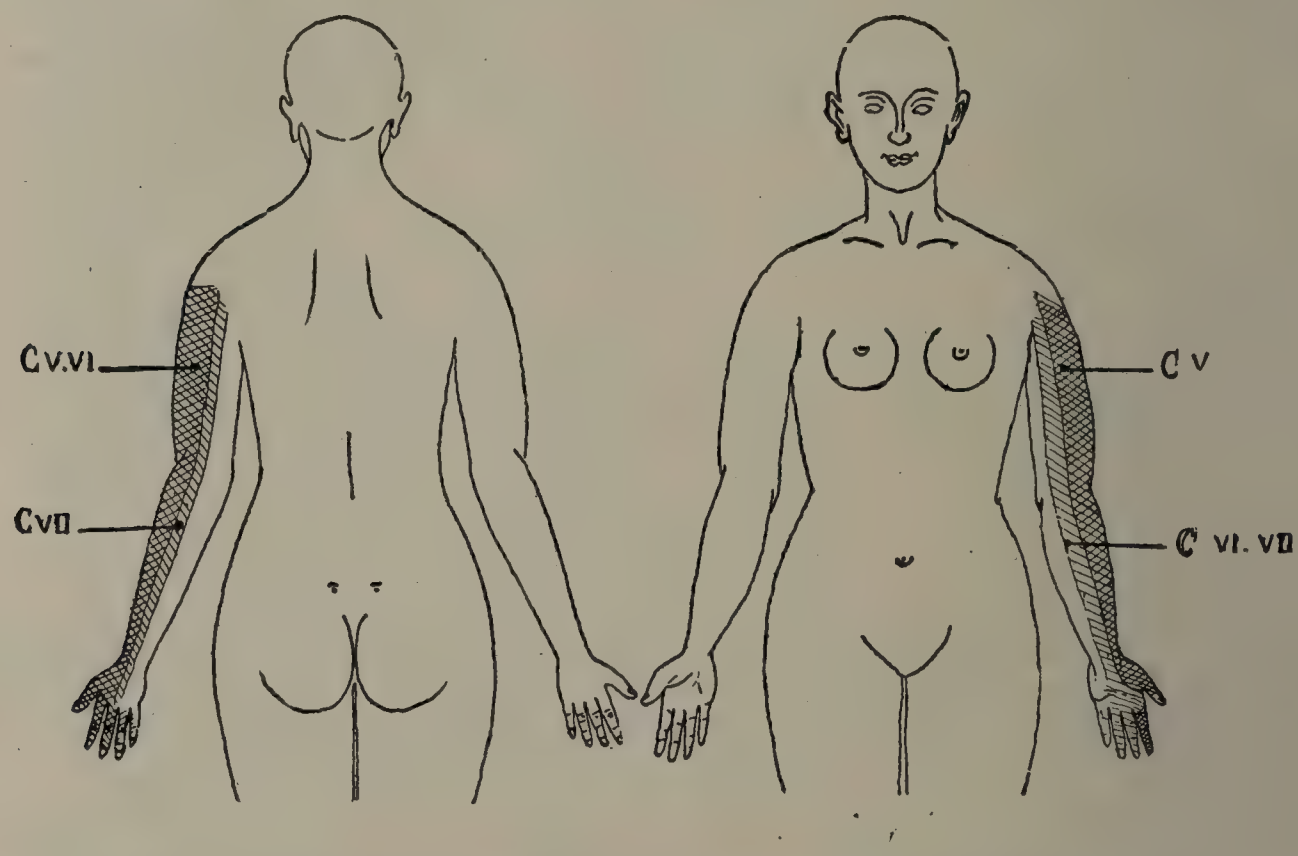


Fig. 233 Fig. 234

Figs. 233 and 234.—Topography of tactile, pain and thermal sensory disturbances in a superior brachial palsy type due to injury to the shoulder. The fifth, sixth and seventh cervical roots are involved. (Dejerine.)

Furthermore, since cervical sympathetic fibers are represented in the communicating branch of the first thoracic, one finds oculo-pupillary signs, first demonstrated by Madame Dejerine-Klumpke (1885). They consist of a contraction of the pupil on the paralyzed side, a diminution in the size of the palpebral fissure (pseudoptosis) and a retraction of the globe of the eye within the orbit (enophthalmus).

Furthermore, lesions here frequently cause changes in the sweat secretion. These are sharply delimited to the head, neck, face, to the level of the third rib in front and the spine of the scapula behind, including the upper extremity, all confined to the side injured.

Inferior Root Type.—This has also been designated the Klumpke palsy. It follows a less complete lesion of the plexus, involving the eighth cervical and the first thoracic. It may result from direct injury of strain, gunshot wound, birth palsy (Erb's palsy), from cervical

rib, or from syphilitic deposits. The small muscles of the hand are involved. The atrophy is rapid. There is edema of the skin, cyanosis, perhaps trophic changes in the nails. The anesthesia is less extensive, being limited to the distribution of the radial and medial antibrachial cutaneous nerves. The oculo-pupillary signs are evident.

Superior Brachial Plexus Palsy.—Here the upper branches, fifth and sixth cervical, are involved. Duchenne described the earlier cases minutely. The sensory disturbances are more extensive than those observed in the inferior type. The pupillary symptoms are absent. The chief muscles involved are the deltoid (axillary), biceps and brachialis (musculocutaneous) and brachioradialis (radial). Abduction of the arm is impossible. Paralysis of the clavicular head of the pectoralis major, supraspinati and infraspinati, subscapularis and teres major occurs. Lesions high up may implicate the serratus anterior, rhomboids and levator scapulæ. There results scapular atrophy, displacements of the shoulder blade (winged scapulæ) and loss of compensatory shoulder movements for the deltoid and serratus loss.

Mixed Types.—These are more frequent. In the great World War a vast medley of plexus palsies were observed.[1]

The more common form of so-called Erb's palsy belongs here. Many of these are bilateral, the two arms, however, being dissimilarly involved. The study of the muscles involved in the atrophy, the electrical changes, the sensory changes to light touch, heat, cold, pain, bony sensibility, and to position, the presence of trophic disturbances (protopathic system changes), and the oculopupillary phenomena determine the roots involved. (See Figs. 23 to 31.)

There are a number of bony conditions which can determine brachial plexus palsies. Tuberculous, syphilitic, carcinomatous, sarcomatous, arthritic infiltrations about the vertebral canal impinging upon the cords of the plexus can give rise to palsies of this type. Similar changes not infrequently also cause pictures which are often confused with brachial neuritis. Some of the severe arm pains with mild atrophies are forms of brachial radiculitis (*q. v.*), either inflammatory or traumatic in origin. In the course of a rheumatoid arthritis one not infrequently encounters these radicular disturbances which are undoubtedly referable to a vertebral arthritis.

Cervical Rib.[2]—Supernumerary ribs from any of the cervical vertebræ, but chiefly the seventh, cause many complex neuritides of a single nerve or of the entire plexus. The symptoms may be grouped as: (1) Local pain, tumor, pressure, bruit; (2) neuritic; (3) vascular, as pulsations, ischemia, gangrene, thrombosis; (4) muscular, as fatigue, atrophy, dysphagia, scoliosis.

[1] Tinel: Nerve Injuries, Wm. Wood & Co., 1917. Athanasi o Benisty, D. Appleton 1917, are two invaluable works on war injuries of the peripheral nerves. Also see Purves Stewart on Peripheral Nerve Injuries, 1919, 2d ed.

[2] Streissler, E.: Die Halsrippen. Ergeb. d. Chir. u. Orth., 1913, **5**, 280. Honeij: Cervical Ribs (with bibliography), Surg., Gynec. and Obst., May, 1920, p. 481. Sargent: Brain, 1927, vol. **44.**

The symptoms usually appear in early adult life and are due in most cases to pressure upon the lowest trunk or nerve entering into the plexus. In many cases they consist of wasting of the intrinsic muscles of the hand, most marked and starting in those of the thenar eminence; if of long standing the reaction of degeneration may supervene. In other cases the principal complaint is pain along the ulnar border of the forearm and hand, or a general weakness or heaviness of the whole limb noticed at the end of the day. Sensory changes vary. In some cases it is slight, in other they are severe, with trophic, vascular[1] and other alterations.

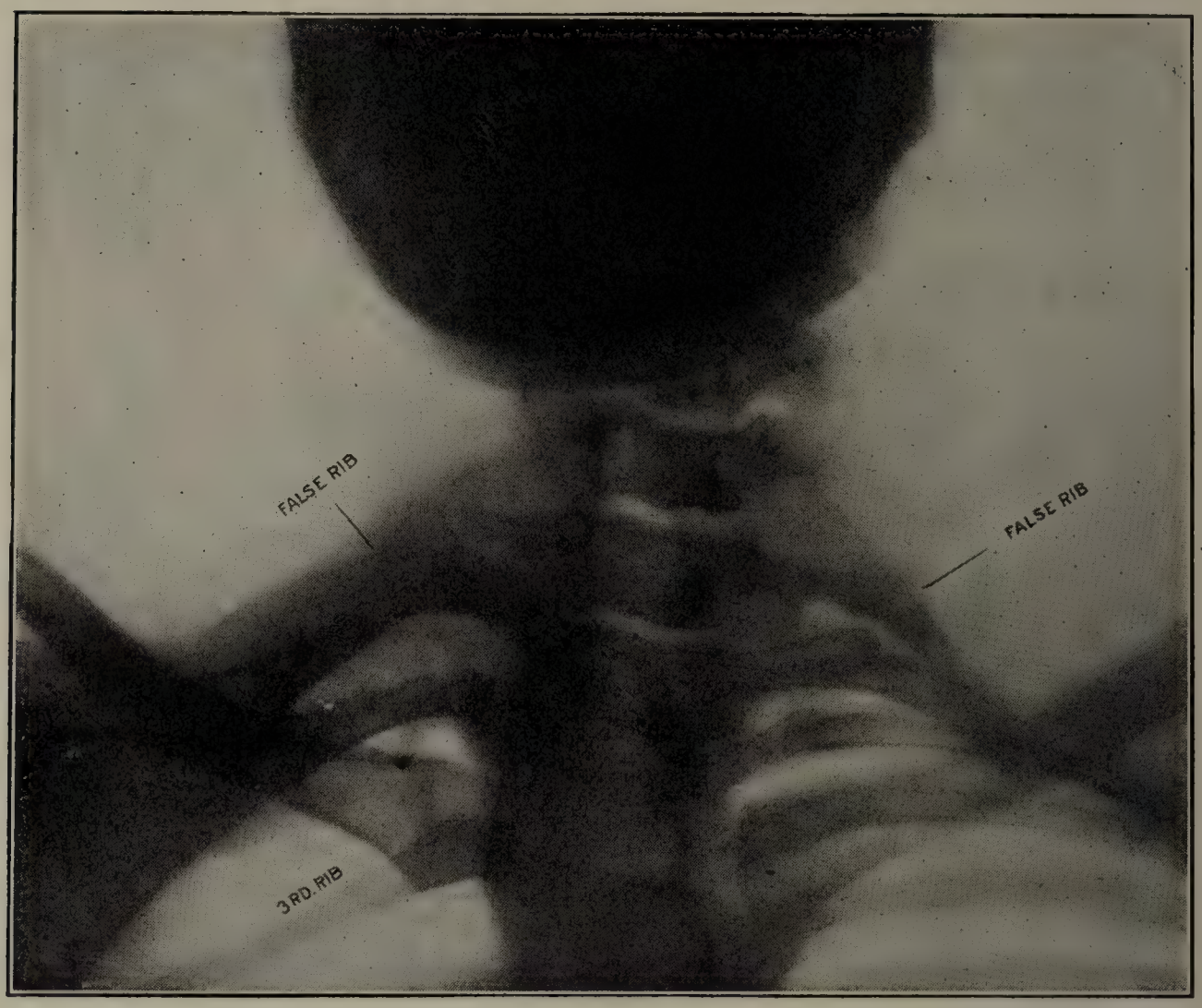


FIG. 235.—Double cervical rib, causing mixed type of brachial palsy. (Goodhart.)

This condition should be thought of in all cases of "brachial neuralgia," or of wasting of the thenar muscles. The ribs can usually be felt but occasionally can only be demonstrated by roentgen-ray examination.

Although a cervical rib be present it does not necessarily mean that it is the cause of the symptoms. Several cases have been observed in which a cervical rib was removed from a patient suffering from syringomyelia to which the symptoms were due. Thomas Murphy has recorded a case in which the symptoms were due to the pressure of the first rib. A similar case is reported by Sherren.

[1] For vascular alterations see Telford: Lancet, October 18, 1913. Todd: Jour. Anat. Physiol., 1911, 1912, 1913; Lancet, 1911, ii, 362.

Treatment consists in removal of the rib together with the periosteum covering it, followed by careful after-treatment.

Course.—General statements regarding the course are misleading. The majority of Erb's palsies due to obstetrical accidents recover, especially when only a few roots of the plexus are involved. Total separation from the cord, as in severe dislocations, falls, etc.; with complete plexus palsy, usually means an incurable affection, not remediable by surgery.

The underlying etiological factor determines the course in a number of others. Palsies caused by cervical rib, or subclavian aneurism do not get well spontaneously, nor does iodid help them. Proper surgery, as indicated by the roentgen-ray findings, may be of service.

Diagnosis.—The rarer nuclear and neuritic atrophies and dystrophies have been mistaken for these radicular palsies, but the study of the sensory changes should at once establish the differences. Spinal gliosis of the upper arm type will show dissociation symptoms, indicating the intraspinal nature of the lesion. Such a dissociation is a retention of tactile sensibility with loss of pain and thermal sensibility. Various neuritides, lead, diabetes, have a peripheral rather than a root distribution. Certain exceptional cases of tabes, with atrophy, can be separated, but may require a cerebrospinal fluid examination. Syphilitic cervical spondylitis cases at times show these symptoms.

Treatment.—There is little treatment for the severe, tearing lesions of the plexus. Whether they can be looped up with other roots to advantage has to be answered in the future.

The cause of the palsies, other than tearing, can often be removed. Thus surgical treatment of neck glands, subclavian aneurisms, cervical rib, spondylitis, and osteoarthritis is efficacious. The treatment of a cervico-dorsal Pott's by proper fixation, and the antisyphilitic treatment of a luetic spondylitis are satisfactory. Rare cases, often bilateral, due to profound secondary anemia, are often very refractory. True radicular neuritis is also stubborn.

The pains are best relieved by analgesics; antipyrin, aspirin, pyramidon, or other combinations are valuable. Heat is usually not well borne, and massage is contraindicated in the early stages. Osteopathic manipulation is a dangerous procedure in the early stages. In certain osteoarthritic cases it proves valuable later.

Simple counter-irritation over the site of the plexus, above the clavicle, is invaluable in many mild neuritic attacks; while for the severer attacks high-frequency currents, violent light therapy, Leduc current at times cause great relief.

Dietetic and general management in the neuritic cases is not to be overlooked. They need fats in ample proportion. This is best supplied through taking large quantities of milk.

Lumbo-sacral Syndromes. — The lumbo-sacral plexus constituents may be injured not only in their pelvic course, as is seen in the typical parturient syndrome, but as a result of war missiles, and particularly

in the great ravages of 1914–1918 many injuries to the roots in their long intraspinal course have been observed. (See Tinel, Athanassio-Benisty, Roussy et Lhermitte). Here in the cauda equina they form a long compact bundle which may be injured in a great variety of ways. Spinal fractures, bullet and shell splinters, hematomyelias cause injuries of the several roots and produce various syndromes, which do not have to be differentiated into trunk and root types. The peripheral nerves contain fibers from different roots, hence root lesions will give rise to a variety of dissociated peripheral palsies. The palsies are apt to be incomplete, unless the lesion is extensive, since the nerves usually are supplied from several roots. The sensory changes appear as radicular strips of anesthesia running along the axis of the limbs, when the sacral and lower lumbar roots are involved, the anesthesiæ are distributed obliquely from injuries of the upper lumbar roots and transversely from dorsal root lesions.

Lumbar Root Syndromes.—The first and second lumbar roots when injured produce slight lesions only as a rule. They supply few fibers to the quadratus lumborum, psoas, and the lower parts of the transversus and anterior muscles of the thigh. These muscles are only weakened. The sensory disturbances are found on the outer surface of the buttock and the base of the thigh spreading over the upper two-thirds of the inner aspect of the groin to the inguinal fold.

The fibers of the third, fourth and fifth lumbar when involved on the contrary produce widespread disturbances. Here all of the anterior and internal thigh muscles are implicated, also the triceps, pectineus and sartorius and if the obturator is caught in the injury the adductors and gracilis are paralyzed.

The sensory loss extends as an oblique band from the middle of the thigh above downward, internal surface to the middle of the leg and down the inner border to and including the big toe. The patellar reflex is lost. There is marked atrophy of the thigh which seems shrunken in just above the knee as if squeezed. A number of muscles are involved which may be studied by referring to plates. Pp. 96 to 102; Figs. 27 to 32, also see Figs. 253, 254, 255, 256, 257, 258.

Sacral Root Syndromes.—The sciatic distribution corresponds to the fibers of the first and second sacral roots, excepting the tibialis anticus which is supplied from the fourth and fifth lumbar, which also supplies a number of the muscles of the leg and the foot, but in the leg the gastrocnemius, outer head, and peronei are supplied by the sacral fibers and in the foot the adductor hallucis and the interossei are exclusively sacral distributions. Extremely complicated pictures therefore result from the cauda lesions which have to be worked out on the basis of the charts (Figs. 236 and 237, 253, 254, 255, 256, 257, 258; Figs. 27, 28, 29, 30; Figs. 31, 32. The sensory distributions of the first and second sacral roots occupy a wide tract from the buttock to the outer posterior aspect of the knee, where it takes a further oblique course to the front of the leg and dorsum of the foot. The Achilles reflex is lost. If the fourth or fifth sacral roots are included

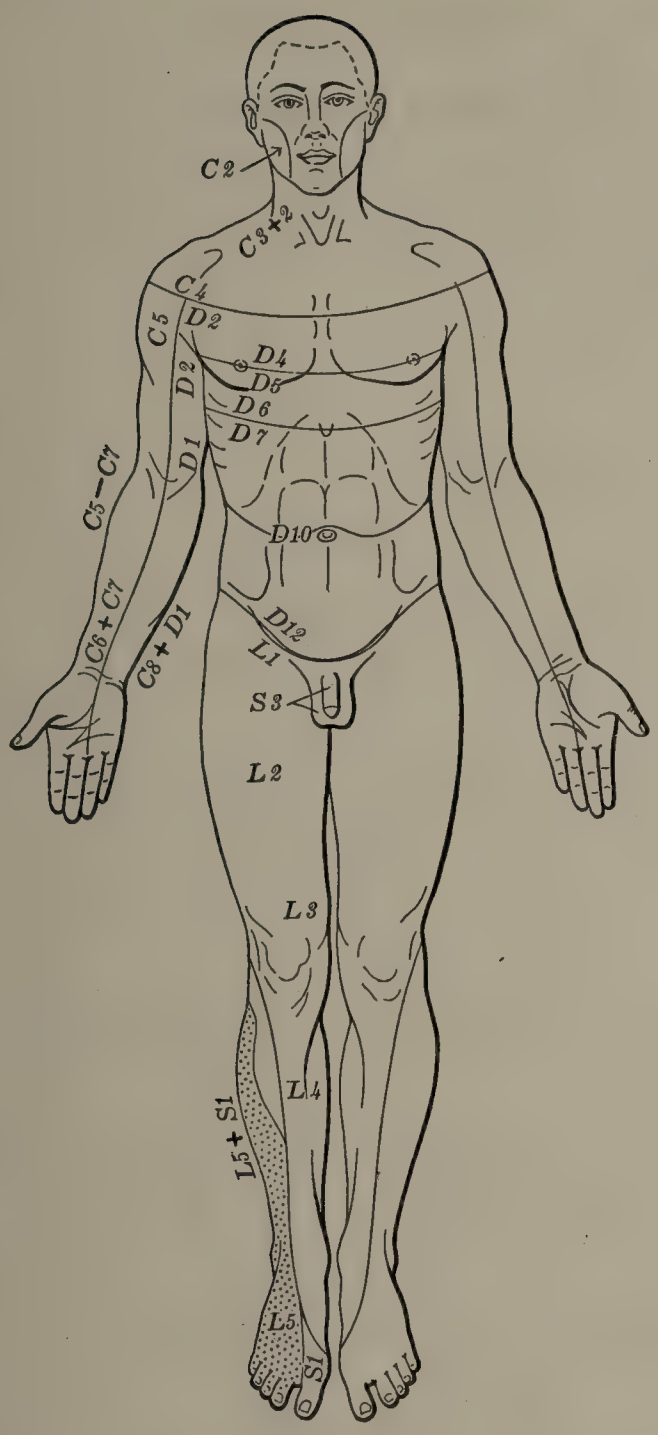


FIG. 236

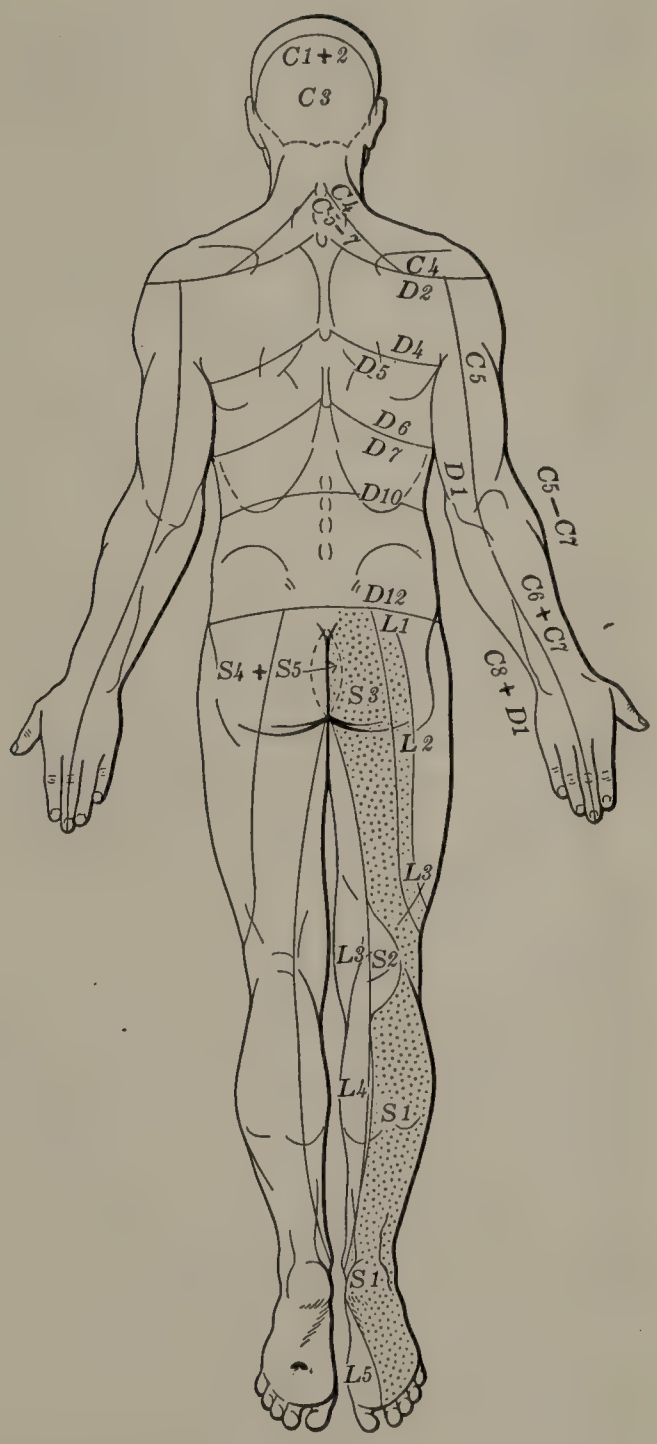


FIG. 237

FIGS. 236 and 237.—Distribution of anesthesia in sacral roots, sciatic chiefly.

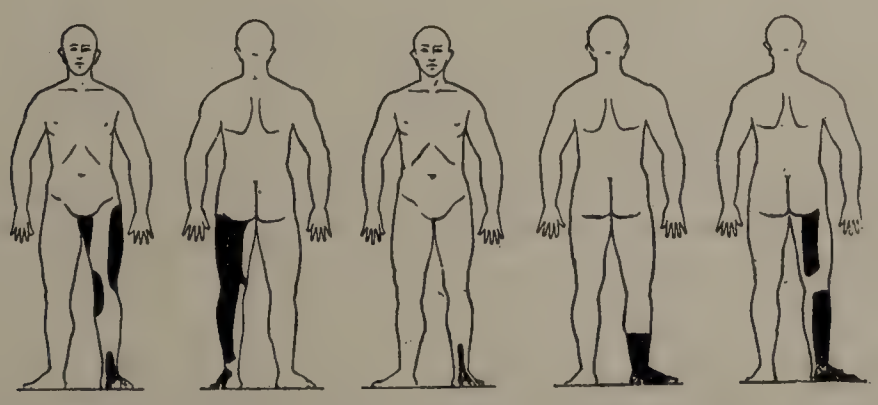


FIG. 238.—Sensory disturbances in five cases of sacral plexus injury due to gunshot wounds. In the first case there was a total palsy in the peroneus and tibialis nerve distribution; in the second the peroneus alone was involved; in the third the peroneus, tibialis, ilio-inguinalis and obturator; in the fourth the peroneus and tibialis; in the fifth the crural nerve distribution alone suffered. (Edinger.) Compare Figs. 253, 254, 255.

or alone injured, incontinence of urine and feces, with failure to feel the same, are added as isolated symptoms.

Pains are usually very marked and persistent. They occur in paroxysms, and are usually extreme. The chief path is that of the sciatic, but they may be located almost anywhere about the thigh. They may be bilateral or unilateral and shift considerably.

Treatment.—This must vary largely upon the type of lesion.

PERIPHERAL PALSIES.

Numerous forms of peripheral nerve palsy are found, varying according to the particular fibers involved. The branches that arise from the brachial plexus have already been described. Any of these may be injured or inflamed with a resulting partial or complete palsy. The various neuritides will not be reëxamined and the following sections will deal with those peripheral palsies largely due to defect or injury.

Defects, Congenital and Acquired.—Muscular defects in the upper and lower extremities are by no means uncommon. They have been described for some centuries. Their precise formulation began with Ziemmsen's work in 1857. The later literature is summed up by Bing,[1] Lorenz,[2] and by Hirschfeld.[3] The causes for these muscular defects are extremely complicated.

Their occurrence is very manifold. Bing was the first to collect the entire group, and Abromeit, in 1909, completed the study which shows that an absence of any muscle of the body may be expected. One in 10,000 shows such defects.

In the majority of the cases the defect is unilateral, and they are more frequent in men.

Abromeit collected reports of 186 cases of defect of the pectoral muscles, the sternocostal portion being most affected.[4] This seems the most frequent of such anomalies. The trapezius was absent in 33 cases, the serratus magnus in 22, quadratus 26, omohyoid 16, semimembranosis 7, rectus muscles 11, pyramidalis, often found absent postmortem, diaphragm 3 times, deltoid 5, sternocleidomastoid 8, etc. The smaller muscles of the hand are not infrequently absent.

Certain combinations of muscles may be absent, constituting a group complex. Atrophy or loss of other parts may be combined and gross anomalies of structure may be combined with marked muscular defects; various monsters, syndactylia, phocomelus, etc. The hereditary nature of syndactylous anomalies has given occasion for some important studies.

The symptoms involve the physiological loss of the special muscle function or the resultant of function from the muscles present in a combination.

[1] Virchow's Arch., 1902, p. 170. [2] Die Muskel Erkrankungen, Nothnagel, 1904.
[3] Kongenitale Muskeldefekte, Lewandowsky's Handbuch, 1911.
[4] See Lewandowsky, vol. **2**, 253.

These patients rarely show as marked loss of efficiency, especially with one-sided defect, as do those who acquire a defect. Acquired defects are usually bilateral. They usually involve a group of muscles; the disease rarely causes a total loss of muscle substance, and anomalies of accompanying structures are missing.

Fibrillary twitchings are often diagnostic of the acquired muscular defects.

The prognosis and treatment require little discussion. Gymnastic exercise of the residual muscular combinations directed to the acquisition of greater efficiency by skilful application of mechanical principles is always an individual goal that cannot be more than mentioned here.

Peripheral Palsies Due to War Injuries.—This section discusses peripheral palsies due to nerve injuries rather than to those due to general neuritic processes. An injury may affect a nerve either in its continuity, or in one of its terminal branches, thus giving rise to different symptoms, and requiring a diverse therapy.

Injury in *continuity* may result from penetrating wounds, traction, pressure, blows or by operation. They may give rise to complete or incomplete division.

Symptoms.—The World War has amplified the existing amount of information relative to peripheral nerve injuries. The large mass of observations cannot even be summarized here. A score of monographs filled with statistical, clinical, pathological and surgical data have appeared (1915–1922) some of which are referred to in the footnotes here.[1] These are but forerunners of hosts which have and will come from the laboratories and clinical records of the war. Tinel in the first of the important monographic researches of the war summarizes the statistical incidence of the main nerve injuries as occurring among the French soldiers. In a series of 639 cases the relative frequency of involvement was as follows: *Upper limb:* 408, distributed as musculospiral 146; ulnar 84; median 67; musculocutaneous 11; circumflex 25; brachial plexus 27; combined lesions 48. *Lower limb:* 231; anterior crural 11; long saphenous 14; sciatic (trunk) 78; internal popliteal 29, external popliteal 37; posterior tibial 18; musculocutaneous 7; anterior tibial 4; short saphenous 9; obturator 1; external cutaneous 3; ilioinguinal 1; lumbosacral plexus 19. Burrows' for English army injuries in a series of 1000 cases, showed the following relative frequency: Ulnar 527; median 242; musculospiral 204; radial 20; posterior interosseus 19; musculocutaneous 24; internal cutaneous 26; brachial plexus, root 17; outer cord 17; inner cord 19; posterior cord 24; cervical roots 12; circumflex 2; facial 2; spinal accessory 2. *Lower limbs:* sciatic 121; external popliteal 97; internal

[1] Tinel, l. c; Athanassio Benisty, l. c.; Frazier, l.c.; Elsberg, l. c.; Revue Neurologique; Neurologie de Guerre; Review of Neurology and Psychiatry; War Neurology; Burrow: 1000 Cases of Peripheral Nerve Injury. Röper: Study of 1200 Cases. Deutsch. Ztschr. f. Nervenh., 1921, **68, 69,** 409. Stopford: Study of 2000 cases, Lancet, October 5, 1918. Dustin: Ambulance de l'Océan, December, 1917, July, 1918. Important for Study of Fasciculation of Nerve Fibers. Stookey, Philadelphia, 1922.

popliteal 8; posterior tibial 8; anterior tibial 9; musculocutaneous 2; external saphenous 1; internal saphenous 2; miscellany 29.

In view of the investigations of Head and Sherren as carried into the field of war neurology by Dejerine and his pupils, it seems possible to separate the peripheral nerve injury syndromes into four: (1) The interruption syndrome; (2) Compression syndrome; (3) Irritation syndrome; and (4) Restoration syndrome.

(1) The *interruption syndrome*, when complete, causes abolition of all of the functions. Paralysis of the involved muscles is complete, tonus is lost, the limbs assume attitudes caused by the antagonist predominance. There is no pain on pressure of the muscular masses and there is muscle anesthesia. To these may be added: Abolition of the tendon, osseous and cutaneous reflexes, exaggeration of the myotatic reaction, amyotrophies, deformities, as dorsal tumor of the tarsus or carpus, modified electrical reactions. The sensory changes need some elaboration and the principles involved applied to the other syndromes to be described later. In complete division Sherren and Head have shown that the sensory peripheral fibers may be divided into three systems:

1. Those which subserve deep sensibility and conduct the impulses produced by pressure. The fibers of this system run mainly with the motor nerves and are not destroyed by division of all the sensory nerves of the skin.

In a part innervated only by this system gradual increase in pressure can be recognized and pain experienced when such pressure becomes excessive. The patient can also appreciate the extent and direction of movements produced passively in all the joints within the affected area.

2. Those which subserve protopathic sensibility. This system of fibers and end-organs responds to painful cutaneous stimuli and to the extremes of heat and cold; it also endows the hairs with power to react the painful stimuli. The distribution of the protopathic fibers usually overlaps greatly the area supplied by similar fibers from adjacent nerves. Head's protopathic fiber system undoubtedly contains much of the vegetative fibers innervating the skin muscles, vessels and secretory organs.

3. Those which subserve epicritic sensibility. The nerve fibers and end-organs of this system endow the part with the power of responding to light touch with a well-localized sensation. The existence of this system enables one to discriminate two points and to appreciate the difference between cold and heat. The distribution of these fibers in large peripheral nerves, such as the median and ulnar, has very little overlap compared with the greater overlapping of the protopathic supply.

These investigations were carried further, particularly with regard to deep sensibility and the distribution of heat and cold spots, by Head and Rivers after voluntary section of the radial and external cutaneous nerves in the former's arm.[1]

[1] Compare Boring: Quart. Jour. Exp. Physiol., 1916, **10**, 1.

To illustrate these changes in sensibility after division of a mixed nerve the ulnar is an excellent example (Fig. 240). After complete division of this nerve at the wrist, if no tendons have been divided at the same time, the patient is able to appreciate those stimuli commonly called tactile. A touch with anything which deforms the skin may be readily appreciated and correctly localized. When pricked with a pin the patient knows that he has been touched but fails to perceive the sharpness of the stimulus (deep sensibility). But if tendons are divided at the same time or the section involves the nerve above the point at which its muscular branches are given off, deep touch may be unperceived. These characteristics are of the utmost importance, many cases of nerve injury have been overlooked from failure to recognize these facts.



FIG. 239.—To illustrate the changes in sensibility met with after complete division of a peripheral nerve. The area inclosed by a line is that in which epicritic sensibility is lost. The shaded area is that of loss of epicritic and protopathic sensation. The unshaded portion is the "intermediate zone." (Sherren.)

The point of a pin and all temperatures are unappreciated within an area which varies somewhat in each case (loss of protopathic sensibility). Surrounding this area and corresponding closely to the distribution of the nerve as figured in Figs. 239 and 240 is a territory within which the patient is unable to appreciate light touches with cotton-wool and temperatures between about 22° and 40° C. (minor degrees of temperature), and fails to discriminate the points of a pair of compasses when separated to many times the distance necessary over the corresponding portion of the sound limb or the unaffected portion of the injured one (loss of epicritic sensibility). Within this area of loss of sensibility to light touch, to which they gave the name of "intermediate zone" the patient is able to appreciate the sharpness

of a pin-prick and to differentiate temperatures below 20° C. and above 45° C., naming them correctly.

Division of a peripheral nerve produces a well-defined loss of epicritic sensibility, a smaller loss of protopathic sensibility with, as a rule, ill-defined limits. In many cases there is no loss of deep sensibility.

Complete division of certain nerve branches produces no objective change in sensibility, these are the musculospiral below the point at which its external cutaneous branches are given off, the radial, and certain cervical anterior primary divisions.

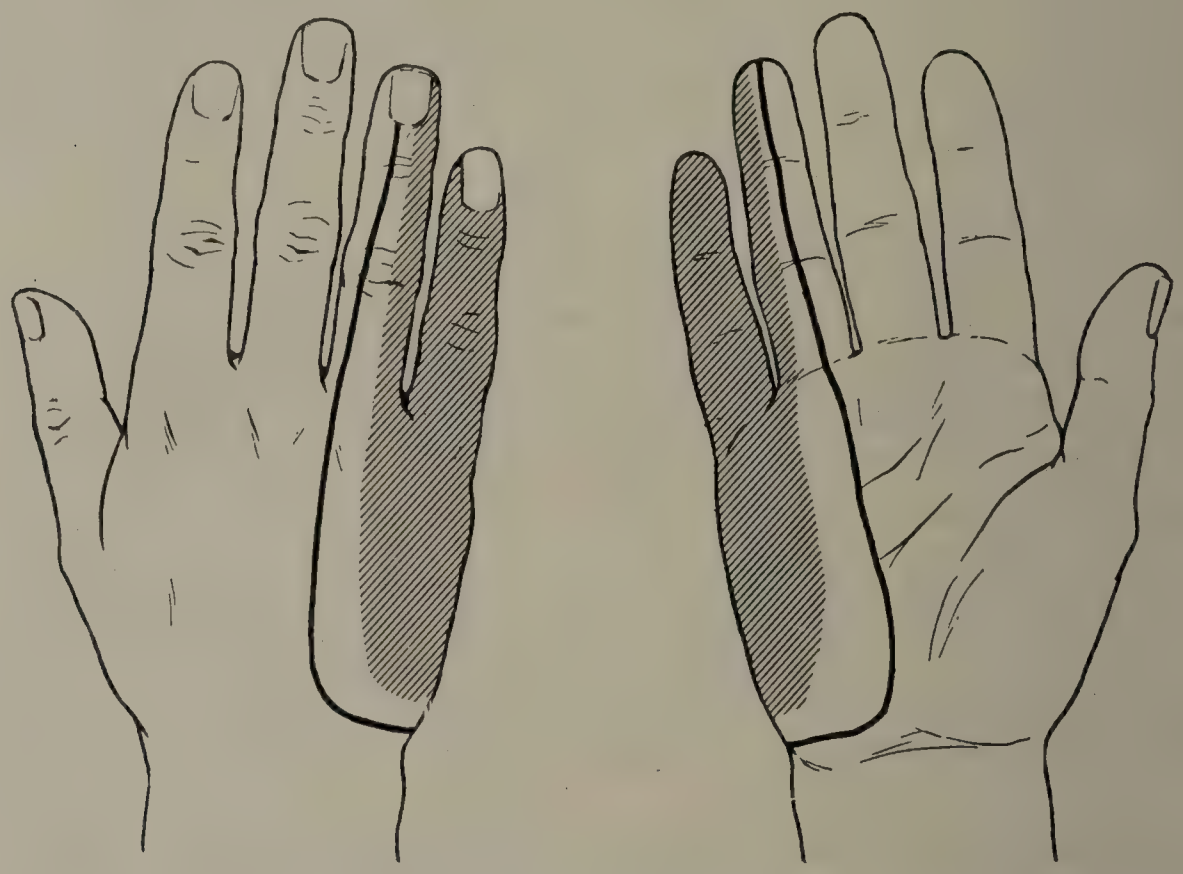


FIG. 240.—Loss of sensibility after complete division of ulnar nerve. (Sherren.)

In incomplete division the sensory symptoms may be *nil*. The patient is conscious of an area altered in sensibility, and it is often possible to demonstrate this by the changed sensibility produced at its borders when a piece of cotton-wool or the point of a needle is dragged lightly across the skin from sound to affected portions (line of change). If the area of changed sensibility is well marked, response to the compass test will be defective.

In cases of greater severity the loss of sensibility to cotton-wool may be absolute, with borders as well defined as after complete division.

When the injury is more severe, impairment or loss of protopathic sensibility results and the sensory loss may resemble exactly that seen after complete division.

Sherren has shown that, contrary to the usual teaching, the motor loss in incomplete division is not more than the sensory loss. Paralysis of muscles of the injured nerve distribution may result after eight to fourteen days. The usual reaction on or about the tenth day is that the muscles do not respond to the interrupted current but do react

to the constant current. The strength of the current needed to cause a contraction is less on the sound side; the contraction is brisk in comparison to the R. D. and polar reversal is absent.

Pain is a frequent after-result in incomplete division; there may also be tender and glossy skin and changes in the nails.

Stages of Recovery.—Following complete division, in from six to sixteen weeks, the restoration of protopathic sensibility commences and is completed in from four to twelve months after suture of divided nerves. Blisters may occur early, but later all ulcers heal; blisters no longer appear. Epicritic touch is unchanged throughout, but in from twelve to fifteen months the whole area is sensitive to light touch and intermediate degrees of temperature. Improvement in the power of accurate localization is the third stage, and is tested out by the compass. The motor recovery is gradual, the electrical reactions of incomplete division first appearing.

It seems certain that no regeneration takes place in the peripheral end of a divided nerve without union with the central nervous system.

After incomplete division of a mixed nerve the loss of sensation and motion may at first resemble that which follows complete division, but the method of recovery is entirely different.

After complete division of a nerve and suture, sensibility to prick is restored before the commencement of recovery of sensibility to light touch. Complete sensory recovery often occupies several years.

But after incomplete division sensibility to light touch and to prick are restored together and, unless nerve fibers have been anatomically divided in considerable number, the power of appreciating two points (the compass test) is soon regained. This is an extremely important point, for upon the recovery of this power of localizing depends the utility of the part for fine work. It is important to recognize that in injuries of nerves without interruption of their anatomical continuity the power of localization returns quickly, unless the injury has been sufficient to cause complete division; in this case the usual three stages are present, but the time of the third stage is much shortened.

Knowledge of this method of sensory recovery, first described by Head and Sherren, is a valuable addition to our powers of diagnosis. If both forms of sensibility are recovering together, it is certain that the injury has not been severe enough to produce complete interruption of conduction in the injured nerve, with degeneration of the whole peripheral end.

Motor recovery after incomplete division follows the same march as after complete division; the muscles nearest the seat of the injury first regain voluntary power and excitability to the interrupted current.

In the cases in which the reactions typical of incomplete division are present, voluntary power usually returns before the reëstablishment of excitability to the interrupted current.

Sensory recovery usually begins in about three weeks, and is complete in about six months; motor recovery in from a few days to ten weeks.

These times of motor and sensory recovery are approximate only, and vary with the severity of the injury and its distance from the periphery. When epicritic sensibility alone is lost recovery is much more rapid than when both forms of sensibility are affected. When the injury affects the brachial plexus, considerably longer time is necessary for the commencement and progress of recovery.

To sum up: After incomplete division of a mixed nerve, both forms of sensibility (epicritic and protopathic), if lost, return at the same time, commencing at a date which varies with the distance of the injury from the periphery from about three weeks at the wrist to six months in the plexus, and also with the degree of the injury. Complete recovery, as a rule, rapidly ensues. Muscular recovery commences at a time which varies in the same way. In cases in which the muscles,



FIG. 241.—End of first stage of recovery. (Sherren.)

though paralyzed, retain their irritability to the interrupted current, recovery commences in three or four weeks, sometimes earlier, and soon becomes perfect. This degree of injury is seen most often as the result of compression of the musculospiral nerve, producing sleep, anesthetic or crutch paralysis. If the reactions typical of incomplete division are present a much longer time is necessary.

After neurolysis, or when the nerve has been relieved from any form of pressure, recovery follows exactly the same lines.

Syndrome of Compression.—There is tenderness of the muscles and of the nerve trunks, and in general many of the signs of complete interruption. There is usually, however, retention of the muscle tonus, the reaction of degeneration is less pronounced, slight resistance movements may be elicited, deep sensibility is less involved. The causative lesions

are about the nerve, splinters, callus, fibroids, bits of cloth, aneurisms, abscesses, hematomata, etc.

Surgical intervention is called for to free the nerve and remove the object pressing upon it. After this the nerve is isolated and placed amid healthy tissue, preferably muscle planes which prevent further pressure. When there is interstitial sclerosis it is useless to plough it up blindfold (Dejerine). Indeed, it is better to leave it alone entirely. In treating sclerosis the greatest nicety of operative technic is required.

Syndrome of Irritation.—Dejerine describes this as characterized by the absence of complete paralysis, absence of the dysesthetic disturbances seen in complete section, already described, persistent hyperesthesia, continuous painful phenomena and trophic disturbances.

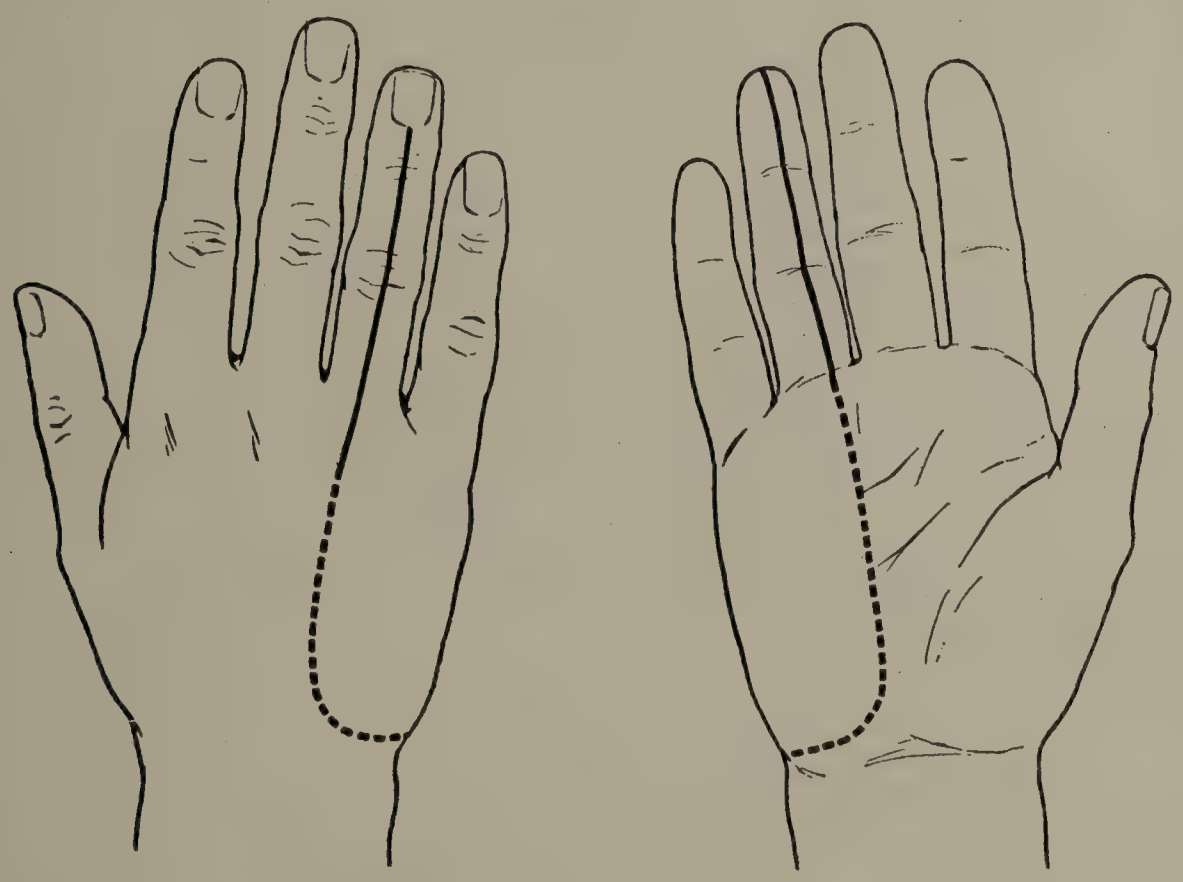


FIG. 242.—Commencement of second stage of recovery. The dotted line marks the area regaining sensibility to cotton-wool. (Sherren.)

These latter may be muscular, osseous, articular or cutaneous (hypertrichosis, etc.). In grave irritation the pains are extremely severe (Causalgia of Weir Mitchell). The slightest motion is painful and wearing. Local applications of water, moist cloths are very grateful. Sometimes the irritant is very trifling, bits of bone, fragments or splinters of foreign bodies hardly touching the nerves. These cases tend to spontaneous recovery.

Syndrome of Restoration.—Sensory symptoms soon appear. These are spontaneous stabbing, darting pains along the injured nerve. These continue from eight to fifteen days like electric shocks and tend to subside later. There is pain on pressure of the nerve trunks. Shrinking of the dysesthetic areas (see Figs. 239–244). Slow return of muscular tissue, of muscle tonus, painful muscles disappear and a

return of voluntary contraction takes place. The electrical reactions remain as before.

Signs of restoration contraindicate operation, but if it be seen that the motor loss continues after the sensory restoration, operation is advisable.

Treatment.—The indications in cases of complete division in all firearm injuries in which symptoms of repair do not occur is to operate whatever may be the lesion. The surgeon should freely resect all the indurated tissues about the nerve or in its course (fibroma, neurofibroma, keloids), and suture the upper and lower segments end to end. In large gaps a graft may be taken from a functionally less useful sensory nerve. If collateral motor branches are cut during the operation they should be sutured at once. One should insist on the great tenderness of nerve structures and insist on great gentleness in manipulation on the part of the surgeon.

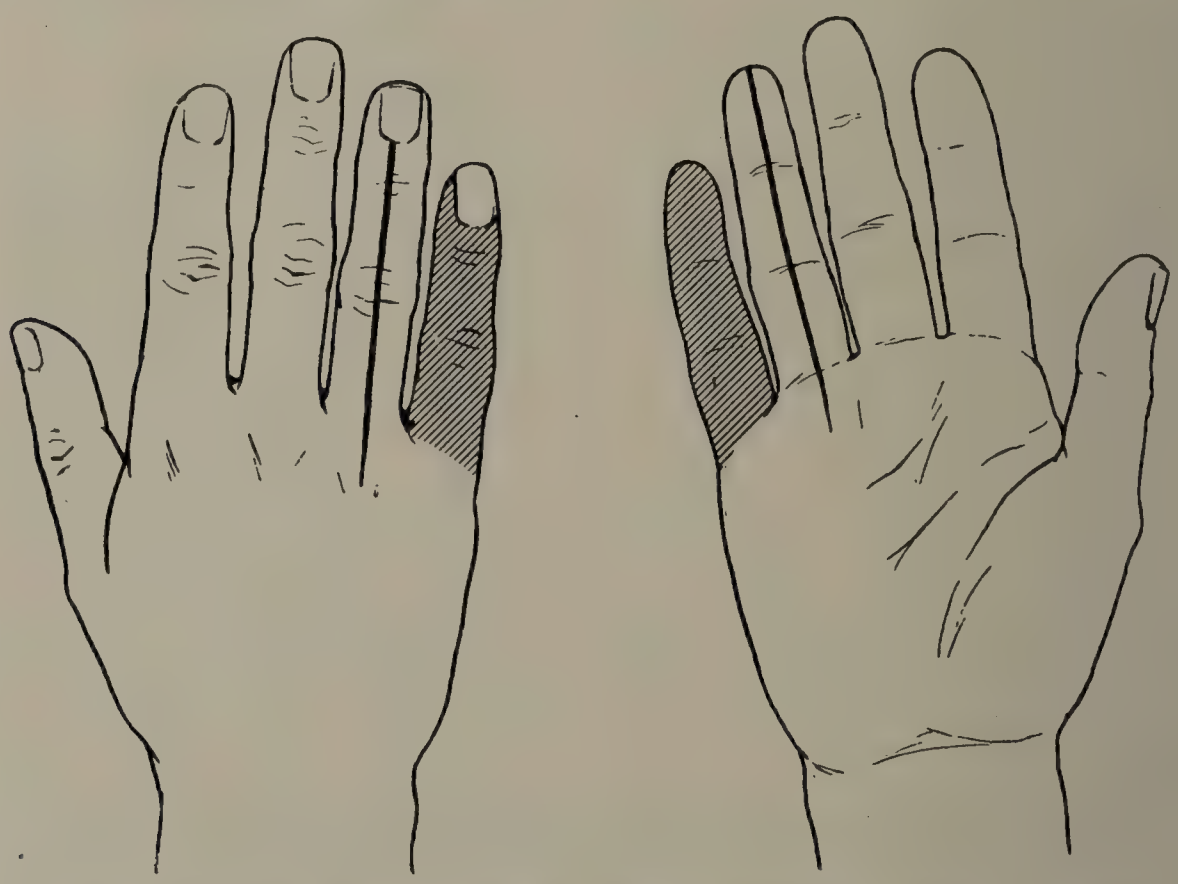


FIG. 243.—Showing method of recovery after incomplete division. (Sherren.)

The general trend in recent war surgery of the nerves is to greater and greater conservatism in operations and to the greater consideration given to the study of the fasciculation in nerve trunks, with careful end-to-end anastomoses of the divided fasciculi, cut well away from neuromatous stumps. For complete discussion of these factors the student is referred to the works of Dustin and others referred to.

Tinel[1] has described a sign which he considers of value in determining the advisability of operation on peripheral nerves, particularly of the extremities. The value of this is affirmed by certain observers (Burrow, l. c.) denied by others (Elsberg[2]). Its final evaluation remains to be deter-

[1] Presse méd., October 7, 1915, p. 588.
[2] Transactions Am. Neurol. Assn., Jour. Nerv. and Ment. Dis., 1919.

mined. If a wounded nerve be gently percussed, or tapped, or gently rolled under the finger, distal to the site of injury, a tingling paresthesia is apt to develop over its surface distribution area. As regeneration along a nerve trunk takes place the sign is elicitable only further and further distally in its course. Thus in early ulnar nerve injury, percussion of the ulnar at the elbow brings out the tingling; a month later if recovery is taking place, pressure on the trunk in the forearm may start the "pins and needles" sensation; still later, pressure at the wrist may elicit the sign. The test can be made quickly and hence is an advantage in busy hospital practice. The general principle is to delay nerve suture, so long as the tingle may be brought out by pressure at progressively more and more distal points.

Injuries to Facial Nerve.—Facial paralysis may result from injury to the nerve (*a*) above the geniculate ganglion, (*b*) between the ganglion and the point at which the chorda tympani is given off, and (*c*) below this point. When injured at (*b*) taste is affected over the anterior two-thirds of the corresponding side of the tongue.

Apart from the so-called "rheumatic" affections of the nerve (Bell's palsy), interference with the function of the nerve in the middle ear as a result of disease or operation is the usual cause. The nerve may suffer in fractures of the skull primarily, or more often from involvement in callus. Outside the skull it may be injured during operations in the parotid region or in the removal of tuberculous glands, or from forceps pressure during childbirth; in most of these cases the "division" is incomplete and spontaneous recovery ensues.

In cases of incomplete division the usual treatment directed toward maintaining the nutrition of the paralyzed muscles must be adopted. When due to middle-ear disease it is an indication for the complete mastoid operation.

When the reaction of degeneration is present, showing that complete division, either anatomical or physiological, has occurred, the treatment to be adopted depends on the cause. If it follows a radical mastoid operation, the sooner operation is carried out after the wound has healed the better; in cases of Bell's palsy it is justifiable to wait for six months. If the nerve is divided during the course of a mastoid operation, the ends should be adjusted in the aqueduct, when restoration of function may be expected unless neuritis is set up as the result of sepsis. If discovered after the operation the electrical reactions should be tested at the end of a fortnight; if the reaction of degeneration is present, the wound should be opened up and an attempt made to adjust the ends. If this fails, nerve anastomosis must be undertaken. It must be remembered that the injury during mastoid operation is in most cases incomplete, and that spontaneous recovery follows the usual non-operative treatment.

Spinal Accessory Nerve.—The external or spinal portion of this nerve is not infrequently divided during the course of operations upon the neck, particularly during the removal of tuberculous glands. In many of these cases the branches of the cervical nerves to the trapezius

are affected at the same time, producing its complete paralysis. The extent of supply of the spinal accessory and the cervical nerves to the trapezius varies; as a rule the upper portion of the trapezius is paralyzed by division of the spinal accessory alone.

The Long Thoracic Nerve (*Nerve of Bell*).—This nerve is most often injured in males between the ages of twenty-five and forty, usually on the right side. The injury is generally caused by prolonged pressure in the supraclavicular region, it occasionally follows violent muscular efforts and direct violence applied to the shoulder.

Paralysis of the serratus magnus rarely occurs alone; it is usually combined with paralysis of the lower trapezius.

The Circumflex Nerve.—Injury to the circumflex nerve is by no means so common as is usually supposed. It has been said to follow direct blows on the point of the shoulder, but in most cases the injury is to the anterior primary division of the fifth cervical nerve, and careful examination will show that the spinati also are affected. In other cases the wasting of the deltoid in common with the muscles around the joint on which the circumflex injury was diagnosed has been found to be the result of a traumatic arthritis.

Careful examination is necessary before coming to a decision with regard to treatment; testing must be carried out for all forms of sensibility. If there is no loss of sensibility, and there is paralysis of the deltoid with the reaction of degeneration, it is extremely improbable that the circumflex nerve is injured. If the signs are those of complete section of the nerve, the age of the patient and his occupation must be taken into consideration; in some cases operation can be avoided by training the neighboring muscles to take the place of the deltoid.

Ulnar Nerve.—This nerve is frequently wounded in warfare, 327 in 1000 of Burrow's series, and by dislocation or fractures of the humerus, and at the elbow-joint. It is frequently wounded at the wrist.

If the injury be above the elbow flexion of the hand becomes difficult. The little finger cannot be moved and the middle and ring fingers cannot be flexed in the last joint. The basal phalanges of all the fingers cannot be flexed. The fingers cannot be abducted or adducted.

Injuries lower down in the forearm cause only a loss of power of the interossei and muscles of the thumb. *Main en griffe* develops.

There is usually a definite deficiency in the prehensile function of the thumb and first finger. This may be used as an indication and gauge of ulnar paralysis, including its influence in causing disability in the occupational sense. In the detection of a weak prehensile function signifying paresis or paralysis of the ulnar nerve, the patient may be made to grasp some thin object, such as a folded newspaper, first between the thumb and forefinger of the normal hand, then between the thumb and forefinger of the affected hand, while the examiner exerts strong traction on the other end of the thin object. Where the ulnar nerve and the prehensile muscles it supplies are normal, the object will be strongly grasped with the thumb lying flat against it, opposite the likewise extended forefinger. On the paralyzed side, on the other hand,

the thumb will be found flexed, and, no matter how much force the patient intends to apply, will be in contact with the object only at its extremity, *i. e.*, with its pulp, and there will be a tendency for the object to slip from his grasp if some degree of force is applied. The reason for this lies in that forcible prehension is accomplished with the adductor of the thumb and the internal portion of the short flexor, which are supplied (except sometimes the deep head of the flexor) by the ulnar nerve. In feeble, delicately adjusted prehension, on the other hand, the adductor muscle is not used, but almost exclusively the flexors of the thumb and index finger, supplied instead by the median nerve. Thus, when the ulnar is paretic or paralyzed, only the feeble, delicate type of prehension is possible.[1] It may be of service to recall that a first dorsal interosseus muscle may remain intact with complete ulnar nerve reaction. Hence its early recovery is not a necessary index of recovery of an ulnar nerve injury.

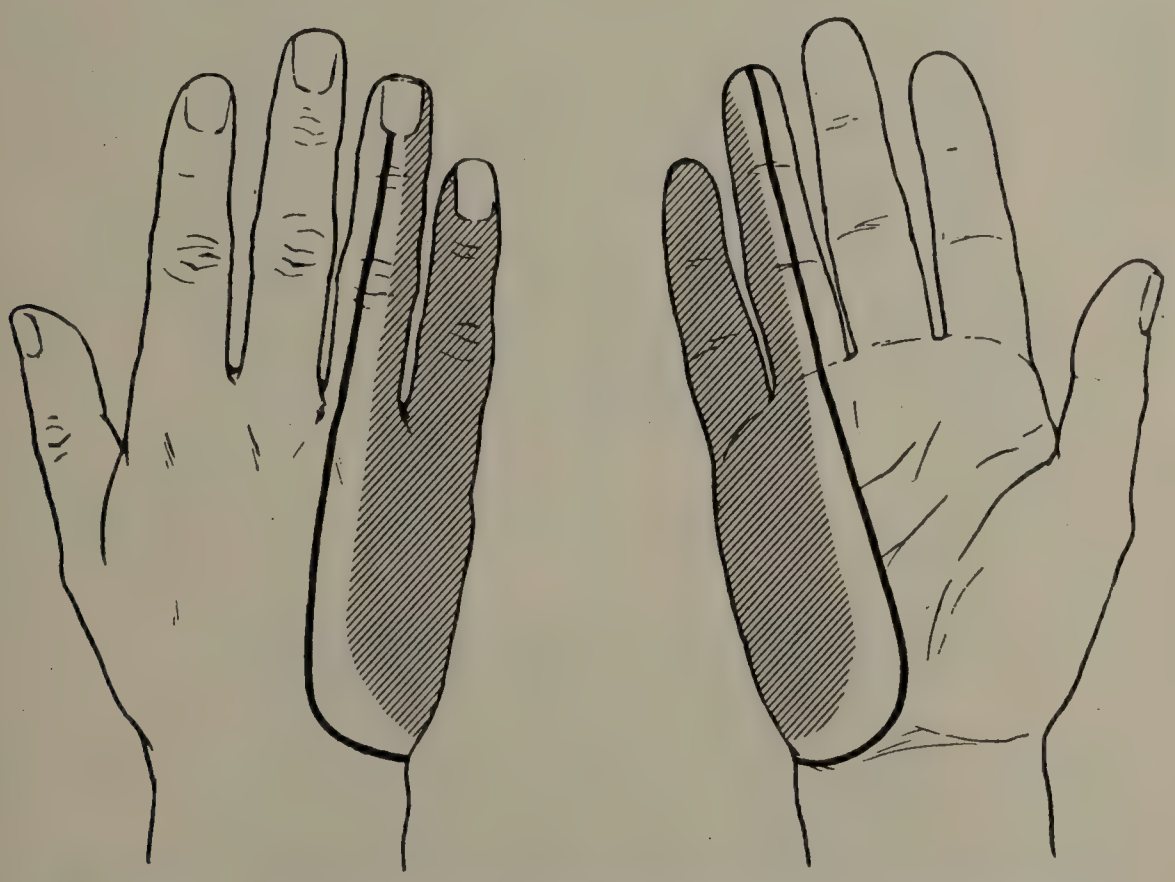


FIG. 244.—Loss of sensibility after complete division of ulnar nerve.

The sensory loss is complete in the little finger, the ulnar border of the hand has diminished sensibility: Epicritic touch and heat loss extend to the ring finger. In wrist wounds the sensory changes are apt to be minimal.

Ulnar palsies most closely resemble plexus and spinal palsies of the eighth cervical and first dorsal and must be carefully separated from them. The Klumpke eye findings are absent in the ulnar palsies.

War experience seems to show that complete section injuries are recovered from after suture very slowly even if the principles of fascicle end-to-end anastomosis are fairly rigidly carried out. Muscular power

[1] Froment: Presse méd., October 21, 1915.

is apt to be lost well up to nine months or more and loss of sensory discrimination is apt to persist as long.



FIG. 245. — Cutaneous anesthesia in complete section of the musculocutaneous (antibrachial cutaneous).

Musculocutaneous.—This nerve supplies the muscles which flex the forearm on the arm. Severe lesions cause paresis of the biceps, coracobrachialis, brachialis anticus. There is also a sensory defect, cutaneous anesthesia in the area indicated on the figures (Fig. 245.)

Median Nerve.—This nerve lies deep in the muscles of the arm and arises by two branches: an external from the sixth and seventh cervical roots and an internal from the eighth cervical and first dorsal. It supplies motor fibers chiefly to the forearm muscles, and sensory fibers to the hand. The chief functions are pronation of the wrist, flexion of the hand on the forearm, flexion of the fingers, by the deep and superficial flexors, in the hand the thenar muscles except the adductor pollicis. In median injuries these movements then are lost. To the patient the loss seems greatest in the fine movements of the fingers and thumb, buttoning, holding the pen, constitute the greatest loss. The two first lumbricales are also paralyzed but the loss is overcome by the intact interossei.

Bullet wounds, fractures and dislocations of the humerus, pressure of callus and from crutches, all may cause median nerve disorder. In Burrow's series the median was involved 242 times in 1000 peripheral nerve wounds. A type of occupation neuritis in workers who use the small muscles extensively is not infrequent in carpenters, seamstresses, cigar and cigarette makers, milkers, ironers, etc.

In addition to the motor signs there are usually distinct sensory disturbances involving anesthesia of the palmar surface of the hand as far as the middle of the ring finger extending up to the wrist. This anesthesia is most marked in a contracted area of this zone as far as the middle of the middle finger, the rest of the zone shading off to the intact sensory distribution. On the dorsum there are also slight disturbances of the fingers from the first joint outward. There is usually a marked atrophy of the thenar muscles and some flattening of the flexor muscles of the forearm. Skin disturbances, ulcers, elongation of the nails occur in severe bullet and tearing wounds. Severe causalgias occur in median nerve injuries.

The general trend of opinion regarding recovery in median nerve injury is that the flexor groups of the forearm recover first and this in from seven to eight months after proper suture. The small muscles of the hand take usually from twelve to twenty months, unless intensive

muscle exercises have been carried on. Vegetative fibers are usually involved and the causalgias resulting in median nerve injury are often quite intractable. Their treatment by alcohol injections is usually unavailing. The most frequent factor is a neuromatous end stump or



FIG. 246.—Paralysis of the median. On the left side the patient cannot bring the thumb in as on the right. (Claude, Dumas et Porack.)



FIG. 247.—Combined palsy of the median and ulnar. Flexion of the hand by the long abductor and the short extensor of the thumb. (Claude, Dumas et Porack.)

scar tissue. Complete resection and fascicle end-to-end anastomosis is advisable in cases persisting twenty months.

Combined lesions of the median and ulnar which are frequent in warfare produce very characteristic syndromes. (See Fig. 247.)



FIG. 248.—Sensory disturbances in median-nerve injury: *a*, hyperesthesia to cotton-wool; *b*, hyperesthesia to pin-prick; *c*, hyperesthesia to heat. Oblique shadings indicate diminution in heat discrimination; horizontal shadings, loss of heat sensation. (J. et A. Deierine et Mouzon.)

Radial or Musculospiral Nerve.—This is preëminently the nerve of extension of the arm and is more frequently involved than any other nerve of the arm. It extends: (1) The forearm on the arm, by the triceps; (2) the hand upon the forearm by the radial and posterior cubital; (3) the fingers on the hand by the common extensor and the extensors of the thumb, index and little finger.

The chief causative lesions are war wounds—204 times in 1000 cases in Burrow's summary, 146 in 639 of Tinel's series—fracture of the humerus, "crutch" pressure, pressure from sleeping with arms over the back of a chair, "Saturday night" paralysis, rarely in surgical anesthesia from overextension of the arm. It is partly involved in lead poisoning.

Symptoms.—The course of the nerve is very definite, and characteristic syndromes occur if the lesion involves the upper, middle, or lower third of its distribution.

The chief syndromes observable are: (1) Total paralysis. These result from pressure in the axilla (crutch—Saturday night paralysis) or surgical injuries of the plexus. Triceps paralysis, loss of olecranon reflex, loss of extension of arm, R. D. triceps. (2) Partial triceps paralysis. High up lesions cause paralysis of outer head and anconeus, with integrity of inner head. Atrophy, on palpation, of outer head of triceps is the chief sign, also loss of faradic contraction. The olecranon reflex is preserved. (3) Paralysis above supinator. This was the most frequent type before the many war wounds. Fracture of the humerus with callus complications caused it most frequently. Inaction of supinator longus, radial extensors, extensor carpi ulnaris,

extensors of fingers are the chief motor signs. Retained olecranon reflex, with integrity of triceps and anconeus. Callus involvements here cause various hyperesthetic phenomena. (4) Paresis below supinator. This causes paralysis of extension of hands and fingers, but the supinator contracts synergically with biceps. Epicondyle fracture causes it most frequently. (5) Paralysis below radial extensors. Here the wrist may be extended but the extensor carpi ulnaris and common extensors are paralyzed. (6) Extensor communis dissociation. This results from lesions below the radial extensors. Extension of the middle finger is here possible. (7) Lesions in middle of forearm may cause paralysis of extension of the thumb. (8) Dissociated palsies. (See Tinel, loc. cit.)

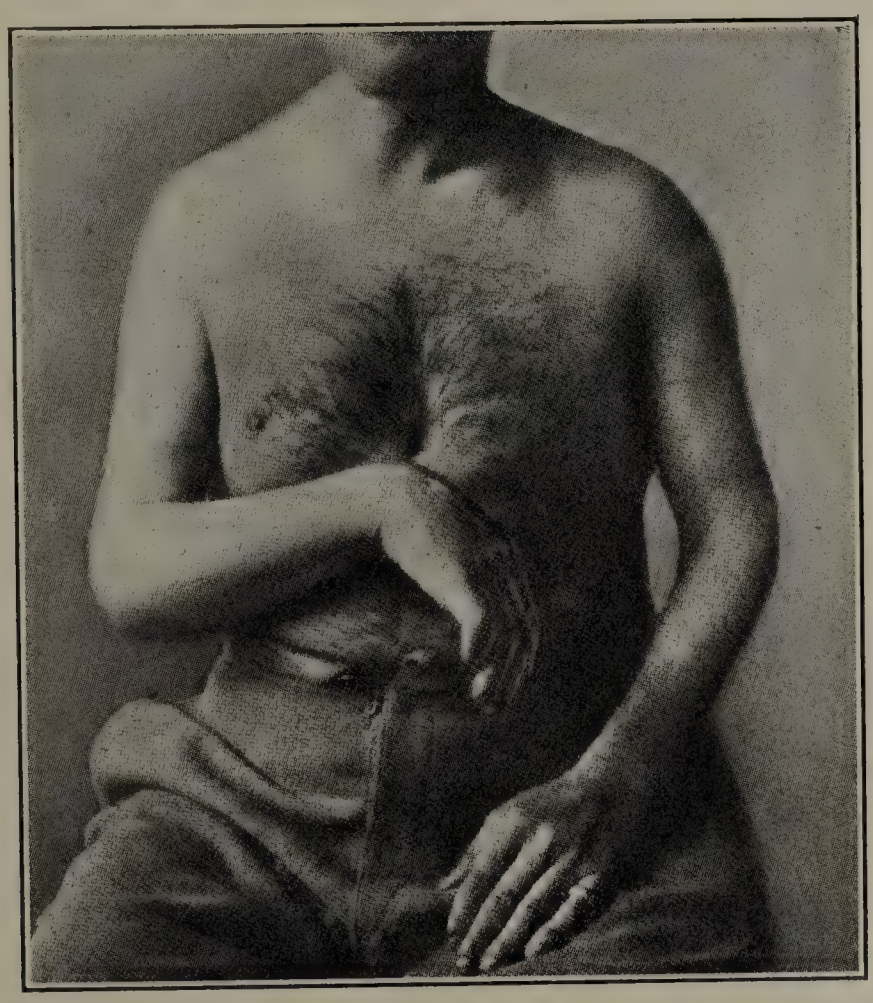


FIG. 249.—Attitude in paralysis of the musculospiral. (Tinel.)

Compression, interruption, and irritation syndromes must also be distinguished. Severe hypotonias may result which may be as disabling as paralyses. In the irritation syndrome various trophic disturbances may develop. Fibrous infiltration of the skin, desquamation, adhesion of skin on dorsal surface of fingers, loss of creases, swelling of digital articulations, tapering of the fingers are pronounced.

Wounds or pressure in the axilla cause a complete palsy. Fractures in the middle third of the humerus may bring about a special syndromy. Supinator sign, *i. e.*, loss of supinator action on forced flexion of the arm. The supinator is really a flexor, its innervation by the musculospiral is anomalous. The absence of the synergic contraction of the supinator with the biceps is a classical sign of musculospiral involvement. In head palsies it is not involved. If the dissoci-

ation is not due to lesion of the nerve *below* the supinator branch, then its paralysis is either to be explained as a spinal, a radicular, polyneuritic or psychogenic palsy.

In complete palsy the arm hangs with the forearm drawn up semiflexed, the wrist dropped, the fingers in semiflexion. Only the first phalanges are paralyzed, however, for if the first phalanges be supported the terminal phalanges are capable of extension by means of the interossei which are uninvolved.

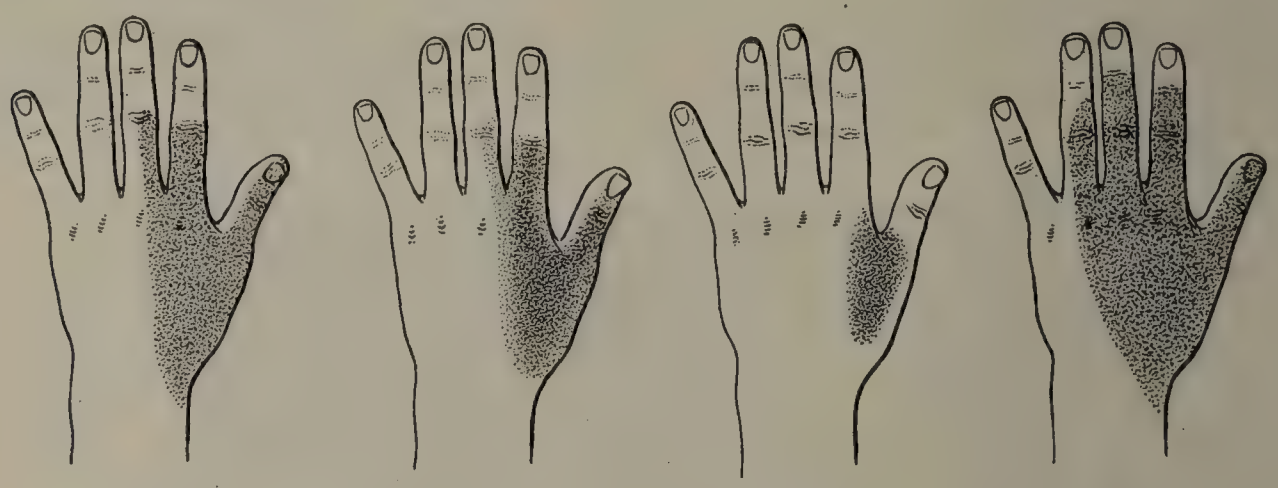


FIG. 250.—*A*. Typical distribution of anesthesia; *B*. Frequent type; *C*. Very frequent; *D*. Extension beyond typical.

When the nerve is injured on the outer aspect of the arm, as in most bullet wounds, cuts, etc., the extensors of the elbow are apt to escape, and at times the supinator, but wrist-drop is a characteristic position. Abduction and extension of the thumb are interfered with, the whole grasp is much weakened from the failure of the wrist support rather than from any palsy of the flexors. Supination is possible with the forearm drawn up by the biceps but not when it is extended.

Wounds in the lower forearm above the wrist cause perhaps only extensor weakness of the wrist.

Sensory Syndromes.—This consists of some numbness and the sense of being cold, alone, or at times with slight anesthesia over the radial branch. Slight prominence on the dorsum of the wrist is frequent from effusion or from relaxation in the extensor sheaths. In fractures of the middle third of the humerus the callus formation may cause considerable hyperesthesia.

The electrical reactions are those of the syndrome of interruption in the severe lesions. They may be less pronounced in the simple compression cases.

Treatment.—After suture restoration of function in the musculospiral seems to take place more rapidly than in any other of the chief forearm lesions. With freedom from severe sepsis, much scar tissue or specially complicating factors, the recovery is apt to be rapid but very irregular. Few patients seem to recover in about constant ratios. The extensors of the thumb are apt to recover the latest. Pain and sensory symptoms are few and rarely persist. Some patients make

complete recoveries in from nine to fifteen months. Motor incoordination is a fairly constant symptom in the first stages of restoration.

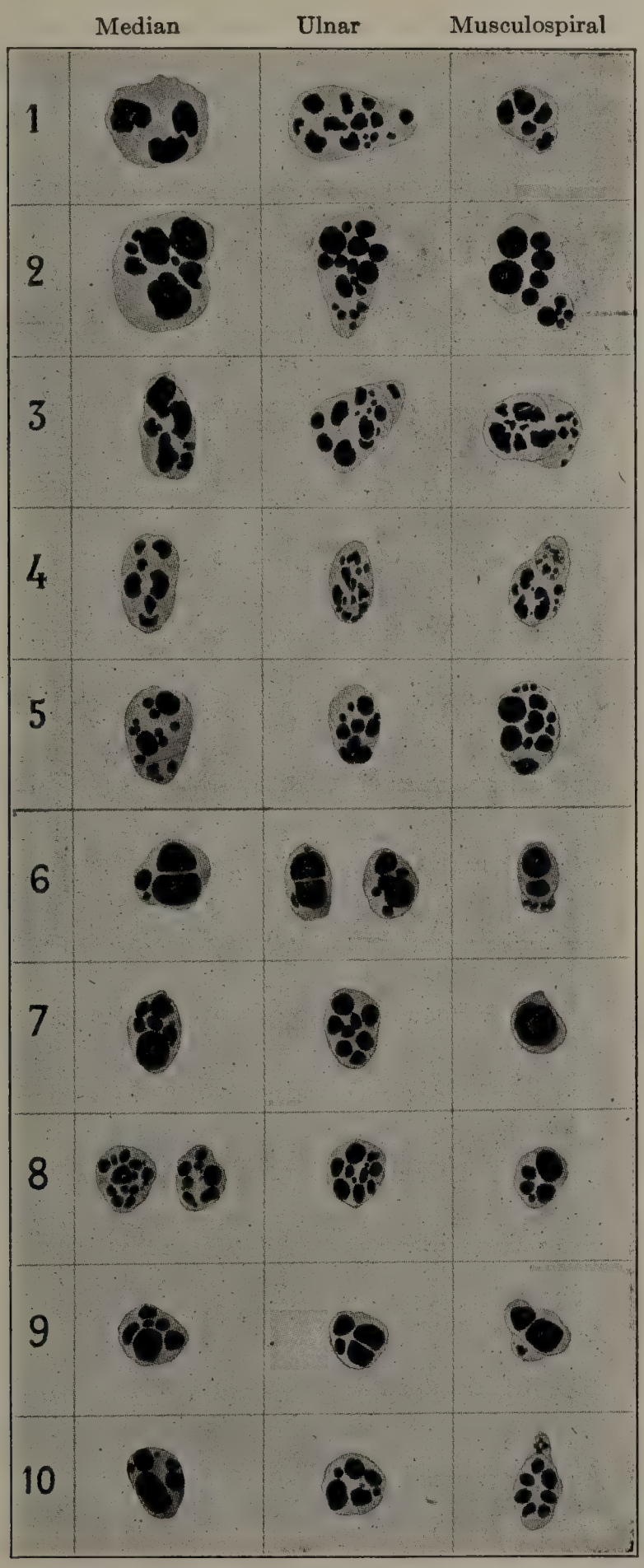


FIG. 251.—Transverse section of median, ulnar and musculospiral nerves at different levels in the upper arm, to show fasciculation. (Dustin.[1])

In the surgical treatment of the injuries of the chief nerve of the arm the principle of end-to-end anastomosis of nerve fascicles has

[1] La fasciculation des nerfs, Ambulance de "l'Océan," July, 1918, vol. 2, Fasc. 1.

become highly important. Reference must be made to the studies already quoted for a more detailed consideration.

Extension, even hyperextension, of the wrist in a proper brace is a *sine qua non* of treatment of the complete musculospiral and radial palsies. The rest of the treatment follows the usual lines already outlined, depending upon the syndrome present. The treatment of the hypotonias and other irritative syndromes requires very great skill. The student is referred to more complete discussions in Tinel's and Athanassio-Benisty's valuable monographs.



FIG. 252.—Involved sensory area in internal popliteal disturbance.



FIG. 253.—Sensory distribution of the external popliteal branch of the sciatic. Anesthesia on back and front of the leg.

Sciatic Nerve.—The sciatic by reason of its great volume, its long course and its many branches is particularly prone to injury, particularly in war, in Tinel's series constituting 25 per cent., in Burrow's 22 per cent. of the total of all peripheral nerve injuries. It arises from the fourth and fifth lumbar roots and the first, second and third sacral, uniting in one large trunk in the lower third of the thigh where it divides with the external and internal popliteal nerves. Its neuropathology may best be studied as (1) Lesions of the external popliteal, (2) lesions of the internal popliteal; (3) lesions of the trunk.

External Popliteal.—The chief collateral branches are the long saphenous and the peroneus; the chief terminal branches, the anterior tibial and the musculocutaneous. The external popliteal innervates the antero-external muscles of the leg, the extensor longus digitorum, the extensor proprius pollicis, the tibialis anticus, peroneus longus, peroneus brevis.

Paralysis therefore causes loss of ability to extend the foot and of the toes, rotation of the foot on the ankle and raising of the internal border of the foot on the ankle. The foot therefore drops, the toes pointed

downward. The gait is of the steppage type. The seated patient with the foot flat on the ground cannot elevate the toes nor raise the sole from

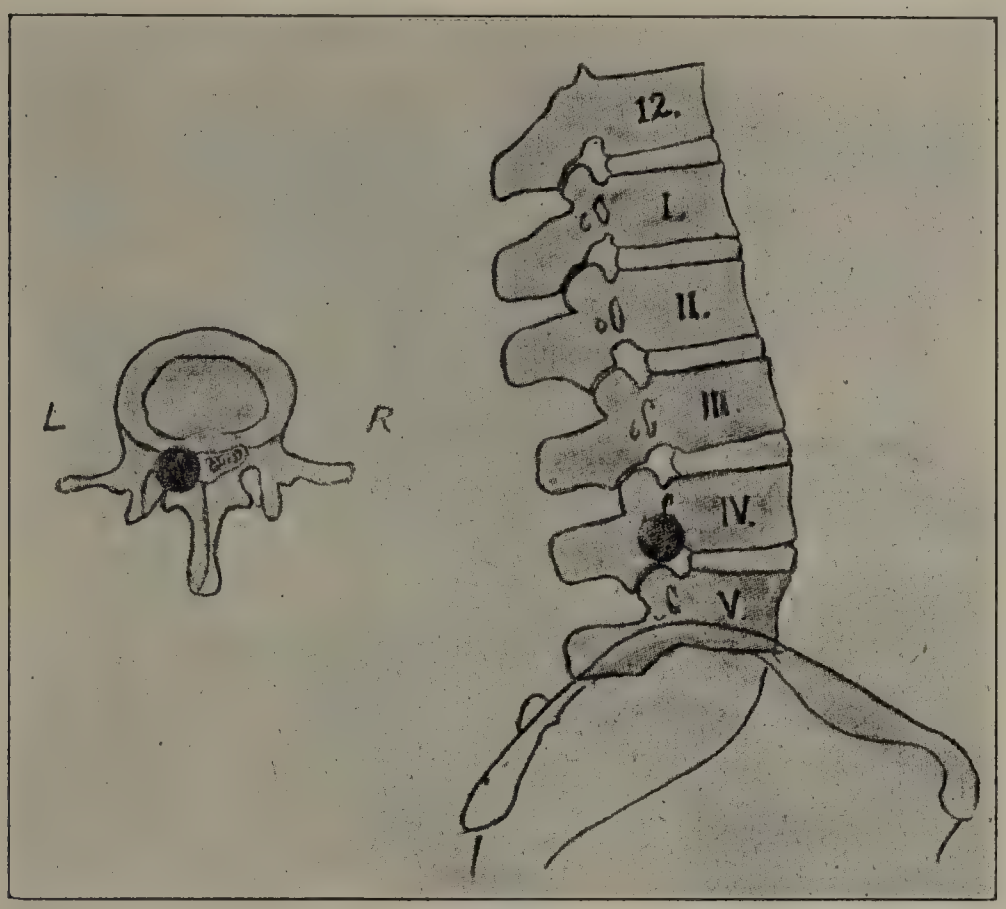


FIG. 254

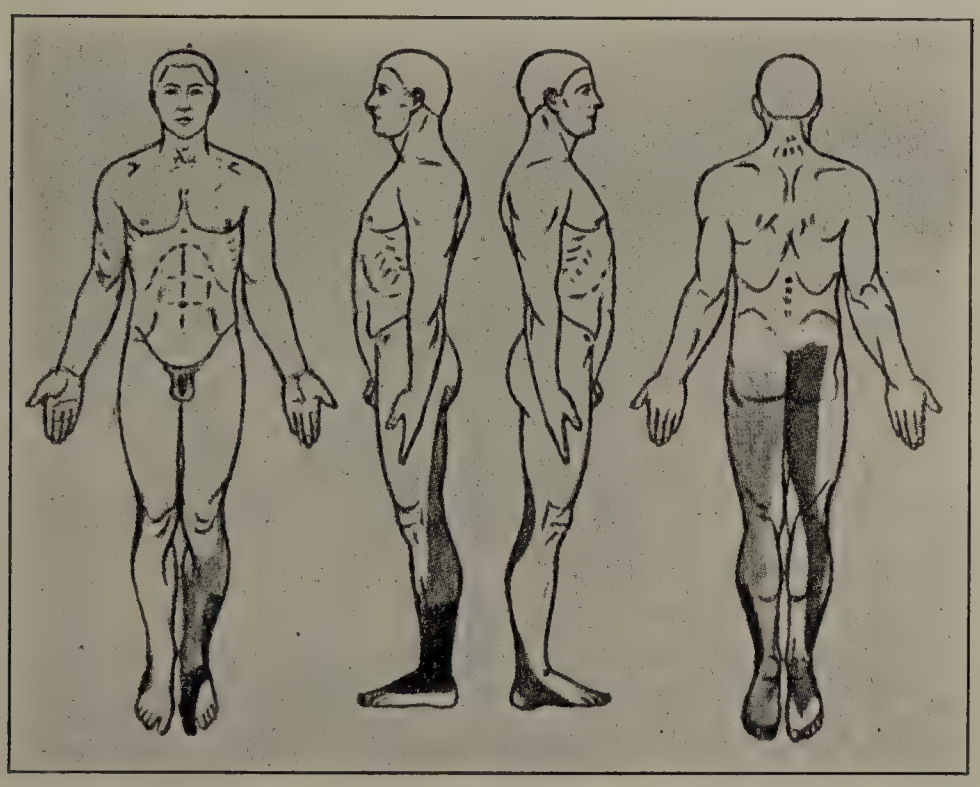


FIG. 255

FIGS. 254 and 255.—Lesion of cauda at fourth lumbar with area of sensory disturbance. (Veraguth.)

the ground. Syndromes of interruption and of compression may be distinguished.

The sensory disturbances are as illustrated. Trophic disturbances are dorsum edemas, paleness or cyanosis of the skin, desquamation, ulcers, hypertrichosis.

The upper antero-external area of anesthesia results from lesion of the cutaneous branch of the peroneus: the posterior from the peroneus; the musculocutaneous on the inferior aspect of the leg and dorsum of the foot. The anesthesia is rarely complete nor constant. It is

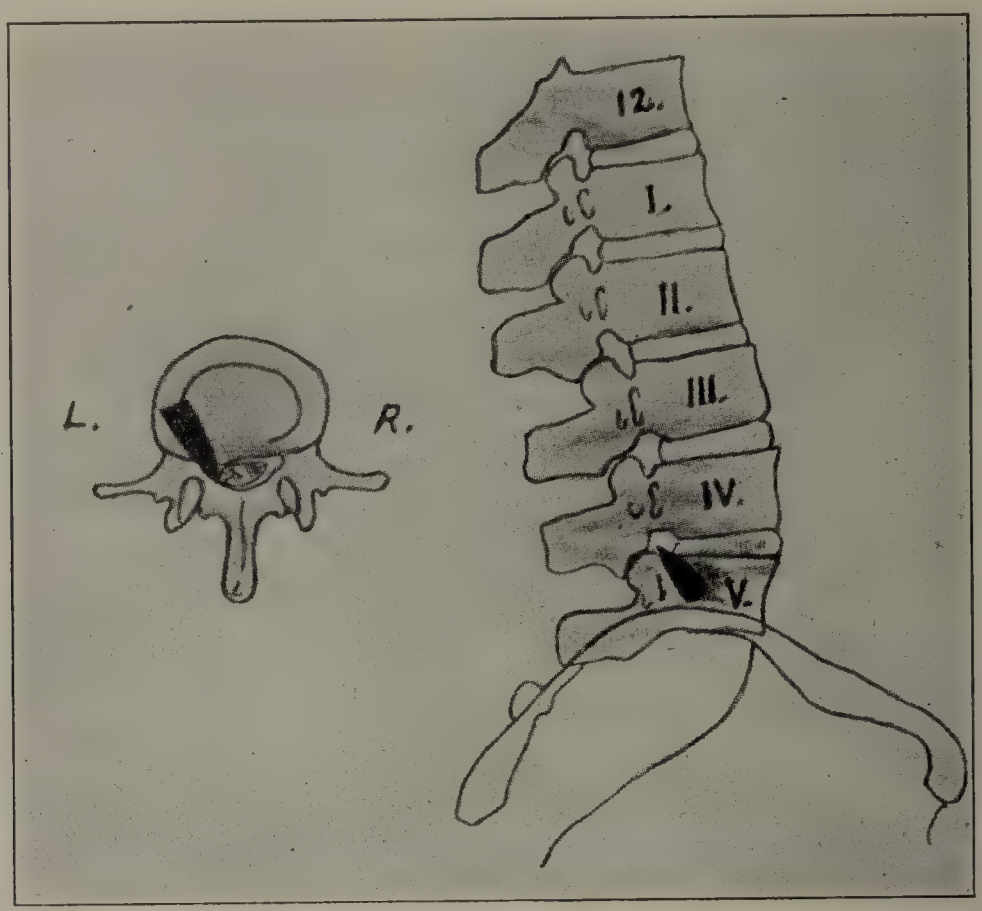


FIG. 256

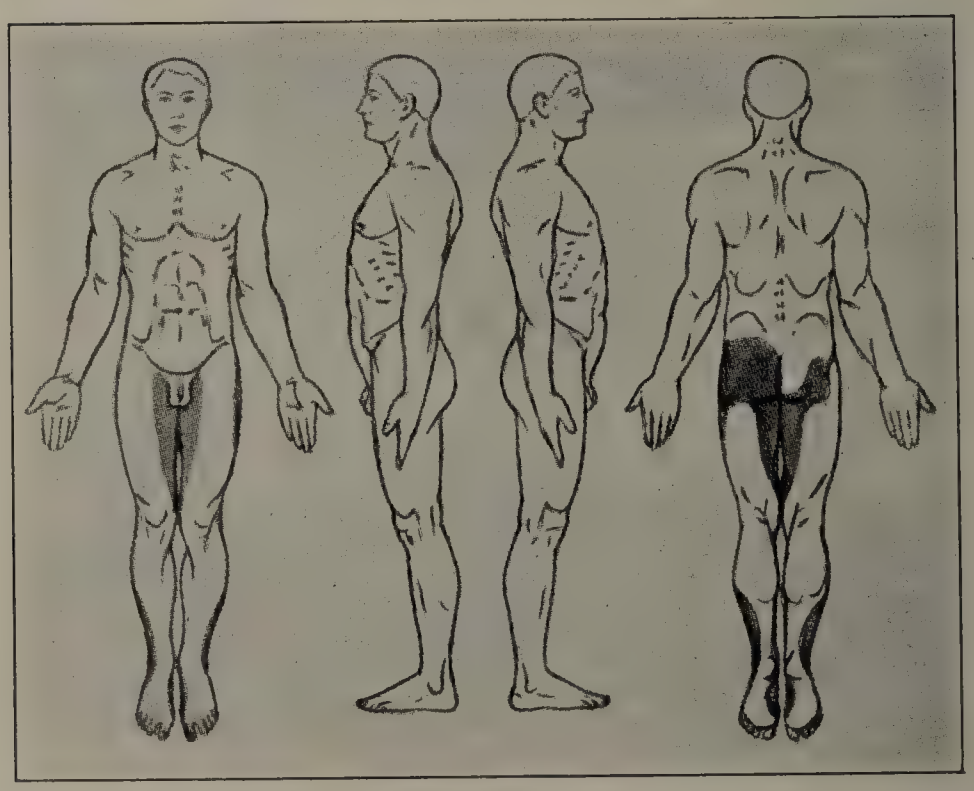


FIG. 257

FIGS. 256 and 257.—Lesion of cauda at fifth lumbar with area of sensory disturbance. (Veraguth.)

chiefly found on the antero-external surface of the leg and the dorsum of the foot.

The external popliteal is the most frequently injured branch. In many respects it behaves similarly to the musculospiral of the upper extremity in war injuries and after suture.

Internal Popliteal.—Lesions here cause loss of function of all of the posterior muscles of the leg and of the plantars. There results a loss of flexion of the foot, of flexion of the toes, enfeebled movements of rotation and adduction, loss of adduction and abduction of the toes.

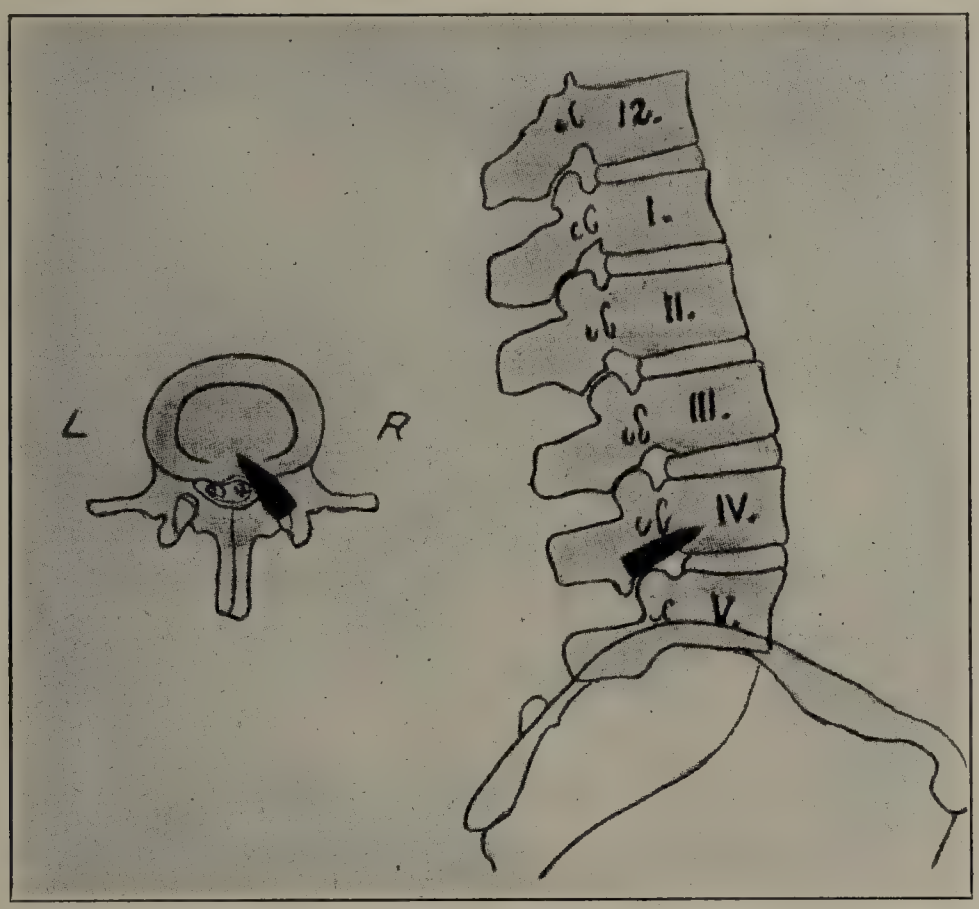


FIG. 258

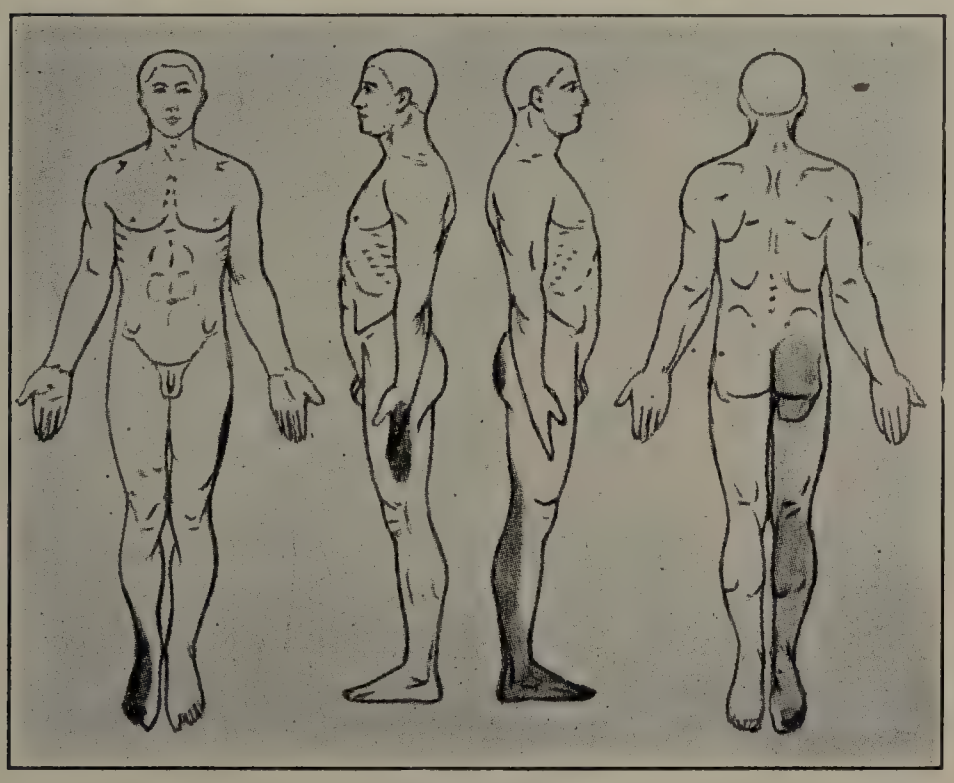


FIG. 259

FIGS. 258 and 259.—Lesion at level of fourth lumbar right. (Veraguth.)

The gait is not markedly altered to casual observation. The foot is put down flat, the patient does not rise on his toes. On rising from a sitting position, no support is given by the toes. The plantar arch is highly curved, pes valgus develops. The toes in repose are hyperextended. There is a loss of the plantar and Achilles reflexes.

Sensory disturbances are marked as illustrated. The deep sensibility fibers seem to travel mostly with this branch. It behaves as the median nerve in many respects and causalgia is here frequent and regeneration is slow and painful.

Hyperidrosis is frequent, ulceration at times occurs. In neuritic types the trophic disturbances are very marked and widespread. In lesions of the popliteal nerves the trophic disturbances are apt to be less than when the sciatic trunk is involved.

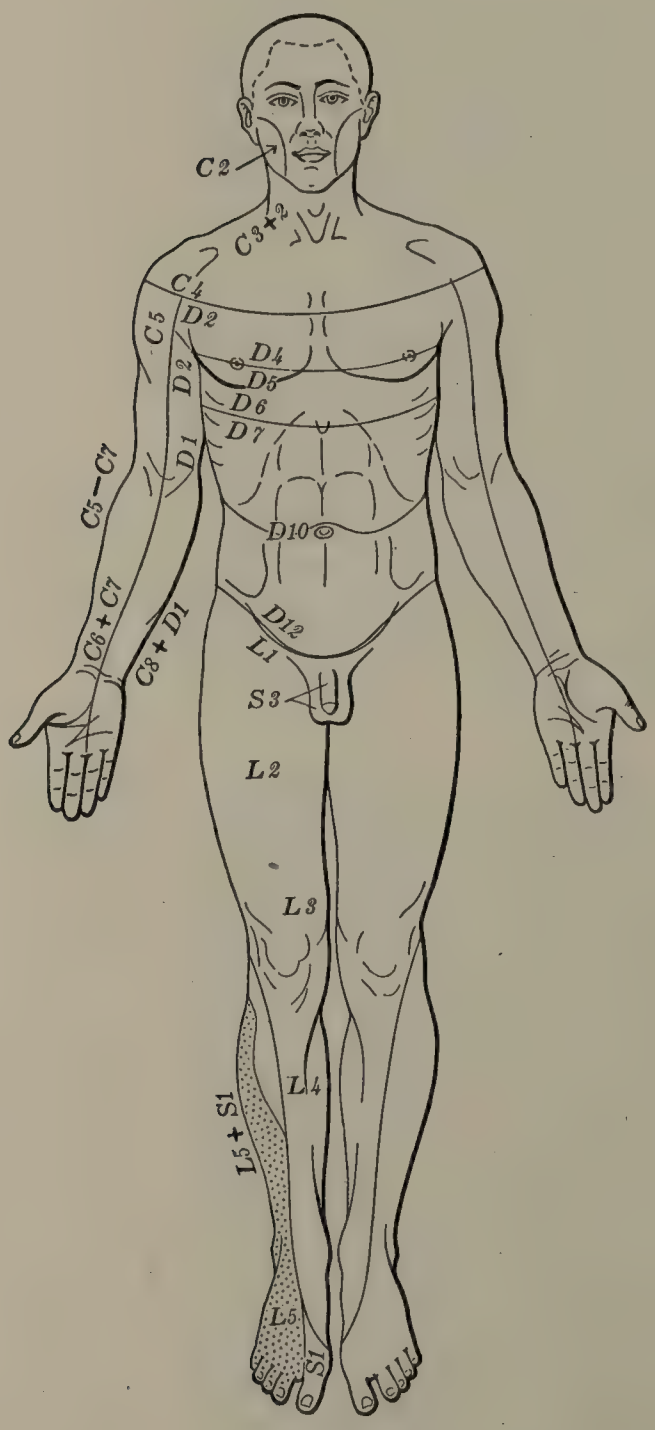


FIG. 260

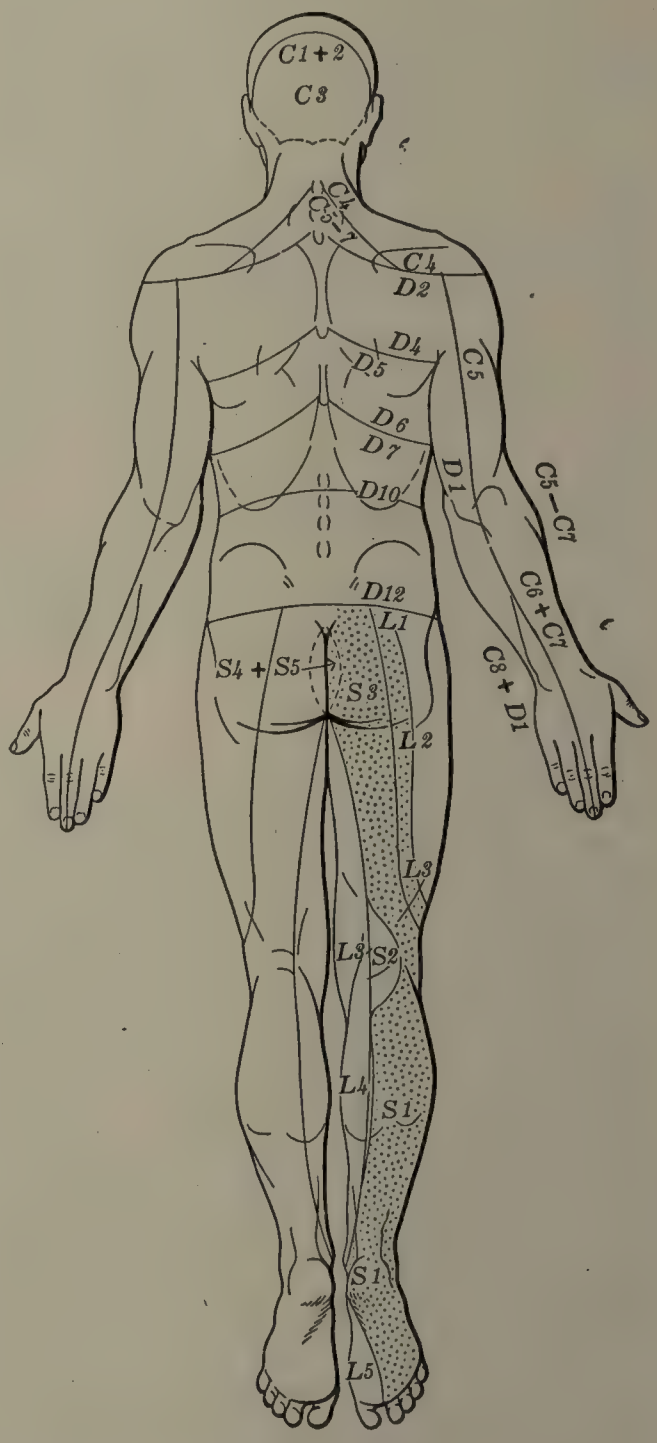


FIG. 261

FIGS. 260 and 261.—Distribution of sciatic anesthesia.

In *trunk lesions* there is a combination of both syndromes just described. There is marked general atrophy of the entire leg which moves more as a stump held rigid by the quadriceps. The glutei may be involved also. The chief types seen are the paralytic, neuritic, causalgic, and neuralgic. Partial and dissociated pictures are the rule especially in the wounds of warfare. Severe pains, tender nerve trunks and trophic changes are the rule. (See Lumbo-sacral syndromes.)

CHAPTER VI.

LESIONS OF THE SPINAL CORD.

The anatomy, histology and physiology of the spinal cord will be discussed only in so far as problems of localization and pathology are concerned.

A large group of disorders, chiefly limited to the cord, come under review. They are here presented as a series of syndromes which affect:

1. Chiefly the peripheral motor neuron (anterior horn cells).
2. Combinations of anterior horn cells and pyramidal tracts.
3. Chiefly pyramidal tracts.
4. Chiefly posterior columns.
5. Vegetative cell groups.
6. Central gray.
7. Combined and diffuse lesions.

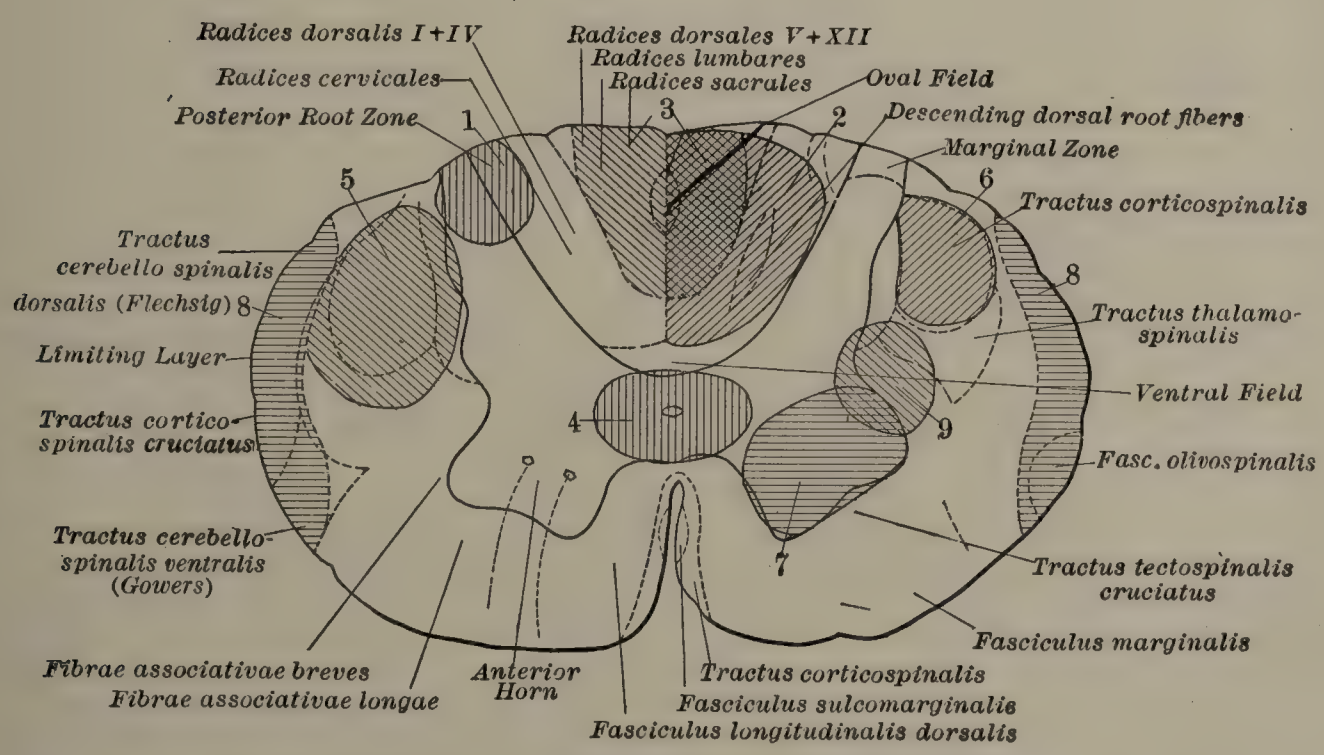


FIG. 262.—Cross-section of spinal cord showing localization of chief structures with lesions. (Veraguth.)

These subdivisions are largely arbitrary. At times they correspond to clinical entities, so-called; again fortuitous combinations occur. Thus a poliomyelitis may clearly delimit, *i. e.*, in its end-results, a group of motor neurons, anterior horn cells, while, on the other hand, spinal syphilis may show any of the localizations just tabulated. In the beginning of a syringomyelia the earliest signs may be those of irritation of the pyramidal tracts, *i. e.*, lateral sclerosis type of lesion.

Areas Involved.	Chief Syndrome.	General Diagnostic Symbol.
1. Anterior horn group No. 7. (Fig. 258)	Varying with metameres involved, paresis or paralysis of muscles, atrophy, atony, loss of reflex in muscle group, R. D.	1. Acute anterior poliomyelitis. 2. Chronic anterior poliomyelitis. 3. Muscular atrophies combined with some dystrophies.
2. Combined (1) and (2) pyramidal tract groups Nos. 6 and 7.	From (1) muscular atrophies, R. D., from (2) increased reflexes, increased tonus, contractural tendencies.	4. Amyotrophic lateral sclerosis. 5. Some multiple scleroses.
3. Pyramidal tract lesions No. 6.	Paresis or paralysis, more usually entire side, with spasticity, increased tonus, increased reflexes, no atrophy, no R. D., associated movements of opposite side.	6. Lateral sclerosis. 7. Compression, tumor, Pott's disease, bony growths, traumatic lesions. 8. Most multiple scleroses. 9. Toxic, pellagra, anemia, arteriosclerotic and senile syndromes.
4. Posterior column lesions Nos. 1 and 2.	If unilateral, Burdach's column only, with deep anesthesia, hyperesthesia, ataxia of limbs lying above lesion. Diminished or lost reflexes; when including Goll's columns, deep anesthesia, superficial hyperesthesia, below site of lesion.	10. Tabetic syndromes. 11. Toxic (lathyric, alcoholic). 12. Rare compressions. 13. Friedreich's syndromes. 14. Agenetic (cerebellar ataxias) syndromes.
5. Vegetative groups No. 9.	Trophic disturbances; vascular disturbances, changes in secretions, hair, skin, etc.	15. Trophoneuroses 16. Angioneuroses 17. Dermatoses (See Vegetative Nerv. System Disease.)
6. Central group No. 4.	Dissociation of thermal and pain senses from tactile discriminations.	18. Hematomyelic syndromes. 19. Syringomyelic syndromes. 20. Tumor, destruction or compression.
7. Combined, general and indiscriminate groups.	Showing various combinations of preceding.	21. Diffuse myelitis. 22. Multiple sclerosis. 23. Syphilitic meningomyelitis.

Atrophy of muscles may then be added, *i. e.*, anterior horn syndromes. Then a loss of pain sensibility with intact touch is seen, *i. e.*, central gray lesion. The arm begins to be edematous and trophic changes develop; vegetative cell group involvement. If the student keeps in mind the cross-section of the cord it may be seen how these various clinical pictures may be developed. (See Fig. 262.)

The chief clinical pictures are summarized in the table on page 490.

A study of the cross-section of the cord will bring these localizing factors more closely into view. The localizing features are brought out in Figs. 27–32, 34–38.

Location of Lesion.	Chief Symptoms.
1. In the posterior root zone.	Irritation causes hyperesthesiæ. Destruction causes loss of superficial sensibility in the root distribution spreading over at least three roots. Ataxia and eventually astereognosis in the extremity involved.
2. In posterior column of one side.	Anesthesia to deep sensibility and to touch. Ataxia of metameres below the lesion.
3. In Goll's columns of both sides.	Anesthesia to deep sensibility and hypesthesia of the lower extremities only even in high lesions.
4. In central gray, especially of anterior commissure.	Dissociated sensibility (thermanesthesia and analgesia in the affected metameres as indicated in the skin distribution).
5. Posterior portion of the later columns with integrity of limiting layer.	Crossed hemihyperesthesia plus the symptoms of 6.
6. Pyramidal tracts.	Spastic paralysis of the caudal metameres below the lesion without reaction of degeneration, often crossed movements, no atrophy and with increased reflexes.
7. Anterior horns.	Flaccid paralysis of the muscles of a number of root zones, atonia and atrophy of muscles of involved metameres; R. D., loss of reflexes.
8. Spinocerebellar paths.	Bilateral involvement causes cerebellar ataxia.
9. Lateral recess.	Vegetative disturbances metamerically distributed.

The majority of these cord syndromes are considered here, some are discussed under their etiological groupings: syphilis of the nervous system, for instance, taking over tabes, spinal-cord gummata, syphilitic meningomyelitis.

ACUTE POLIOENCEPHALOMYELITIS.

Historical Note.—This disease has affected mankind for centuries. Mitchell reports shortening of the femur, presumably due to this disease, in an Egyptian mummy.[1] Jonathan's son (II Samuel, iv, 4), possibly had this disease. Numerous drawings and paintings of the

[1] See Garrison's mention of Eighteenth Dynasty of Egypt picture in the Carlsberg Glyptothek at Copenhagen, Soc. Fr. d'hist. d. méd., 1911, **10**, 408, and reprinted as Plate A by Dana, Nervous Diseases, 10th edit., 1926; Jour. Am. Med. Assn., 1919, **2**, 709. Moodies, Roy: Paleopathology.

fourteenth to sixteenth centuries (Hieronymus Bosch)[1] attest to its prevalence in those times. Throughout the period of the later Latin writers it was usually included under paralyses, hemiplegias,



FIG. 263.—Engraving by Bosch in the fifteenth century, among them some of the characteristic deformities of poliomyelitis and modes of progression used before modern surgical therapy.

[1] E. Holländer: Die Medizin in der klassischen Malerei, 1903, p. 116 (Fig. 66 for example of marked quadripedal progression in severe poliomyelitis). Also see Richer: l'Art et la Médecine.

etc. Underwood, in 1784, described a disease, to him new, "Debility of the Lower Extremity," that gave a direct impetus to its definite separation as a type by Jacob v. Heine in 1840. In 1819 Jörg gave an excellent case history, and in 1843, independent of Heine, Rilliet and Barthiez contributed an important article to French literature. They called the disease an essential paralysis, and thought it had little spinal pathology. Badham, in 1835, described a group of cases undoubtedly of poliomyelitis. One of his children had an ocular palsy.

Although much was written previous to Duchenne's time, his work in 1855 marked the beginning of a new era in the study of this disease and in Heine's second edition, 1860, the status of the disease at that time is well reflected. In 1865 attention was first called to the possible relation of infantile to adult poliomyelitis by M. Meyer, which study was followed by a large number of further contributions from Charcot's students.

The anatomical era may be said to have opened with Cornil in 1863, and there then grew up the Charcot thesis of a primary affection of the ganglion cells of the anterior horns, which has had to give way to a more extensive pathology through the recent studies of Wickmann, Harbitz and Scheel, Flexner, Strauss and others (1907–1916) and the flood of observations on epidemic encephalitis (1917–1922) with which syndrome poliomyelitis has some close relationships.

Seeligmüller's masterly monograph in 1880 practically contained the standard teachings up to the appearance of Medin's[1] work in 1896, when the epidemiological features of the disease were brought out. In later years Lövegren (1904), Wickmann (1905–1907), Harbitz and Scheel (1905) have still further refined the clinical and pathological aspects, while Flexner, Landsteiner, Popper and others have been able to convey the disease from the human to monkeys and thence to other monkeys (1909–1911).

The most recent monographic treatments of the subject are found in Wickmann's[2] very able contribution and Müller's equally valuable monograph (1911), one by Römer[3] (1911), a shorter study by Peabody and Draper (1913),[4] and a valuable study by Batten[5] and another by Ruhräh and Mayer (1917)[6].

Etiology.—The disease is an acute infectious disease. The most recent (1922) tenable hypothesis is that the causative factor is an ultra microscopic something giving rise to a filterable virus which virus has partially been isolated and in different strengths seems to cause different types of lesions. It has close affinities to the virus of encephalitis epidemica and by certain observers is thought to be identical. The virus is found chiefly in the salivary secretions. In many respects the virus resembles that of rabies, of encephalitis,

[1] See translation of this study by Jelliffe and Dederer: Alienist and Neurologist, 1916.
[2] Nervous and Mental Disease Monograph Series, New York.
[3] Wm. Wood & Co., New York.
[4] Rockefeller Institute.
[5] Batten, F. E.: Brain, 1915.
[6] Lea & Febiger: New York and Philadelphia.

and of parotitis.[1] It has been conveyed by direct injection of the virus into the brain, through the uninjured and scarified nasal mucous membrane, and through injection of the intestinal lymph glands of affected animals (Flexner).

An immunity of yet unknown length seems to be established by one attack. Second attacks, though rare, do occur.[2] The disease seems to be conveyed by direct contact, through indirect contact, and through salivary, nasal and gastro-intestinal secretions. It does not seem to be highly contagious. Inoculation immunity has been obtained in monkeys.

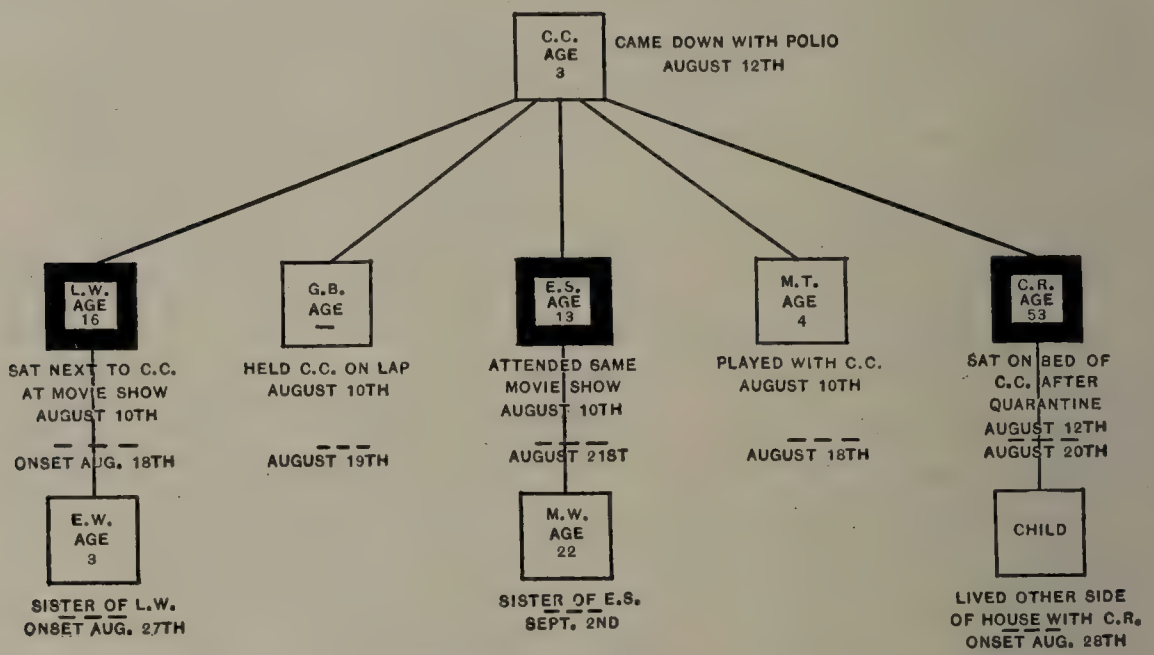


FIG. 264.—Development of a contact group of poliomyelitis cases in Jefferson County during August, 1916. (Courtesy of Dr. Mathias Nicoll, Jr., New York State Board of Health.)

Epidemic extensions as such have now been studied for nearly sixty years, and Cordier first expressed a belief in its contagiousness. Badham's early case reports seem to indicate a small epidemic. Medin definitely proved it (1896). Some eighty or more epidemics have been reported to 1922. The most recent pan-epidemic apparently started in Norway and Sweden in 1903–1904, spread to the United States in 1907–1912, to Germany and France in 1908–1911, with isolated far-lying outbreaks in Cuba, Australia, etc. The epidemic of 1916 in the vicinity of New York is the most extensive on record. The recent world wide epidemics of encephalitis (1916–1921) and influenza must be studied in this connection.[3]

Rural districts have suffered greatly, and density of population has not seemed to play a large role; northern countries seem favorable to

[1] See Studies by Levaditi, Harvier, Nicolau and others: Compt. rend. d. seances d. l. Soc. de biol., Paris, 1921, **85**, *et seq.* See paragraphs on Epidemic Encephalitis, Ann. de l'Inst. Pasteur, February, 1922. Rosenow: Newer Findings, Minnesota Med., 1926, **9**, 231. Walshe, F. R. W.: Lancet, February 12, 1927.

[2] Taylor, E. W.: Jour. Nerv. and Ment. Dis., September, 1916, for complete discussion. Moncrief: Ibid., 1919.

[3] See important epidemiological discussions by Crookshank: Influenza, London, Heinemann, 1922.

the outbreak of the disease, and the season of greatest intensity is usually in the warm months of July, August and September. Some epidemics have occurred in winter. (Wickmann.)

The majority of the cases occur in children from one to five years of age. Intra-uterine cases are known[1] and individuals as old as sixty have contracted the disease. Certain epidemics have shown marked

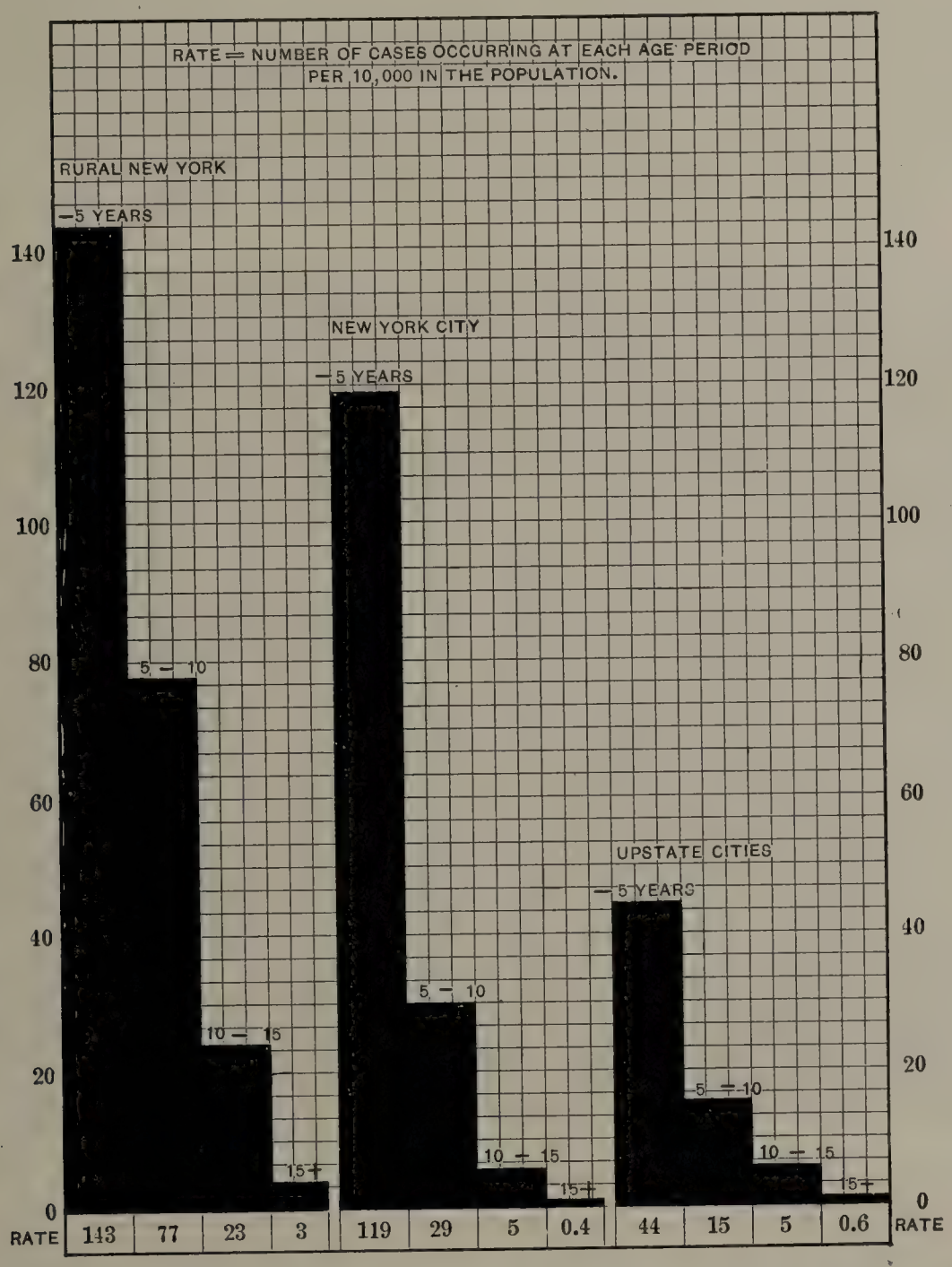


FIG. 265.—Comparative incidence rates of poliomyelitis, by age periods, in different sections of New York State during the epidemic of 1916. (Courtesy of Dr. Mathias Nicoll, Jr., New York State Board of Health.)

variability in the matter of age incidence, the Swedish epidemic of 1905 having as high as 10 per cent. adults. In large epidemics adults seem more often affected. The male sex has seemed to be more often involved, but the differences are not very marked. Nationality seems to play little role, although it has been assumed that Scandinavians

[1] Batten: Brain, 1910. Collier: Epidemiology, British Med. Jour., April 23, 1927; Public Health News, Trenton, 1925, vol. **10.**

are peculiarly susceptible. This may only be an indication of the more careful study given by these authors. Predisposing hereditary inferiority factors are as yet incompletely studied.[1]

The incubation period varies from one to ten days, the majority showing a period of from one to five days. In experimental poliomyelitis of monkeys the incubation time is about six to nine days.

Pathology.—The older concepts of a primary intoxication or of an inflammation of the motor horn cells exclusively must be abandoned. Acute poliomyelitis is due to an acute interstitial inflammation (a meningomyelitis), involving all parts of the nervous system.[2] There is a congestion, infiltration and edema of the leptomeninges, cord, medulla, pons, cerebellum and cerebrum. The dura is usually less

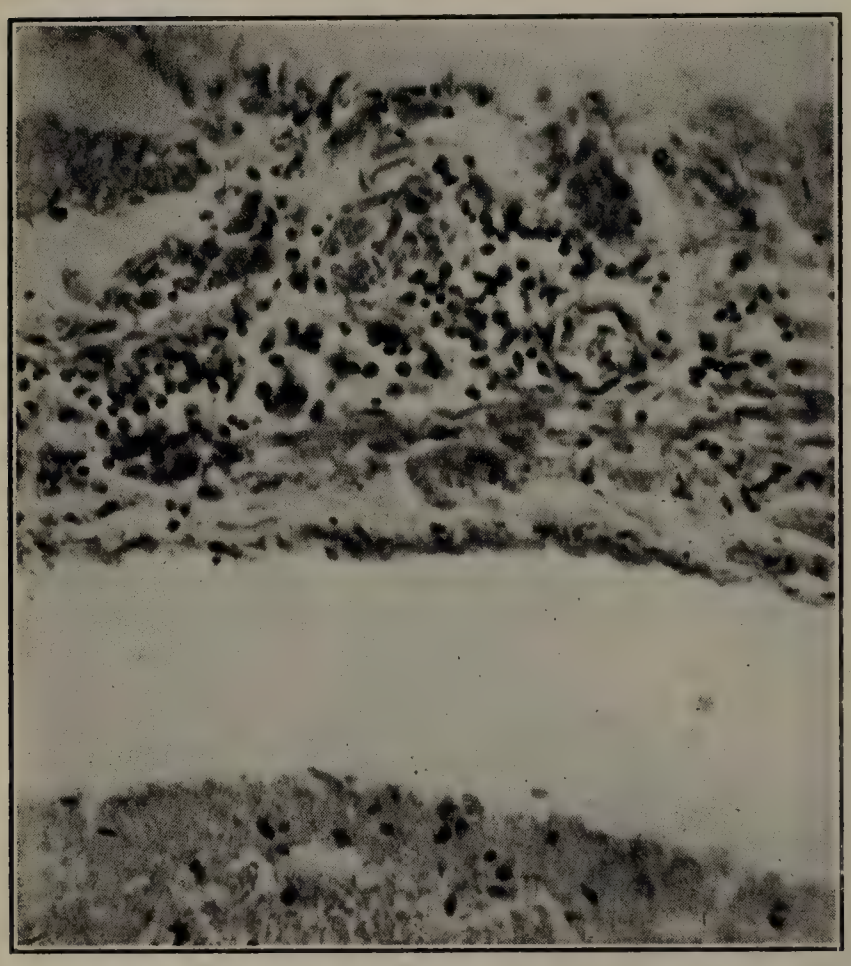


FIG. 266.—Acute poliomyelitis, showing associated meningitis at the edge of cord.



FIG. 267.—Acute poliomyelitis, showing the vascular congestion and the surrounding infiltration area of the anterior spinal artery.

markedly involved; the pia is congested and infiltrated with round mononuclear cells (lymphocytes), particularly in the sacral and lumbar regions. The vessels are congested and their sheaths infiltrated, the progression of the inflammatory reaction apparently following the vessels from the periphery to the interior of the cord. The cerebrospinal fluid is increased in quantity, almost opalescent early in the disease, with markedly increased lymphocytes, in some of which Lafora and Hough have found pictures resembling the Leishman-

[1] Paulis: Jour. Med. de los ninos., 1921, **12**, 35.

[2] See Howe, H. S.: Jour. Nerv. and Ment. Dis., 1918, **49**, and 1919, **50**, for complete recent summary of pathological histology.

Donovan bodies. The fluid later becomes clearer, but still shows a pathological increase in leukocytes.

Within the cord the inflammatory process follows the pial processes into the depths of the anterior fissure and along the sheaths of the central vessels. The posterior root fibers and the spinal ganglia are also infiltrated.

The vascular lesions are particularly noticeable and the interstitial and ganglionic changes depend largely upon them. The vessels throughout are dilated and engorged, the capillaries often being enormously distended. This marked hyperemia is found throughout, and vascular hemorrhagic lytic changes are frequent but small. As in rabies, and to some extent in syphilis, there is a marked perivascular

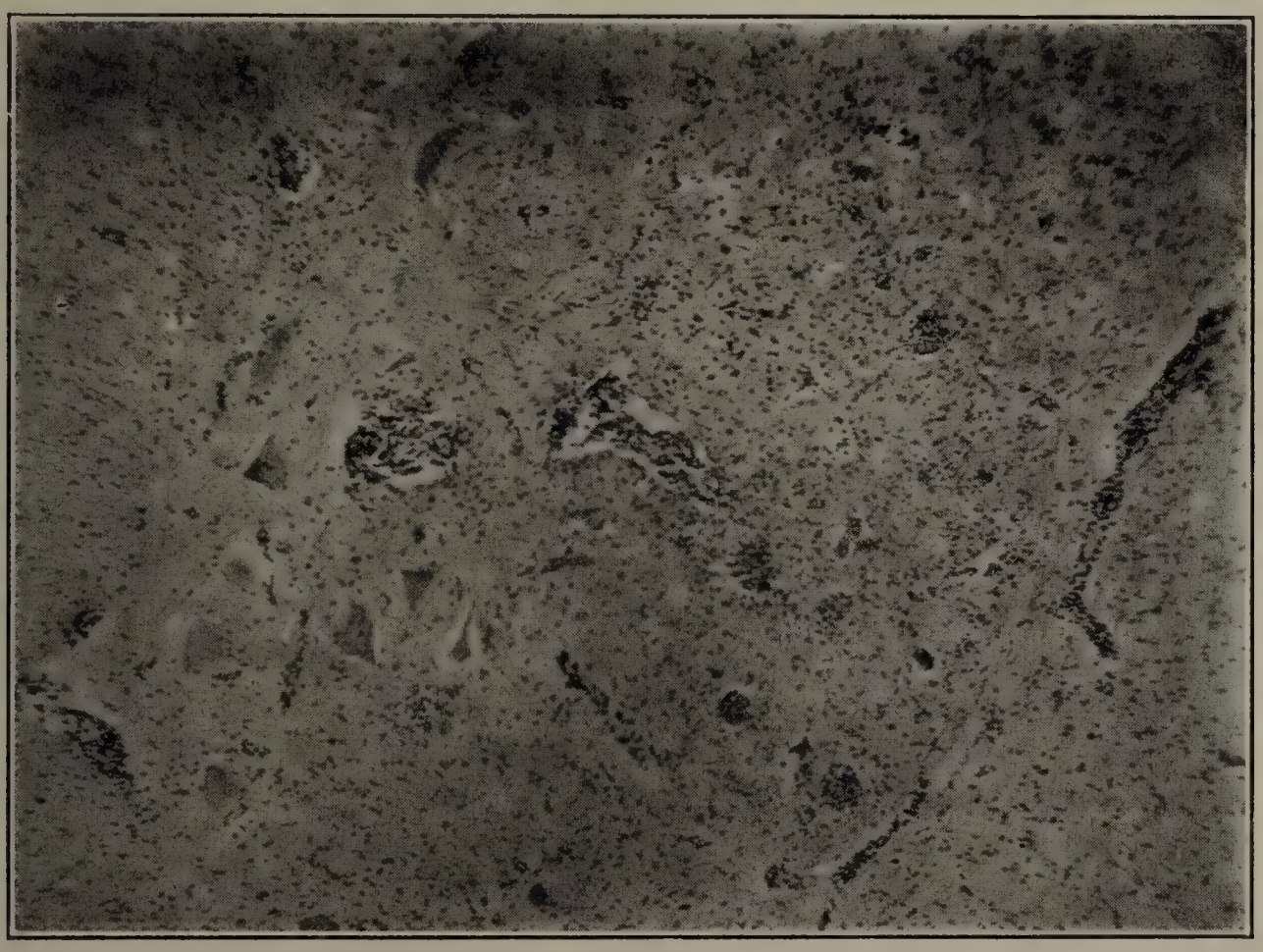


FIG. 268.—Acute poliomyelitis. The various stages of destruction of the anterior horn cells.

or intra-adventitial infiltration, apparently of lymphocytes, not plasma cells. Interstitial changes in the gray and white matter, chiefly of an infiltration of cells, and proliferation of glia cells, occur. By reason of the rich blood supply the gray matter of the anterior horns bears the brunt of the inflammatory edema and hyperemia, with destruction of many of its motor cells. This is a secondary process. Harbitz and Scheel believe that small abscesses are formed, but this is an exception if it does occur.

The ganglion cells undergo varying degrees of degeneration, somewhat proportional to the infiltration, the axis-cylinder finally breaking down. Amid the degenerated cells many normal ones may be found, and the distribution of degenerated areas is very variable, the

sacro-lumbar cord being more seriously affected, although any part of the cerebrospinal axis may be involved. All classes of ganglion cells go under, but the lateral dorsal cord regions are much less involved; however, many vegetative system cells are injured.

As a rule the functional involvement by reason of the edema, hyperemia, and infiltration, is far in excess of the permanent anatomical loss, hence the widespread character of the paralysis in the early stages and the marked degree of recovery possible. Only a small proportion of the primarily involved ganglion cells degenerate entirely. The fiber pathways may show permanent infiltration changes with the production of anomalous spastic phenomena.

The spinal nerves are involved usually at their junction with the cord, and some polyneuritis may be present early in the disease.

The medulla, pons, cerebellum and cerebrum all are involved to a greater or lesser degree. Certain cases show that the main lesion is in one or more of these regions, rather than in the spinal cord. True encephalitis, with varying degrees of feeble-mindedness, is a frequent end-result of these involvements of the upper-lying nervous structures. Isolated cortical involvement is also occasionally seen.

The other organs of the body do not escape. There are evidences of an acute general infection everywhere.[1] The hypophysis may be involved as also other endocrine structures. Lymphoid tissues are affected, including the thymus.

Symptoms.—The study of the recent epidemics has shown a great variability in the affection, but practically all of the cases show the effects of an acute infection as prodromal and early signs. The later course of the disease, dependent upon the locations chiefly involved, permits a separation into several types of which Wickmann recognizes eight classical pictures.

Prodromata are usual. They vary in the different epidemics. They consist of fatigability, loss of appetite, slight digestive disturbances, with nausea, looseness of the bowels, coryza or bronchial irritation (bronchitis, bronchopneumonia) with slight elevation of temperature. Conjunctivitis may occur; lymphatic swellings are usual. The patient may thus suffer for twenty-four to seventy-two hours before the acute prostrating effects of the disease become manifest. Some few cases show almost complete recovery after such prodromata, and then again taken ill suddenly or may go on to recovery (abortive cases). Careful observation will probably reduce the number of cases reported as coming on without prodromata.

The cerebrospinal fluid in the prodromal stages may show opalescence with very marked lymphocytosis. The blood changes are apparently not constant. La Fetra has reported a leukocytosis of from 13,000 to 20,000; whereas Müller has found a leukopenia of from 3000 to 5000, and also finds leukopenia in experimental monkey poliomyelitis. The lymphocytes are increased. No parasites have

[1] Walter: Deutsch. Ztschr. Nerv., vol. 45, No. 2.

been found in the blood up to the present time. Many bacteria have been isolated but it is not yet shown that any are specific etiological factors for the epidemic form, although it is known that a sporadic poliomyelitis syndrome may arise from a number of different infections.

After twenty-four to seventy-two or more hours the temperature suddenly rises. It varies from 99° F. to 102.5° F. or even 105° F. to 106° F., and bears little relation to the severity of the disease. Abortive cases have shown high temperatures and severe cases little. Chills and convulsions occasionally attend the rise in temperature. The temperature curve is not characteristic, being either remittent or continuous. Subnormal temperatures occur and indicate marked cervical involvement.

Headache is a frequent symptom. It is often severe and usually frontal; it may be occipital, resembling a meningeal headache. Prostration is marked, diarrhea is extremely common, vomiting is frequent and constipation is not unusual. Respiratory symptoms are not marked, save when respiratory palsies occur. The kidneys show little, and the heart's action is that in an acute infection, in general with a tendency to show nervous tachycardia, especially in the cases with medullary involvement. Weakness is the rule.

Early profuse sweating is a frequent symptom and skin eruptions are occasionally seen, although herpes is rare in contrast with cerebrospinal meningitis. Other exanthemata occasionally appear.

Nervous Symptoms.—As a rule there is considerable restlessness especially in younger patients. The children are peevish, petulant or very fretty. Sleep is often disturbed with frequent crying out. Some children lie drowsy or apathetic and may, though rarely, show coma, delirium or convulsions. Pain is a frequent early symptom and may be very severe. Marked hyperesthesia is usual (90 per cent.), and is brought on by the least attempt at motion of the limbs, pressure on the nerve trunks, touching of the skin. Heine called attention to this in 1840. Movements of the head and spine are particularly painful and some patients are extremely anxious and fearful, whimpering continuously, closely resembling, in the early stages, meningeal cases. There is frequent photophobia and hyperacusis.

The infiltration in the cord causes other sensory symptoms, such as paresthesiæ and anesthesiæ. Stiffness is not infrequent, with slight though not marked retraction of the head (Kernig's sign) in some and varying degrees of rigidity of the limbs, with contracted position of the lower limbs particularly, are frequent.

Twitching and jerking of the limbs are also frequent.

The stage of paralysis soon sets in and, according to the predominant localization, following Wickmann, eight types may be distinguished. These types represent general tendencies rather than hard-and-fast differences. The further symptomatology will be given in accordance with these divisions. These types are: (1) spinal poliomyelitis forms (the commonest type); (2) acute ascending types (Landry's paralysis); (3) bulbar or pontine forms; (4) encephalitic forms; (5)

cerebellar and ataxic forms; (6) meningitic forms; (7) neuritic forms; (9) abortive forms. To these we would add the (8) vegetative types in which the Jacobsohn's nuclei are invaded with consequent trophic and visceral vegetative symptoms. These are frequently mingled in the general paralytic medley.

1. **Spinal Forms.**—There is usually an early prodromal weakness, or even a paresis which is very widespread. This develops to a relatively marked paralysis, sometimes within twelve to twenty-four hours, but more often in from two to five days, occasionally after a week.



FIG. 269.—Abdominal muscle palsy—poliomyelitis. (Frauenthal.)



FIG. 270.—Paralysis of left arm and left leg. (Ruhräh.)

Careful observation has shown that the paralysis is a gradual, rather than an abrupt one. It begins with weakness, advances to paresis, and finally becomes a definite paralysis, whereas the weakness, however, is very widespread, the palsies are less so, and the paralyses even more restricted. When the ultimate stages are reached, the weaknesses clear up, the palsies gradually disappear, and the residual paralyses often represent but a small part of what appeared to be a wholesale devastation. This is in strict accordance with the pathological features.

The distribution of paralyses is due to factors concerning which there is little definite information. Any muscle of the body may be involved. Involuntary muscles are frequently implicated (vegetative nuclei) with chronic ptoses, vascular disorders, intestinal symptoms, etc. From a purely statistical study of the cases it has been found that the lower limbs are involved twice as often as the upper; in some epidemics four times as often. The entire limb is rarely involved permanently, but special muscle groups are picked out. Thus in the lower extremities the quadriceps, the peronei, and the tibialis anticus are the oftenest affected; in the upper extremities the scapular and deltoid muscles. Asymmetry in the final picture is the rule. The muscles of the trunk are involved next most frequently, while the arms are least frequently involved. The trunk muscle palsies are often overlooked, however.



FIG. 271.—Anterior poliomyelitis. Extreme flexion deformity at the hips, inducing quadripedal locomotion. (Gibney.) Compare some of pictures of Bosch (Fig. 263).

In young children it is almost impossible to localize the paralyses in the early stages, especially as many such patients go through the initial stages of the disease without there being a suspicion of the real difficulty. Here the loss of the reflexes, the hypotonus, the careful scrutiny of the position of the limbs, the behavior to passive motion and resistance movements and the tickling responses lead to a correct appreciation of the difficulties. They also permit a diagnosis of rudimentary and of mild cases. Babies in arms behave as though they were lumps of dough, and the mother notices the hypotonus.

Weakness of the muscles of the abdomen is an important early diagnostic feature, especially in the differentiation from meningitis.

The involvement is usually bilateral and diffuse. The muscles are hypotonic, and swell out as though the intestines were inflated with gas. There is often a loss of the epigastric and abdominal reflexes—symmetrical or asymmetrical. The patients, further, are unable to come from a horizontal to a sitting position. In some patients the abdominal muscles are alone involved. Obstinate constipation usually accompanies the abdominal palsies.

Of the back muscles the latissimus dorsi are the oftenest involved. The glutei are also somewhat implicated. Children with these palsies waddle when they walk. They behave like children with muscular dystrophy on rising from the floor.

Only rarely is the diaphragm implicated. They are usually among the fatal cases. Unilateral diaphragmatic palsies have been observed.[1] The muscles of the hips are involved in at least two-thirds of the cases.

The following table from Wickmann will serve to indicate the general run of the cases. The figures represent the study of 868 cases in the Swedish epidemic of 1905:

1. Paralysis of one or both legs	353
2. Paralysis of one or both arms	75
3. Combined paralysis of arms and legs	152
4. Combined leg and thigh paralyses	85
5. Combined arm and thigh paralyses	10
6. Isolated thigh paralysis	9
7. Paralysis of the entire musculature	23
8. Ascending paralysis	32
9. Descending paralysis	13
10. Combination of spinal and cranial nerve paralyses	34
11. Isolated cranial nerve palsies	22
12. Localization uncertain	60
	868

The figures of the Committee of the New York Neurological and New York Pediatric Societies give similar results.[2]

Sensory Disturbances.—The older teachings that sensory disturbances are uniformly absent is not true. Almost invariably there is in the beginning of the disease a marked hyperesthesia. The slightest touch causes marked reaction. Loss or diminution of sensation is also not uncommon. Complete anesthesia is rare, but has been observed. Hypesthesiæ are common, and may include both thermal and pain sensibilities. In young children the difficulties of observation cause one to overlook these sensory anomalies.

Vegetative Involvements.—The bladder is frequently involved in the early states. Urinary retention is frequent, incontinence is rare. As a rule the disturbance is transitory, hence overlooked. It is frequently thought to be a symptom of the general infection, but it is due to central lesions.

Constipation is not unusual, and marked disturbances of the intestinal functions do occur. These are often of the nature of colitis,

[1] Sylvester: Boston Med. and Surg. Jour., September 16, 1915.
[2] Nervous and Mental Disease Monograph Series No. 7, New York, 1910.

mucous colitis, etc. A chronic intestinal weakness may be a residual of a poliomyelitis.

Vascular disorders are very frequent. Skin lesions from reduced skin tone are often met with in later life. Visceral ptoses, relaxed ligamentous attachments, and other vegatative nerve-level disorders have been observed. Bony lesions are very frequent.

Atrophic Stages.—For a variable length of time, a year at least, the palsied or paralyzed muscles gradually regain their form and their function, but sooner or later, depending on the grade of central involvement, a more or less permanent state of inactivity is reached—a residual period in which an account of stock may be taken. In this stage one meets with the definite atrophies, the beginnings of the various deformities, changes in the bones and joints, and the residual secretory and trophic anomalies.

The various deformities belong more in the domain of orthopedics and cannot be taken up here. Pes equinus, pes calcaneus, pes valgus, pes varus, hyperextension of the knee-joint, genu valgum, genu varum, scolioses, kyphoses, lordoses, torticollis, flail arm, etc., are among the more common residual deformities. Seeligmüller's monograph, already cited, treats of them at great length, also consult monographs of Ruhräh and Mayer, Lovett and Vulpius.

The permanent vasomotor disturbances are chiefly cold extremities and cyanosis. Dryness of the skin is frequent.

2. **Acute Ascending Form.—Landry's Paralysis.**—Landry, in 1859, described an acute ascending paralysis, which later study has shown to be, for the most part at least, a true polioencephalomyelitis. In certain cases it is a neuritis. Rabies also has a Landry paralysis type. Here the palsy shows itself usually first in the lower extremities, then the muscles of the hip, the abdomen, the thorax, and the cranial nerves are involved, and death generally occurs through implication of the cardiac and respiratory synapses. Death takes place in from four to five days, usually with clear consciousness, or slight coma. The sensibility is usually intact, or only slightly dulled.

Occasionally the patients recover, and then show the residuals of a severe spinal poliomyelitic type, with mixture of bulbar or pontine features.

A descending form is also to be distinguished. This is much rarer. Here the bulbar symptoms develop early, and the spinal extension shows later.

The majority of the fatal cases of epidemic polioencephalomyelitis show the type of a Landry paralysis.

3. **Bulbar and Pontine Forms.**—Here the features that stand in the foreground, either as initial or as residual conditions, are the cranial nerve palsies. A large number of the patients with poliomyelitis show some cranial nerve complications, but when these are the chief features, and the spinal palsies are the minor complicating factors, then one speaks of the bulbar and pontine types.

Many of these patients show isolated palsies—others have two or

more cranial nerve involvements. The facial is oftenest implicated (10 per cent.), next the hypoglossal. These are usually unilateral although Medin has described a bilateral facial palsy. Eye-muscle palsies are infrequent; the third and fourth less often than the sixth (Wickmann, Müller).

Ptosis, ophthalmoplegia interna, externa, nystagmus are among the rarities. Pupillary disturbances are not infrequent, according to Lundborg. Here both light and accommodation reflexes are interfered with. Very rarely one observes amaurosis with optic-nerve involvement. The presence of choked disk in meningitis is an important differential. The IXth, Xth, XIth are involved, usually in the fatal cases, occasionally unilateral pharyngeal and laryngeal palsies are residual conditions. Speech disturbances may be present, but are



FIG. 272.—Eye palsies, poliomyelitis. (Frauenthal.)



FIG. 273.—Facial nerve palsy, poliomyelitis. (Frauenthal.)

infrequent. Complete central deafness has been observed. Midbrain involvements with peculiar tremors, vertigoes, forced movements, athetoid movements, paralysis-agitans-like movements, rotary movements, ataxias, choreas, myoclonias, cerebellar ataxias, cerebellar gait, etc., are among the curiosities. Howe[1] has described an interesting thalamic syndrome case. The 1917–1920 encephalitis epidemica should be borne in mind in considering the possibilities of these syndromes.

4. **Encephalitic or Cerebral Form.**—Strümpell called particular attention to the possibility of a pure encephalitic form of the disease, thus widening the conception from a poliomyelitis to a polioencephalomyelitis. Medin's valuable study confirmed his teaching, and the investigations of recent years have still further amplified the find-

[1] Neurol. Bull., 1919, vol. 2.

ings, especially the numerous cases in which an epidemic encephalitis cannot be distinguished clinically from a poliomyelitis, and also the familial cases where a poliomyelitis is the picture in one child, encephalitis in another. Here the inflammatory reaction spreads throughout the entire cortex, as in the cord. The central and frontal gyri, the basal ganglia, the internal capsule, and centrum ovale are most frequently involved.

Here, in addition to headache, stupor, and convulsions one encounters spastic palsies, hemiplegic or diplegic in type, usually associated with bulbar palsies.

These cerebral forms are by no means so rare, they are often fatal. They are also rare in experimental monkey poliomyelitis. Müller is inclined to regard the spastic palsies that occasionally occur as due to pontine rather than to motor cortex involvement, whereas Wickmann lays particular stress upon the probabilities of their cortical origin.

5. **Cerebellar Forms.**—Medin described forms in which the patients showed ataxia in walking, with staggering or ataxic gaits, and others behaving like Friedreich's disease of acute onset. These forms are closely allied to the bulbar and pontine types, and also may be occasioned by extension of the disease to the cerebellum. The latter structure is almost always involved to some extent in this disease.

6. **Meningitic Forms.**—Here meningeal symptoms occupy the foreground. Headache, vomiting, pain in the neck with stiffness, Kernig's sign, stiffness of the back, opisthotonos, convulsions, strabismus, somnolence, and unconsciousness are present. These cases either then develop marked spinal and bulbar symptoms of the ordinary type, or the symptoms recede with either minor residuals, eye palsies, etc., or go on to complete recovery.

7. **Polyneuritic Forms.**—The study of recent epidemics has shown the great frequency with which pain is found in the initial history. In many cases there are painful nerve trunks, with Lasègue's phenomenon, and great hyperalgesiæ over the entire body, resembling polyneuritis. Anatomically, however, marked neuritic changes are wanting. These cases Wickmann prefers to call neuritis-like.

8. **Vegetative Types.**—In many of the patients with severe mesencephalic or spinal involvements the vegetative nuclei are implicated with the well-known trophic and secretory disturbances. There are, however, a number of cases in which some of these nuclei, from the ciliary ganglion down to the sacrum, are alone picked out by the pathological process. There result a great group of minor cases with residual difficulties left in the vegetative arc reflex functionings. Various bladder and bowel, gastric and other syndromes are among these Acute appendicitis may be, for instance an initial poliomyelitic symptom of this group.[1] Diabetes mellitus has been observed, as also in epidemic encephalitis, as resulting from mid-brain pathway inter-

[1] Dubs: Schweiz. med. Wchnschr., 1921, No. 11, p. 248, reports three such cases. Also see studies of Levy, Dresel, v. Bergmann, L. R. Müller and others on various metabolic anomalies seen in poliomyelitis and encephalitis.

ruptions. Some of these are confused with so-called neurotic or psychogenic cases.

9. **Incomplete or Minor Forms.**—The study of the recent epidemics has shown that in a number of patients the illness began with the characteristic symptoms of poliomyelitis, and then went on to recovery without any palsies. In others again, widespread, though mild palsies with hypotonia developed and complete recoveries occurred within a short time. Wickmann brought these facts into prominence, and showed that these were to be regarded as abortive cases. The most frequent forms under which these cases develop are (1) that of a mild meningitic type with the usual prodromal signs, associated with the neck symptoms, stiffness, pains, sometimes opisthotonos and the like; (2) cases with the symptoms of a general infection only; (3) cases running a course like an influenza; (4) cases with marked gastro-intestinal signs.

Wickmann has estimated that at least 15 per cent. of all the cases can be grouped under this rubric, while Müller believes them to occur much oftener, indeed, more often than the usual type. Their significance in the epidemiology of the disease is great, because it is highly probable that its spread may be conditioned by these ambulatory abortive cases. They are more frequent in children, but may also be present in adults. If Müller's stand be correct, viz., that they occur oftener than the well-developed forms, their importance from this standpoint is enormous. The question of rarity of the disease after puberty may be solely conditioned by the fact of previous minor attacks in many individuals. Müller states that in the study of small epidemics he has found that in the abortive cases there is a marked tendency to show the same symptoms in their epidemic extension. Thus gastro-intestinal cases give rise to gastro-intestinal cases, respiratory to respiratory, meningeal to meningeal, etc.

Diagnosis.—Sporadic and epidemic polioencephalomyelitis may be conditioned by the same etiological factors although other causative factors may cause rarer sporadic cases. If an epidemic is in progress, particular attention should be given to all acute infections, whether they show marked palsies or not, and careful examination be made of nerve tenderness, hypotonias, tendon reflexes, and limb motility. Most cases develop either in the guise of general infections with temperature, or with distinct local symptoms, either in the respiratory or gastro-intestinal tracts, or in the meninges. Influenza, polyneuritis, angina, bronchitis, gastro-enteritis, typhoid and epidemic cerebrospinal meningitis come into review. A marked general hyperesthesia, and a distinct pathological tendency to perspiration is significant. The careful studies of Peabody, Draper and Dochez suggest the general blood picture of an acute infection. There is a general polymorphonucleosis, with a fairly constant lymphocyte increase. 30,000 white cells per cu. mm. is not unusual, the polynuclears often using 10 to 15 per cent.; a diminution in lymphocytes, 15 to 20 per cent. may also be present. Sleepiness, to drowsiness of the children, during

the day, wakefulness, and fretfulness at night, easy fatigability, weakness of the extremities, loss of muscular tonus, especially in the abdominal muscles, with meteorism and loss of the abdominal reflexes, point to poliomyelitis. An early lumbar puncture will resolve many of the difficulties. The cerebrospinal fluid is rarely normal. There is an increase in the cells, sometimes to high numbers, chiefly 70 to 80 per cent. polynuclears. Later the lymphocytes are increased markedly, chiefly the mononuclears. Influenza is separated with considerable difficulty, especially since recent epidemiological studies of influenza leave one seriously in doubt as to just what influenza is. It certainly is not only a reaction to the Pfeiffer bacillus. Influenza is a highly complex problem only just begining to be understood.[1]

Polyneuritis also offers particular difficulties. This is rare in children apart from diphtheritic neuritis, and is usually quite symmetrical in its development. The time needed for development of the palsies is longer, the pains are more persistent, there are usually more objective sensory disturbances, particularly deep sensibility, and early edemas are more frequent.

In diphtheritic neuritis, cardiac irregularities are the rule, in poliomyelitis the exception; the palsies of the palate are further characteristic in the former.

Pure neuritic-like forms of poliomyelitis are sometimes present, but there is here more tendency for a mild dissociation syndrome, diminution of pain and temperature sensibility. Neuritis is more apt to include all the forms of sensibility, or gives a diminution in touch (epicritic) with an increase in pain sensibility. In very small children these differences are difficult to bring out. Williamson lays stress upon the loss of bony sensibility in neuritis, whereas in poliomyelitis it is rare.

Further differentials from Wernicke's polioencephalitis superior, myelitis, hematomyelia, meningitis, botulism, myotonia congenita, hysteria, Parrot's palsy in hereditary syphilitics, etc., must be sought for in monographs. The separation from epidemic encephalitis opens up a large problem in contemporary epidemiology. If v. Economo had not called his cases "sleeping sickness" it is not impossible they would have been called poliomyelitis.[2] Draper studying the English cases of encephalitis did call some poliomyelitis, but was in doubt about others. Amoss[3] and others believe they have established specific immunological distinctions. The blood serum of convalescent cases of poliomyelitis whether in man or monkey possesses a neutralizing power apparently absent in epidemic encephalitis cases.

In the meningitic forms, and in many of the ordinary spinal cases

[1] Crookshank: Influenza, London, 1922.

[2] For complete discussion of this important problem see recent work by F. G. Crookshank on Influenza, London, 1922. In the Vienna epidemic (1920) of encephalitis infiltrative changes in the spinal cord were found, but rarely as intense as those seen in poliomyelitis (Cf. Economo. Wien. klin. Wchnschr., 1920, p. 329; Münch. med. Wchnschr. 1919, p. 1311).

[3] Jour. Exp. Med., 1920–1921.

the separation from spinal or tuberculous meningitis is very difficult. The lumbar puncture here usually clears up the diagnosis. Clinically, the more marked mental symptoms, the marked degree of stiffness of the spinal column, Kernig's sign, and at times papillary edema, ear complications, and herpes, all speak for meningitis. In tuberculous meningitis the spinal fluid findings and the longer course usually establish the diagnosis.

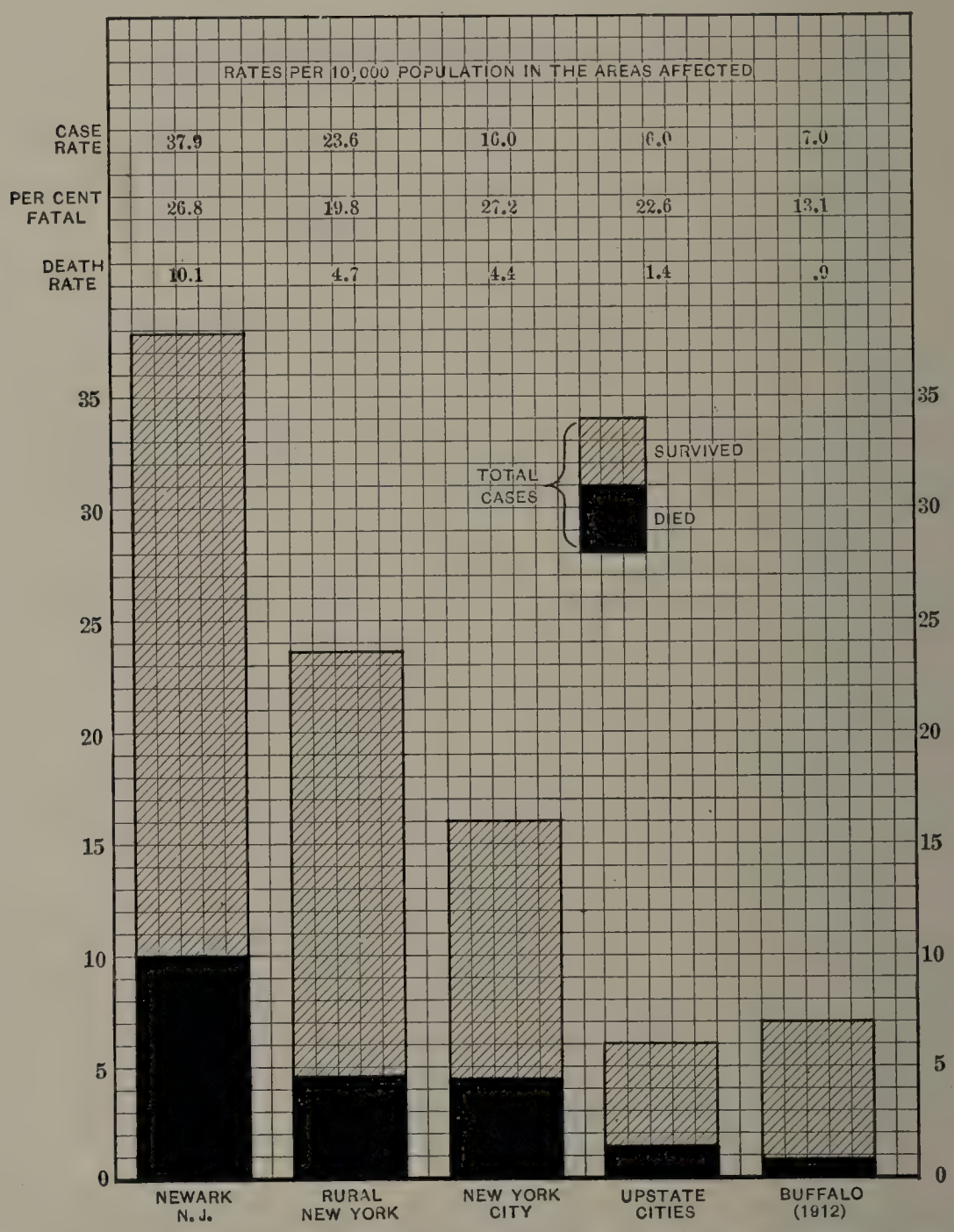


FIG. 274.—Relative severity of poliomyelitis epidemic of 1916 in certain areas, judged by the comparative incidence and death-rates recorded. (Courtesy of Dr. Mathias Nicoll, Jr., New York State Board of Health.)

Prognosis.—The older teachings that the disease is rarely fatal and always shows persistent palsies, must be modified in both directions. Many fatal cases do occur, and complete recoveries are frequent. In certain epidemics the mortality is very high (42 per cent.), in others only 10 per cent., counting only those patients with evident palsies. In the New York epidemic of 1907 the mortality was approximated as 5 per cent. In that of 1916 the mortality was much higher (20 per cent.).

If the very mild cases are included the percentage falls markedly. The mortality is higher in the older patients, as high as 50 per cent. in one epidemic (Lindner and Malley). The period of danger lies usually in the fourth and fifth day of the disease. In the second and third week bronchopneumonia is a dangerous complication.

As for complete recoveries, they have varied from 10 to 50 per cent. in the various epidemics, and are more common in younger children than in those over fourteen years of age. (See Wickmann.)

From the standpoint of electrical prognosis, the older views are certainly false. The whole subject is in need of entire revision. A total loss of faradic excitability after a week is no certain criterion of permanent palsy, as has been taught by Oppenheim and others.

Reparation takes place most rapidly in the first six months, but continues throughout a year or more, and with continuous, rational treatment weak and paralyzed muscles will continue to improve for many years.

Treatment.—Prophylaxis, treatment of the acute stage, and of the chronic stages are to be distinguished.

Prophylaxis.—Isolation and disinfection are as yet unprecise in their application. The mode of transmission of the virus is as yet unknown, and whereas there is little difficulty in isolating the severely attacked, the abortive cases are not, and rarely can be, properly regulated. That abortive cases do carry the disease seems definitely proved. The proper length of time for isolation has not been determined. Wickmann regards three weeks as sufficient, Müller eight weeks. As yet the data do not permit definite counsels.

The virus in monkey poliomyelitis seems to have been found in the nasal mucous membranes and in the feces. Hydrogen peroxide, 1 per cent. solution, and menthol solutions can be used for the former, formaldehyde or carbolic acid for the latter. Special attention should be directed toward the pocket handkerchief.

Rooms may be disinfected with formaldehyde. Preventive inoculation, analogous to Pasteur rabies treatment, may become practicable in time.

Acute Stage.—Absolute rest in bed is to be enforced, and if an epidemic be in progress, children with mysterious diarrheas, pseudo-influenzas, mild neuritic pains, etc., with fever, should be kept in bed, even after they appear to have recovered. The patients who have been sick for a couple of days, and who get up and around, and are then suddenly stricken down, are in reality very numerous. Foresight here cannot be overvalued.

Counter-irritation to the spine, by mustard plaster or other means; prompt catharsis, first by enema and then by saline cathartics, should be employed. The motor restlessness and pain are best controlled by analgesics, and the various salicylate preparations. Codeine may be employed, but its inhibitory action upon the bladder should not be overlooked. Lumbar puncture is often of signal service.

Diaphoresis by hot packs is helpful for the pains, muscular soreness,

and, possibly, in aiding elimination. Frequent treatment by free use of deep, warm baths, 102° to 104° F., is highly desirable. The warm water relieves the sense of pain and the irritability markedly, and is greatly appreciated by the patient. The baths can be repeated every three or four hours; the time in the bath is from ten to twenty minutes. On taking the child from the bath, he should be rolled in a blanket and dried in bed. Continuous warm baths, 94° to 96° F., are worth trying.

In the later stages the bath treatment is invaluable for straightening out the contracted limbs and aiding in active motion.

Urotropin may be administered. It is thought to reduce to formaldehyde in the cerebrospinal fluid in sufficient amounts to act as an antiseptic. The hypothesis lacks definite experimental confirmation.

The use of the serum of those who have had an attack of poliomyelitis, preferably recently, has seemed to be of service in some patients. The best results have seemed to follow the combined interspinal and intravenous injections of the serum. It is of service only in the preparalytic stages. Horse sera are being evolved. Parenteral protein treatments have also their advocates. What the value of these different efforts may be is not yet (1927) decided.[1]

Certain very severe and apparently fatal cases may be saved by continuous artificial respiration with oxygen administered by pressure apparatus, and in low blood-pressure and in threatened respiratory paralysis cases, adrenalin by mouth, injection, or by rectum has definite value. 3 c.c. to 1 to 1000 solution, every four hours, diluted with 2 c.c. salt solution by intraspinous injection is Meltzer's recommendation.

Chronic Stages.—After the acute stage has passed there is the long, hard stage, lasting for weeks or months or years, of partial or total paralysis, great weakness, extreme tenderness, nervous irritability, and muscular pains, with gradually developing deformities.

The diet should be full and regularly administered. A careful inventory should be made of every affected muscle, both in terms of its functional capacity, and its electrical excitability, the milliampères necessary for bringing about contraction being carefully noted. Full measurements of the limbs should be taken. Careful note must be taken of the position of the limb in bed, and appropriate support given to relieve pain, avoid stretching, and diminish contraction. Bedsores, sore heels, elbows, etc., need very careful treatment.

Chief reliance is now laid upon massage, passive motions and resistance motions. Here the deep, warm bath is invaluable, serving to help the motions of the limbs, and to straighten out the contractions. Movements in water are more easily performed, and small children can play in the deep tub for hours. Special exercises should be planned, both for the water and in bed. Special calisthenics must now be developed, depending upon the muscle group involved. Especial emphasis must be laid upon the patient's conscious and voluntary participation

[1] Rurähl, loc. cit.

in these exercises. The best method to carry these out is by the Mensendieck system which is a scientific type of muscle kinetics.

Electricity is of almost no service whatever. The physiological stimulus of muscles comes from the cerebral cortex. Hence muscle exercises, controlled by the patient's active desires, not formal calisthenics, is the best type of training.

Particular attention must be directed to the mental life of the child. He is apt to be morose, reticent, shy, and resentful, becomes very sensitive if his deformity be marked, and selfishness and exactingness develop easily if pampered because of his weakness.

In the later stages, and for those old enough, swimming is the best exercise.

The correction of the deformities is a matter for the orthopedic surgeon. Mechanical aid should be given as soon as possible, if it carries out a progressive therapeutic principle. Operative procedures should be conservatively considered, and not used too early. These patients make wonderful recoveries unaided, or by the persistent use of the Mensendieck exercises. These exercises proceed on the basis of conscious effort to develop movement in the paralyzed limbs first analyzing the complex function of a group of movements and picking out the components in the group least affected and developing them. In many individuals there are sound muscles or muscle fibers in a group loss. Mensendieck would carefully isolate all the components and educate the patient to individualize on each and every muscle function through cortically (consciously) started stimuli. This analytic and psychological method is far ahead of the usual routine calisthenic methods so widely employed. Many operative procedures are, however, imperative. Nerve splicing, and tendon splicing have their special indications.

Electrical therapy is of use only as a bridge to gap the period after the first onset until such time as definite willed movements can be started.

Drug therapy is of purely symptomatic value. Iron, strychnine, calcium are the most useful remedies, and meet special indications.

PRIMARY PROGRESSIVE MUSCULAR ATROPHIES.

The spinal motor neuron is differentiated into three parts, termed the anterior horn cell, the motor nerve fiber, and the muscle plate. It would be a great advance toward the simplification of the vexed questions regarding this group if a clinical division could be made in strict accordance with the pathological alterations of these parts. To a certain extent only is this justified. For categorical purposes then one can distinguish:[1]

1. The progressive nuclear atrophies, or myelogenous or myelopathic types: *Etiology:* Heredity, trauma, cold, toxins, infections. *Anatomy:* Primary degeneration of the anterior horn cells, with

[1] Kügelgen: Beiträge zur neuralen progressiven Muskeltrophien, Arch. f. Psych., 1909, 45, 944.

secondary fiber or muscle changes. *Forms:* Infantile, Hoffmann-Werdnig; adult, Aran-Duchenne; mixed forms.

2. The neural neuritic, or spinal neuritic atrophies: *Etiology:* Heredity. *Anatomy:* Primary degeneration of the motor nerve fibers with secondary ganglion, cord, and muscle changes. *Forms:* Peroneal-arm type (Charcot-Marie-Tooth); tabetic type (Dejerine-Sottas); peroneal type and arm type (Sainton and Haenel).

3. The muscular dystrophies or myogenic types. *Etiology:* Heredity Possible vegetative nerve disorder (endocrinopathic). *Anatomy:* Primary changes in muscles. *Forms:* Hereditary (Leyden-Möbius); juvenile pseudohypertrophy (Landouzy-Dejerine); and mixed forms (Erb-Zimmerlin).

The separation of these forms has occupied the attention of neurologists since about 1849 when Duchenne first attacked the problem of dismemberment of this large group with muscular wasting.

For many centuries clinicians had described progressive muscular wasting. Pictures and images in stone and wood of the muscular atrophies and muscular dystrophies dating from the fifteenth, sixteenth and seventeenth centuries are in existence. Van Sweiten, Abercrombie, and others gave general descriptions. This group was first broken into by Duchenne, in 1849, by the loose description of a special type, which a year later Aran (1850) supplemented. Cruveilhier, in 1853, and Luys, in 1860, sharpened the picture somewhat by their demonstration of the exclusive implication of the anterior horns. In 1865 Charcot isolated the amyotrophic lateral scleroses within the group. Duchenne himself had, in 1853, also set aside the pseudohypertrophies, the muscular features and varieties of which were later demonstrated by Eulenberg (1866) Charcot, Leyden, and Dejerine. Wachsmuth, in 1864, recognized the bulbar forms. Finally, Duchenne also called attention to the presence of sensory anomalies in certain of his 1853 studies on the atrophies; these were for the most part syringomyelias, which Kahler and Schultze separated definitely in 1882. Another small group were the neuritic atrophies, first clearly recognized by Dumesnil (1864). Thus it took thirty years for the sorting out of this medley of muscular atrophies; the groups as they are at present recognized, at least two of which, syringomyelia and amyotrophic lateral sclerosis, being entirely set outside of the muscular atrophies *per se*. One result was that the original disorder of Aran-Duchenne was so much reduced that finally Marie, in 1894, tried to sweep it away entirely, but his iconoclasm has not yet been justified.

In this volume the muscular dystrophies are removed entirely and treated as vegetative nerve disorders of the muscle (see Part I), rather than as disorders arising in the spinal muscle synapses.

Group 1. The Progressive Nuclear Atrophies: (1) Spinal, (2) Bulbar, and (3) Mesencephalic Forms.

1. **Spinal.**—Three main forms are here to be recognized. (*a*) Chronic poliomyelitis *per se*, with lesions limited primarily to the anterior horn

cells; (*b*) Aran-Duchenne's disease, progressive spinal muscular atrophy and (*c*) infantile hereditary types—Werdnig-Hoffmann.

The two former types are much alike, and many authors maintain their identity. They are here considered separately, since such a course seems justified clinically if not pathologically.[1]

(*a*) **Chronic Poliomyelitis.**—Here are grouped those rare subacute or chronic muscular atrophies usually occurring in the late years of life, in which certain toxic agents seem to account for the disorder. Such toxic factors are lead, diabetes, syphilis, and other unknown toxemias. Heredity is sometimes present.[2] Severe shell explosion injuries may cause it.[3]

The anatomical lesions are a progressive destruction of the anterior horn cells, with chronic vascular alterations.



FIG. 275.—Chronic poliomyelitis showing atrophy of hands.

Symptoms.—There is a progressively developing flaccid paralysis, with antecedent weakness and diminution or loss of tendon reflexes, but without sensory or trophic signs. The nerve trunks are not tender or swollen. Fibrillary twitching appears, the myotatic irritability is increased, reaction of degeneration is present. There are no disturbances of the rectal, vesical, or genital functions, and the bulbar nuclei are rarely involved.

[1] Marburg, Lewandowsky: Handb. d. Neurol., 1911, **2**, 280, for complete literature. Lövegreen: Zur Kenntnis der Pol. acuta und chronica, Karger, 1904. Koopman: Deutsch. Ztschr. f. Nervenh., 1926, **95**, 165.

[2] Dana: Familial Form of Progressive Spinal Myopathy, Jour. Nerv. and Ment. Dis., 1914, **41**, 681.

[3] See Aran-Duchenne section.

The localization may vary; the legs or arms first showing weakness or atrophy, sometimes one arm or both, one arm and one leg, or both legs. The atrophy and palsies usually take place slowly, with at times periods of more rapid development, and also stationary periods during which no advance is made for years, or the patient slowly recovers.

The distribution of the atrophic muscles is characteristic. Certain muscle groups are spared. The arrangement is segmental, not radicular. The triceps may escape with all the other muscles of the arm attacked, or the flexors of the fingers may be intact. In the legs the tibialis anticus may stand out unimpaired. The course is usually

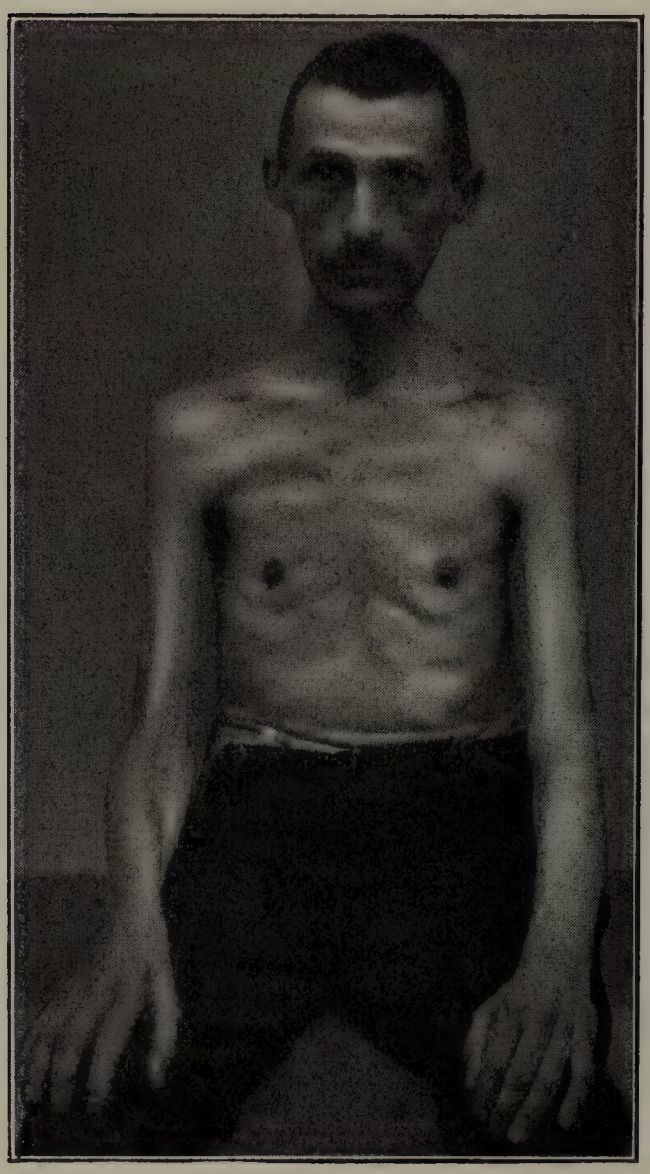


FIG. 276.—Primary nuclear atrophy. (G. M. Hammond.)

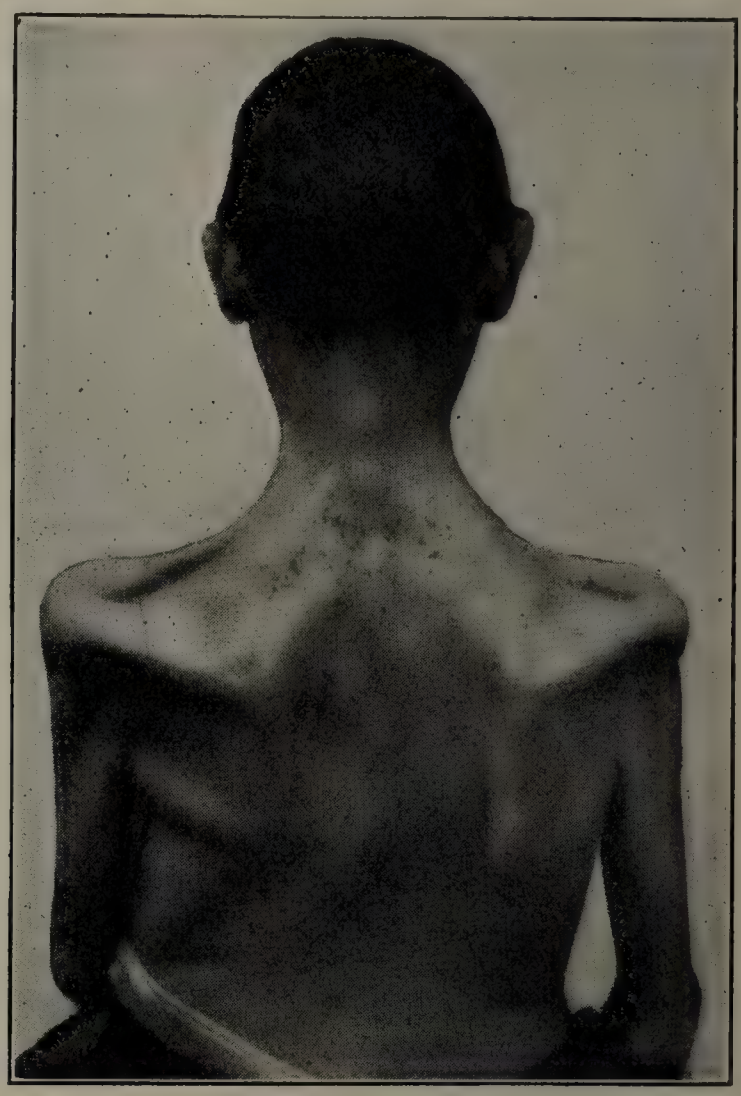


FIG. 277.—Primary nuclear atrophy. (G. M. Hammond.)

very chronic, lasting years. Subacute onsets are more favorable. Some patients recover, especially those with suspicion of a radiculitis or a neuritis. In the progressive cases the patients at times develop bulbar symptoms, and die of aspiration pneumonia, or weakness.

Treatment.—Rest, hydrotherapy, gentle massage, electricity, overfeeding are the chief indications. Syphilitic cases need specific therapy.

(*b*) **Aran-Duchenne Type.—Progressive Muscular Atrophy.**—*Etiology.*—Here heredity may play a part in the development of a familial form, seen in infancy, and also in adults (Bernhardt). It may follow acute poliomyelitis. Other factors, as toxemias, exposure to cold and

wet, overexertion, are not definitely established. In the world war shell concussions, probably causing minute hematomyelias, possibly vacuum caisson changes, have resulted in diffuse disseminated types of muscular atrophy.[1] Syphilis may also be an etiological factor.[2] Occupation atrophies are at times incorrectly included here. Pathologically there is degeneration of the anterior horn cells and fibers, with some secondary degenerations in the anterolateral columns.

It is a rare disorder. The syndrome begins very slowly, the upper extremities are attacked, and rarely the lower. The smaller muscles of the hand are initially affected. Weakness, atrophy and flaccid paralysis are the sequence. Fibrillary twitchings accompany the atrophy and there is reaction of degeneration in the muscles involved. Sensory and trophic disturbances are lacking and the tendon reflexes are diminished or lost. Early overexcitability of the tendon reflexes may be noted in both of these forms.

Oppenheim has called attention to the tendency for these atrophies to jump from one muscle group to another. The atrophy is very gradual; the disease advancing slowly for years. Remissions occur, but recoveries are very unusual.

The distribution of the affected muscles varies considerably. Death usually results from paralysis of the muscles of respiration.

Diagnosis.—The separation of these two forms is at times impossible. There is a tendency for the former to advance more rapidly, to involve the lower limbs, to advance to the medulla, to show longer remissions, and to recover. Further, there is a greater tendency to segmental distribution in the muscle groups affected in the former type, with marked loss of power followed by atrophy. In the latter type the atrophy seems to precede the palsies, and the patients are often very adept with their residual muscles.

The sensory disturbances of occupation atrophies, of neuritis, of syringomyelia should exclude these, while the increased tendon reflexes of amyotrophic lateral sclerosis eliminates that disorder. Radiculitis needs separation. Here the atrophies are apt to be unilateral, and are radicular in distribution. There is a low-grade neuritis, and the Dejerine-Klumpke syndrome appears in the cervical type.

Treatment.—This may be treated as the preceding affection, but such therapy seems of little avail. Strychnine, arsenic, and fats are indicated. Newer ideas must be gained if any effective control of this disorder is to be hoped for; the older methods are useless.

(*c*) **Infantile Hereditary Forms.** (Werdnig[3]-Hoffmann[4]).—These are rare hereditary types, beginning in infancy, first described in 1891. The disease begins gradually, usually in the pelvic girdle and thighs

[1] Levi: Rev. Neur., 1918, p. 476. François: Rev. Neur., **23**, 2, 494. Claude Presse méd., October 11, 1915; Soc. Méd. d. Hôp., February 11, 1916.

[2] Souques: Nouv. icon. de la Salpêtrière, 1915, **27**, 297. Lerouge: Thèse de Paris, 1913.

[3] Archiv. f. Psychiat., vols. **22**, **26**.

[4] Deutsch. Ztschr. f. Nervenh., vols. **3**, **10**, **18**. Pette: Ztschr. f. d. ges. Neurol. u. Psychiat., 1924, **92**, 324.

(iliopsoas, quadriceps femoris). Later the back, neck, and shoulder girdle are involved. Finally the distal extremities are involved. The intercostals and diaphragm are often affected. Occasionally the bulbar muscles atrophy. The atrophies are usually symmetrical, often associated with apparent hypertrophy (adiposis).

There is gradually increasing paresis and loss of skin and tendon reflexes. Fibrillary twitching seems to be absent and a peculiar trembling of the fingers is described. Scolioses, talipes, and other contracture states develop. These little patients often acquire unique modes of locomotion. Faradic currents are borne better than galvanic currents, but reaction to both is diminished or lost. The course is progressive, rarely stationary.[1] Mental reduction is not usual. They may be identical with Oppenheim's amyotonia congenita.

2. **Bulbopontine Types. Chronic Progressive Bulbar Palsies.**—In these forms the progressive atrophy is limited to the muscles of the face, tongue, palate, and larynx.

Etiology.—Little is known of the causative factors in most of the cases. Certain toxic factors—lead, syphilis—have seemed to play a role at times; constantly recurring electrical shocks were a factor in one patient personally observed; the majority give no clue as to etiology. Some follow as late atrophies on an old poliomyelitic or epidemic encephalitic foundation. Neuroendocrine cases are known and even psychoneuro-endocrine factors have been observed.[2] A few cases are observed in childhood, still fewer in adult manhood; the majority occur after thirty-five years.

Symptoms.—There is a slowly progressive weakness of the tongue and muscles of the cheeks and lips, followed by fibrillary twitchings and slowly progressive atrophy. Speaking becomes fatiguing, and slight changes in the voice become apparent. In eating the patients find they must use their fingers in dislodging food from behind the teeth in the cheek. Gradually increasing difficulty in swallowing is observed, and difficulties in breathing appear. Finally the speech becomes alling, dysarthric, the lingual letters *d, t, l, r, n, s,* are first slurred over, then the labial letters *b, p, f, m, w, o, v.* The laryngeal weakness causes hoarseness, monotony, and finally aphonia. The pharyngeal muscles and those of the tongue do not act in concert, and liquid food regurgitates through the nose.

The face becomes atrophied below, the lips thin and folded. The tongue is atrophied, wrinkled and tremulous and finally lies inert on the floor of the mouth. The patients cannot protrude the lips, nor whistle. The temporals and masseters also become affected, and the movements of the jaw become impossible. The masseter, pharyngeal, and vomiting reflexes are absent. The sensibility is intact. The upper face region, including the levator palpebræ, is rarely affected.

Vasomotor disturbances are occasionally observed. Vagus involvement causes cardiac irregularity.

[1] Batten: Lancet, June 3, 1911. Senator: Charité Annalen, 1902. Lange: Deutsch. Ztschr. f. Nervenh., 1910, p. 40.

[2] Hernandez: Prog. de la Clin., 1921, **9**, 737 (Spanish).

The usual progression is from the tongue to the lips, then to the other facial muscles, and finally to the fifth, and hypoglossal and vagus muscles. An apparent increase in saliva, an annoying symptom, is largely dependent upon the inability to swallow.

The usual course extends over four or five years, death taking place most frequently from pneumonia or from inanition.

Pathology.—Here one finds changes in the bulbar nuclei, precisely analogous to those found in progressive spinal nuclear atrophy. The cortico-bulbar tracts may be involved somewhat, but rarely to the degree found in amyotrophic lateral sclerosis, yet intermediary forms are to be expected.

Diagnosis.—Unusual types of gliosis, of multiple sclerosis, tumors, gummata, arteriosclerosis, general paresis may cause somewhat similar pictures at first, but these also soon show other symptoms, indicating that the lesion is not confined to the nuclear structures. Pseudobulbar palsy shows palsied muscles, still electrically reflexly excitable, without atrophies. Myasthenia gravis shows the typical electrical myasthenic reaction.

Prognosis.—In syphilitic cases the prognosis is not good. Most cases have a serious prognosis but newer methods have helped.

Treatment.—One should always be on the lookout for specific causes of the syndrome, especially in the initial stages. Here a nihilistic therapy will be recognized too late for recovery.

Galvanization has seemed to help the swallowing of some patients. Atropine, hyoscyamus, etc., or other related drugs can be used to control the salivation, and the pains and discomforts of coughing, dyspnea, etc., relieved by narcotics. Specific therapy is valuable in the Wassermann positive cases and organotherapy is valuable in some neuroendocrine cases.

3. **Pontomesencephalic Forms.—Chronic Progressive Ophthalmoplegia.**—Chronic progressive ophthalmoplegias as a part of an amyotrophic lateral sclerosis, of tabes, of general paresis, multiple sclerosis, tumor of corpora quadrigemina, or other organic disease are not rare. As pure, chronic nuclear affections they are infrequent. Uthoff credits them as high as 14 per cent. of the chronic ophthalmoplegias.

There is beginning weakness of the eye muscles, usually the external rectus, with slight internal strabismus, frequently worse at night. Diplopia is not usually present, because of the gradual development of the disorder, and its symmetry. In hereditary types, in infants, binocular vision may not have developed.

The internal eye muscles are usually intact, although occasionally irregular pupils occur; light-immobile pupils are not present although loss of accommodation movements has occurred. Ptosis is not infrequent; usually worse on one side.

In a completely developed case the Hutchinson face develops; bilateral ptosis with inability to move the eyeballs.

The disorder is usually progressive. Starr has described a stationary condition, and some patients recover entirely.

Treatment.—When a Wassermann test has revealed a positive reaction, mercury and iodides are indicated; otherwise general tonics, strychnine, iron, arsenic, and general dietetic treatment directed to the reduction of arteriosclerotic changes. Endocrinopathic stigmata should receive careful attention and glandular therapy attempted if such are present.

Group 2. The Neural, Neuritic, or Spinal Neuritic Atrophies.

Hard-and-fast lines cannot as yet be drawn between the neuritic atrophies and certain nuclear atrophies on the one hand, nor certain dystrophies on the other. Jendrassik, (1911), speaks of them as "dystrophy forms with degeneration of the peripheral nerves."[1]



FIG. 278.—Charcot-Marie-Tooth disease. Atrophy of the legs below the knees and of the arms below the elbows. (Starr.)

Many forms of neural atrophy have been described. The most characteristic are: (1) The peroneal-forearm type—Charcot-Marie-

[1] Willny: Ueber die neurale Form der progressiven Muskelatrophie, Deut. Zt. f. Nerv., 1924, **83**, 1.

Tooth; (2) the tabetic, or hypertrophic interstitial neuritic type—Dejerine-Sottas, Marie's familial form; (3) the peroneal type—Sainton; (4) the arm type—Haenel.

1. **Peroneal-forearm Type.**—Charcot, Marie (1886) and Tooth described this form which is characterized by muscular wasting in the distal parts of the extremities, from knee and elbow outward.[1]

Etiology.—Heredity is the chief factor recognized.[2] Eichorst found 13 cases in six generations and Haenel 32 in four, while others have found only 1. Syphilis may bring about a very similar picture.[3]



FIG. 279.—The neuritic form of muscular atrophy. (Spiller.)

Symptoms.—The feet are usually first involved. There is early equinus position from loss of power of extension of the great toe. Then the leg, from the knee down, thins and atrophies. The gait thereby becomes either widespread or steppage. A similar process

[1] Rev. de méd., 1881, 1886; Brain, 1887, **10**, 243. Spiller: Jour. Nerv. and Ment. Dis., 1907. Souques: Nouv. iconog. de la Salpêtrière, 1915, **25**, 175. Currie: Glasgow Med. Jour., 1927, **107**, 28, on state of bones. Davidenkoff: Rev. Neurol., 1926, ii, 447, on the influence of cold in causing transitory paresis in these cases.

[2] See Geneological Tree of Twenty Members in Maslianka: Thèse de Paris, 1913, and in Souques: loc. cit., 1915.

[3] Levi: Nouv. iconog. de la Salpêtrière, 1917, **28**, 260.

begins in the forearms, usually advancing from the muscles of the hand. Monkey hand, or main en griffe develops. Rarely the upper extremity atrophy precedes. The shoulder, arm, neck, back, hips, and thigh muscles remain intact.

Sensory changes are usually present, and slight pains, intermittent and lancinating in character, coming and going, hyperesthesia, paresthesia, hypesthesia, especially to cold, are present. Painful nerve trunks are occasionally found. Vasomotor disturbances are frequent. Cold, marble-like skin with increased secretion is observed.

The knee-jerks are often initially increased; later they are diminished or absent. The Achilles reflex is usually absent, as is the radial-periosteal reflex.



FIG. 280.—The neuritic form of muscular atrophy. (Spiller.)

Reaction of degeneration in the regions affected is the rule. Fibrillary contractures and muscle unrest are constant, often persisting during sleep. Tremors and choreic-like movements are not uncommon.

Course.—The disease usually begins in childhood, advances slowly, usually progressively, sometimes halting for long periods.

Pathology.—Since Hoffmann's description (1889) of a neuritis in the distal peripheral nerves, this disorder has passed as a mixed neu-

ritic atrophy, but later Siemerling, Gierlich,[1] Kügelgen,[2] Spiller,[3] and others have shown changes, not only in the peripheral nerves and muscles, but that there were extensive changes in the other parts of the nervous system, in the eighteen to twenty cases thus far autopsied (1910). These were parenchymatous and fatty degeneration of the muscles, ascending degeneration of the peripheral motor nerves, especially intermuscular branches, with chronic interstitial neuritis, degeneration of spinal ganglia, atrophy and loss of anterior horn cells, sclerosis of columns of Goll and Burdach, with changes in lateral columns and posterior roots.

2. **Tabetic Type** (Dejerine-Sottas[4]).—This is apparently a hereditary form, the symptoms beginning in childhood. The muscular changes are similar to those seen in the Charcot-Marie-Tooth forms, but the sensory nervous system is much more markedly involved. There are present pupillary changes—myosis, or even Argyll-Robertson pupils, beginning or complete Romberg, nystagmus, and ataxia. Pain is present. The peripheral nerves are enlarged and palpable. Reaction of degeneration was stated to be absent by Dejerine and Sottas.

It may easily be confounded with a juvenile tabes. Dejerine maintains that it has no relation to the Charcot-Marie-Tooth atrophies.

Other forms are described in which the atrophies are limited to the lower extremity (Sainton), to the upper extremity (Haenel), and to the femorotibial region (Eichorst). The transition types are many.

Diagnosis.—A complete separation of all the forms is not possible in the present state of our knowledge. The Wassermann reaction and cerebrospinal fluid examination will probably throw considerable light on the hypertrophic neuritic types of Dejerine, and may remove them from this group entirely; also cases described by Marie under a similar name.

For the classical cases of neuritic muscular atrophy there is little ground for confusion, but the numerous aberrant forms introduce difficulties.

The separation from the dystrophies is usually made on the ground of pseudohypertrophy and the absence of reaction of degeneration in these cases. Certain transition forms are undifferentiable.

Chronic polyneuritis is rarely hereditary (unless one follows Oppenheim in classing the neuritis atrophies here under consideration as hereditary chronic multiple neuritis). In polyneuritis there is rarely a club-foot, the progress is more rapid, and recovery is apt to take place. With tabes, only Dejerine's type can cause confusion. The age and heredity are the chief features. Newer studies on complement-fixation and spinal fluid are wanting.

In amyotrophic lateral sclerosis the increased reflexes, the extension to the bulbar nuclei, the spasticity, and rapid course are characteristic. Multiple sclerosis, syringomyelia, chronic poliomyelitis, myotonia, and hereditary ataxia occasionally call for differentiation.

[1] Arch. f. Psychiatrie, 1909. [2] Ibid. [3] Loc. cit.
[4] Arch. de neurol., No. 91, ser. ii, vol. 17; Rev. de méd., 1897; Rev. neurol., 1902–1906.

Prognosis.—Usually poor, but the disease is very chronic, and patients die after twenty to thirty years of intercurrent disorders. Sometimes stationary periods are met with.

Therapy.—Electricity, baths, massage, internal secretions, general tonics, and orthopedics are useful. A few cures are known.

AMYOTROPHIC LATERAL SCLEROSIS.

History.—The position of the progressive muscular atrophies with their many clinical variations, and the anomalous pathological findings is still in need of more detailed analysis. Charcot, in 1865, separated from this mass a special group which combined the features of a poliomyelitis and a lateral sclerosis. Atrophy with spasticity was the determining feature, and in 1872–1874 after previous studies with Joffroy and Gombault he gave it definite form. Dejerine later (1883) showed that the glossolabiolaryngeal palsies of Duchenne were a constituent part of the disease, and the work of Kojewnikoff, Marie, Roth, Florand, Holmes, Spiller, Probst, Raymond, Cestan, Marburg,[1] Matzdorf, v. Bogaert and others still leave this syndrome unexplained.

Etiology.—This is a comparatively rare disease. Little is known concerning its causes. It has been thought of as a primary atrophy of the motor system—an abiatrophy, but this is only pushing the explanation back a step further. It is most prevalent in early adult life, thirty to forty, but it is also known to occur in children (Erb-Seeligmüller), and Soques, Roussy, Raymond, Probst and others have described cases occurring in the fifth decade. Women seem to have the disease somewhat oftener than men, but the differences are not striking. The statistics of the Vanderbilt Clinic for ten years show the reverse conditions—11 men and 6 women.

Arteriosclerosis, infection, intoxication, syphilis have each been shown to play some role in the causation in some patients. The relationship of trauma is still unsolved. Bing has reported a case following an ascending neuritis of the median from trauma.[2]

Pathology.—The pyramidal system is chiefly implicated but other structures—posterior columns (Moeli, Margulies, Marburg), cerebellar pathways (Pilcz), posterior horn cells (Sarbo), are at times involved.

The cortico-spinal tracts are also degenerated. This degeneration can be followed to the Betz cells of the Rolandic cortex in practically all of the cases of any duration.[3] The frontal cortex may be involved. In some patients the degeneration in the pyramidal tracts has not been traced beyond the pons. (Oppenheim, Medea, Marburg.)

[1] Marburg: Lewandowsky's Handbuch. See Matzdorf (Ztschr. f. d. ges. Neurol. u. Psychiat., 1924, **94**, 703) for complete discussion of pathogenesis and literature to 1924 also Schmink and Heinze: Deutsch. Ztschr. f. Nervenh., 1924, vol. **83**, 76.

[3] Erb: Deutsch. Ztschr. f. Nervenh., 1911, vol. **44**. Bing: Rev. Suisse de méd., July 24, 1915.

[2] Gerber and Naville: L'Encéphale, 1921, **16**, 113. Probst: Sitz. d. math. natur. Klin., **3**, 193. Schroeder: Jour. f. Psych. u. Nervenh., 1910, vol. **16**.

Degeneration in the bulbar motor nuclei is the rule, and in the cortico-bulbar tracts, as was demonstrated by J. Dejerine in 1883. The third, fourth, and sixth nerve nuclei usually escape, though a few recorded degenerations here are known (Hoche, Pal, etc.).

Degenerations can also be traced in the corpus callosum. Following the degeneration there is some replacement sclerosis. This widespread degeneration in the motor system may be accompanied by other degenerations, however, although such degenerations seem to stand out less prominently in the clinical picture. Thus, degenerations in the lateral columns, Clarke's columns, posterior longitudinal bundles, spino-cerebellar tracts, and ventro-lateral ground bundles are recorded. Matzdorf describes changes in the globus pallidus, as did Pilcz[1] in the putamen.



FIG. 281.—Amyotrophic lateral sclerosis showing atrophy of the arms.

Symptoms.[2]—The disorder may begin in almost any motor nerve region: bulbar, cervical, dorsal or lumbar; more than one area may be initially affected simultaneously, but the usual beginning lesions are predominantly in the muscle groups of the cervical cord. The order of involvement is predominantly radicular.

The usual symptoms are muscular weakness, with wasting of the muscles, usually distally, with increased reflex excitability. The patients first note that the hands get tired, they are clumsy in dressing and undressing. Buttoning and unbottoning, sewing, writing, and the carrying out of the more delicate finger manipulations become increasingly difficult, first from fatigue, then from stiffness and motor palsy. Atrophy of the thenar and hypothenar eminences occurs, then

[1] Jahrb. f. Psych. u. Nervenh., 1898, vol. **17.**
[2] Büscher: Arch. f. Psychiat., 1922, vol. **66.**

progresses to the muscles of the forearm, arm, and shoulder-girdle. Weakness and stiffness then make themselves evident in the lower extremities, and a similar progression takes place. Then after months or a year or so the muscles of the lips, of the tongue, and swallowing and phonation gradually become implicated. The lips become thinned, (tapir mouth) the tongue atrophies, speech fatigue is followed by inability to speak; swallowing occurs with regurgitation, cardiac irregularities develop, and the patients die of aspiration pneumonia, weakness or other intercurrent affections.

With the atrophies there develops a gradually increasing spasticity. The tendon reflexes are increased, there is increased jaw-jerk, or mandibular clonus; the biceps and triceps, and scapular reflexes are increased, patellar clonus, increased knee-jerks, increased Achilles, ankle-clonus, Babinski and Oppenheim reflexes are manifest.

The muscles themselves show increased myotatic irritability, fibrillary contractions are common, or the atrophies may be so marked that flaccid conditions develop. The reaction of degeneration occurs.

Sensory anomalies are extremely rare. Pain is probably present at some period in many cases; it is that of fatigue or spasm, however. The bladder and rectal functions are not usually involved, and the majority of the patients show only the involvement of the motor neurons.

Mental symptoms are prominent late in the disease; increased emotionalism is usually present, and as the patients are often much depressed, the involuntary crying, which is very frequent, is augmented thereby. Van Bogaert has discussed these very fully.

As has been noted, the onset may be in any part of the motor system—thus the disorder may progress for some time, even to death, as a bulbar type of palsy, with increased reflexes in the motor cranial nerves; death occurring in some instances before there are any spinal symptoms. Again the distribution may be hemiplegic, and then triplegic, and finally quadriplegic and bulbar.

Again the disorder may bear the stamp of a lower extremity paraplegia, or an upper limb diplegia; again, whereas the distal muscles are usually initially involved, some patients show proximal atrophies in either upper or lower extremities; and the more cases reported the greater are the possibilities encountered in the way of anomalous localization.

But throughout, after a certain lapse of time, the combination of weakness, atrophy, increased reflex activity, without sensory changes, stamps the process as essentially a motor degeneration, and whereas one may even find the spastic phenomena antedate the atrophic changes, yet in the end the two level up, unless the course has been unusually active, and death results early from the disease or from intercurrent disorder. Here the pathological picture may seem to contradict the clinical findings, and the interpretation remains that the patient did not live long enough to show the classical syndrome.

On account of this polymorphism, certain authors have erected types which may be useful clinically, but which are rarely borne out, save for

certain periods, during the development of the disease. Thus Raymond and Cestan[1] make (1) *an ordinary spinal type* with the classical *main en griffe* of Charcot, increased reflexes, particularly in the upper extremities, less marked in the lower, with occasional failure of the Babinski reflex; (2) *labioglossolaryngeal type*—apparently more frequent in women—which begins in the muscles of the lips, tongue, pharynx, or larynx, occasionally with facial palsies, inability to close the eyes, etc., with fibrillary twitchings, lively masseter-jerk. Some of these, but not all, run a rapidly fatal course from four to nine months, and die before the lower neurons show their characteristic changes; (3) *an amyotrophic type* with pronounced atrophies which overshadow or mask the spasticities and the increased reflexes. These patients resemble chronic poliomyelitis, often for years, and constitute a most difficult group to differentiate. (4) *A spastic type,* in which the reverse condition exists, and increased reflexes and spasms, contractures, etc., antedate the atrophies. These resemble multiple sclerosis, lateral sclerosis, etc.

Finally, it should be recognized that most patients represent transition forms; they are not pure types, and the apparent clinical picture of amyotrophic lateral sclerosis is due to other than the heretofore held classical pathological foundations of this disorder.[2]

Course and Duration.—The average time of duration of some hundred or more cases analyzed is about two years; but this by no means tells anything about any individual case. Death in four months after the onset has been the result in a score or more of cases, some have persisted ten years, and a few questionably diagnosed cases even much longer. The rule is toward steady progression; there are few leaps and regressions, as is seen in multiple sclerosis, and so far as is known the disease is a fatal one. A few stationary cases are reported. Bulbar palsies, when not among the initial symptoms, usually develop before the second year, and are often the cause of death by choking, or dyspnea. Cardiac irregularities, pneumonia, exhaustion, secondary infections, these are the usual modes of death. Suicide is unusual.

Diagnosis.—The chief disorders needing separation are multiple sclerosis and progressive spinal muscular atrophy or chronic poliomyelitis. The former rarely shows atrophies, and the eye-grounds, ocular muscle signs, and characteristic tremors should separate this; the latter disorder when it shows increased reflexes, as is occasionally the case, is separated with great difficulty. Lumbar puncture has not as yet helped to nosology.

Treatment.—No specific is known. Overfeeding, light massage, much rest, avoidance of fatigue of affected muscles, caffeine, strych-

[1] Rev. Neurol., 1905.

[2] For more recent studies of this disorder see: Blaeden: Med. Jour. South Africa, 1917, **13**, 59. Hassin: Med. Rec., 1917, **91**, 228. Kempner: Med. Klin., 1918, **14**, 729. Hoffman: Deutsch. med. Wchnschr., 1918, p. 1151; München. med. Wchnschr., 1918, p. 1062. Rhein: New York Med. Jour., 1917, **105**, 915. Rossi: Cron. d. clin. med., 1917, **23**, 161. Throckmorten: Jour. Iowa State Med. Soc., 1917, **71**, 177. Gerber and Naville: l'Encéphale, 1921, **16**, 113. Matzdorf: Ztschr. f. d. ges. Neurol. u. Psychiat., 1924, vol. **94**. Marburg: Lewandowsky's Handbuch.

nine, are the only methods at present of service. Certain anomalous thyroid insufficiencies have been associated with progressive muscular wasting, and indicate a field for experimental therapeutics. Parathyroid myatonias, myasthenias, family periodic paralyses, etc., offer a suggestion in this line of a perverted parathyroid activity that may be seized upon for the study of the mineral metabolism, especially of calcium. Personal cases treated along lines suggested by the mineral metabolism of the body offer a glimmer of hope in a hitherto hopeless disease. Syphilitic cases need specific therapy.[1]

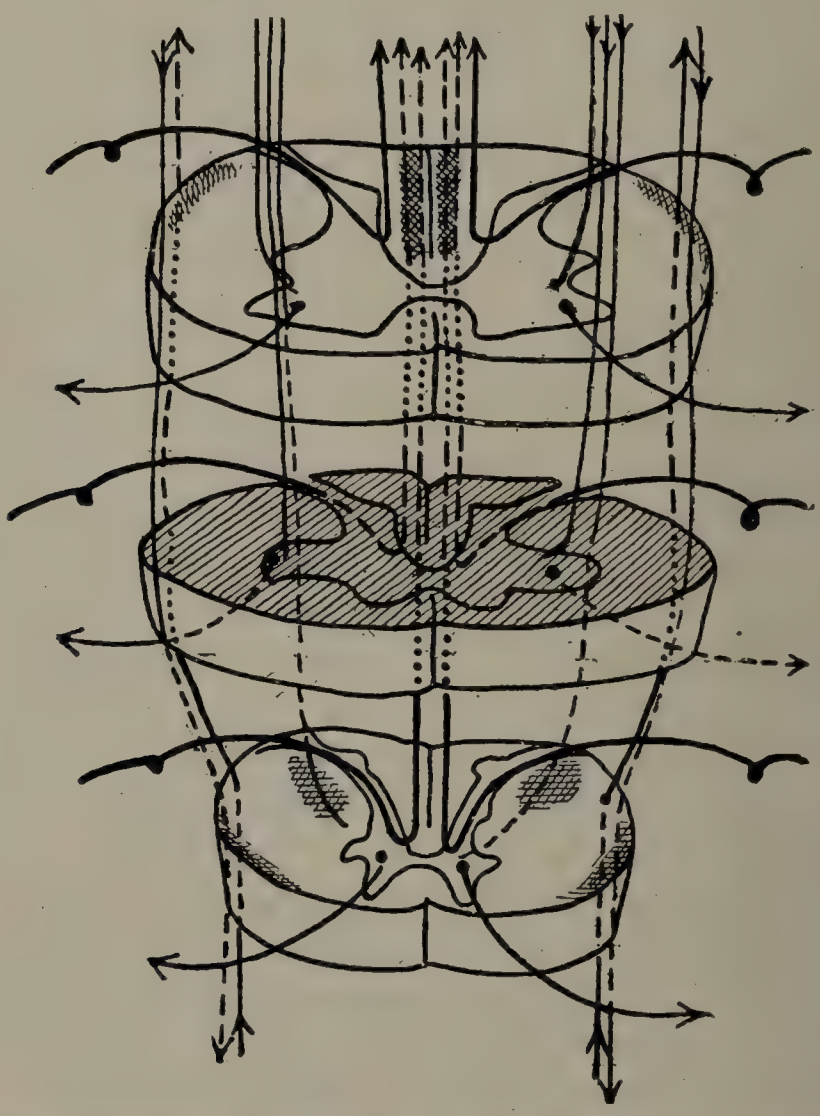


FIG. 282.—Total cross-section of spinal cord at the level of C_8 showing the results upon the long fiber tracts. Middle section indicates level of injury. Cross-hatching above and below indicate the degenerations or loss of function. (Veraguth.)

FRACTURE, DISLOCATION, CONCUSSION AND MYELITIC SYNDROMES.

Spinal cord injuries result from bullets, penetrating instruments, or from blows or falls.

Bullets and cutting instruments cause hemorrhage, with, as a rule, partial or complete severance of the cord, with septic infections of the cord and meninges. Blows and hard falls occasion fractures or dislocations, with crushing of the cord to a greater or lesser degree. Mild

[1] Two cases recently seen suggest important psychogenic factors. Whether such are post or propter hoc cannot yet be determined. In each libido blocking was evident and massive.

injuries may bring about bruising of the cord, or occasion minute hemorrhages within the cord or the pial or dual spaces only. Sometimes excessive exertion, long marching, severe athletic exercises, sudden spinal torsions, etc. may produce definite lesions. Dislocation of a vertebra practically always causes a crushing of the cord; the so-called dislocations without spinal cord injury are more apt to be wrenches of the vertebræ or very limited dislocations. Fractures may result with but few spinal symptoms; crushing of the cord causes

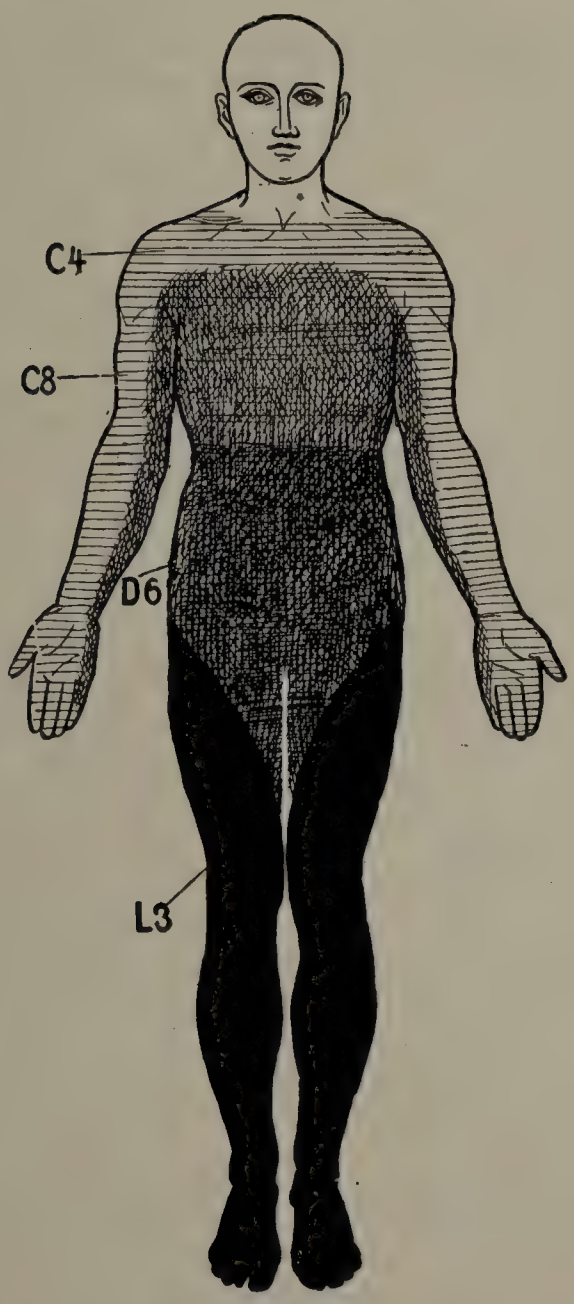


FIG. 283

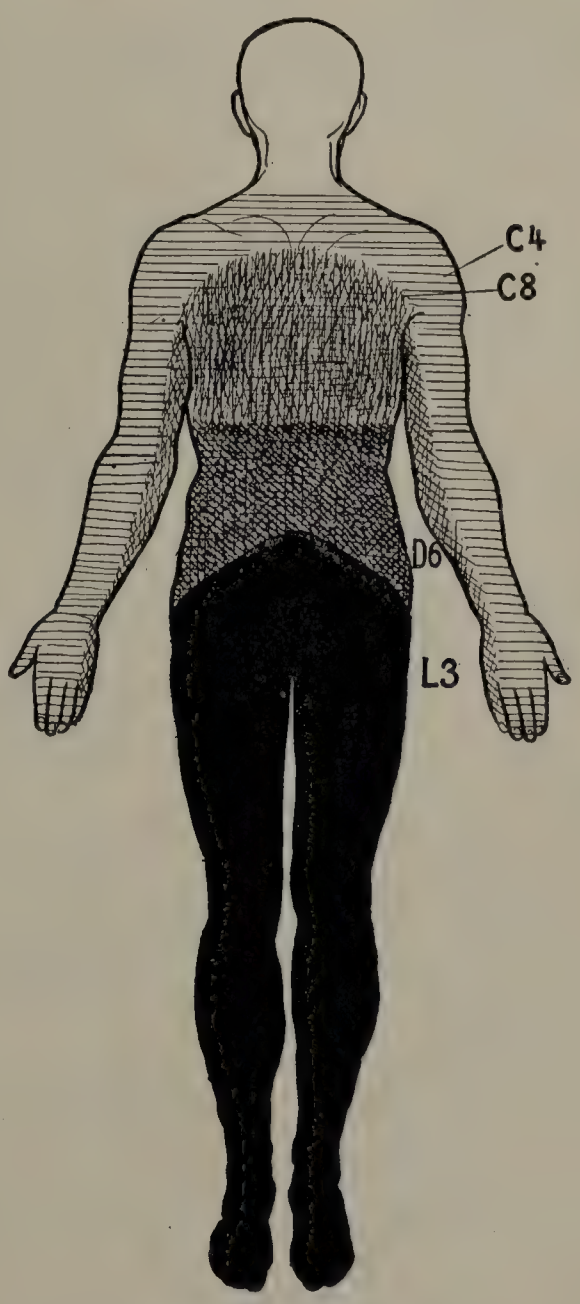


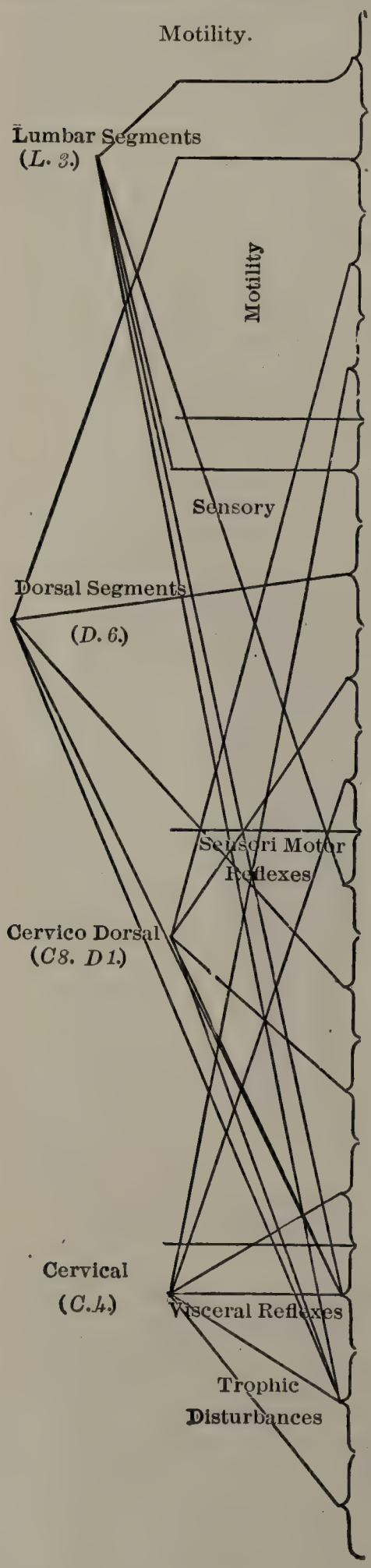
FIG. 284

FIG. 283 and 284.—Showing superficial sensibility disturbances in complete transverse lesions of the cord at the levels of C_4, C_8, D_6 and L_3, respectively. (Veraguth.)

more or less disintegration, usually accompanied by severe hemorrhages within or without the cord substance (hematomyelia, dural hemorrhage). Hematomyelia usually extends up and down the cord from the point of injury, sometimes involving several segments of the cord.

Symptoms.—Fractures or dislocations of the spinal vertebræ usually cause local deformity, much pain and muscular rigidity, particularly on motion. Roentgen-ray examination reveals the nature and extent of the bony lesion. The motor and sensory phenomena at the level and below the site of the lesion indicate the location, extent and character of

the injury to the spinal cord. The symptoms usually develop immediately following the injury, become slightly progressive, if hemorrhage only, and then slowly recede until, after a variable length of time,



Site of Lesion.	General Function.	Initial Syndrome.	Residual Syndrome.
	Motility.	Symmetrical flaccid palsy of the lower extremities.	Symmetrical spastic paralysis of lower extremities to level of *L.* 4.
Lumbar Segments (*L.* 3.)		Symmetrical flaccid palsy of the abdominal muscles and lower extremities.	Symmetrical spastic palsy of the abdominal muscles and lower extremities.
	Motility	Symmetrical palsy of preceding plus flaccid palsy of intercostals.	Symmetrical spastic palsy of preceding plus intercostals.
		Preceding signs plus flaccid palsy of upper limbs and disturbances of breathing.	When not resulting in death total bilateral spastic paralysis.
	Sensory	Symmetrical loss of superficial and deep sensibility of the lower extremities. Retention of spinal sensory functions below lesion.	
Dorsal Segments (*D.* 6.)		Preceding plus loss of superficial and deep sensibilities between *D.* 6 and *L.* 3.	
		Preceding plus loss of superficial and deep sensibilities between *D.* 1 and *D.* 6.	
		Preceding plus loss of superficial and deep sensibility between *C.* 4 and *D.* 1.	
	Sensori Motor Reflexes	Total loss of patellar, Achilles, plantar reflexes. Cremasteric preserved.	Patellar reflex retained. Achilles increased. Clonus and Babinski and contralateral Babinski obtained.
Cervico Dorsal (*C8. D1.*)		Preceding plus loss of abdominal reflexes.	Preceding plus spontaneous abdominal movements.
		Preceding with oculopupillary disturbances.	Preceding with oculopupillary disturbances.
		Preceding without oculopupillary signs but with loss of reflexes of upper extremities.	Preceding without oculomotor disturbance and increased reflexes of upper extremities.
Cervical (*C.4.*)		Total bladder and intestinal palsy.	Total bladder and intestinal palsy with later hyperirritability; also of genital reflexes without priapism.
	Visceral Reflexes		
	Trophic Disturbances	Symmetrical dilatation of vessels of lower extremity.	Objective symmetrical chilliness and cyanosis of the legs. Later sacral decubitus. Skin and nail disturbances.
		Preceding with vascular dilatation in upper extremities.	Preceding with objective symmetrical cold and cyanosis of the arms. Shoulder-girdle decubitus possible

Fig. 285.—Localization of symptoms at different levels of the cord. (Veraguth.)

usually from two to three months, the residual symptoms indicate the permanent impairment of function.

The chief syndromes, which develop from injury to the spinal cord at different levels, are here described by means of charts. A study of the muscle palsies, the sensory anomalies, the pilomotor reflexes, the sweat secretions, and the numerous detailed cutaneous and tendon reflexes is indispensable in making a localizing diagnosis. Occasionally osteoarthropathies develop from spinal lesions. They are of value in localizing diagnosis.

It is important to recall that the spinal cord segments and the vertebral segments, while nearly corresponding during infancy, do not in the adult. It is rare that absolutely symmetrical involvements result from spinal injury.

A total destruction of the spinal cord will result in initial and in residual symptoms. This may arise from accident, bullet wound, diving, caisson disease, myelitis, tumor—Pott's disease chiefly. The course, progression and mode of treatment will depend entirely upon the causative lesion.

The Great War has added an enormous amount of new material to the subject of spinal cord injury. Whereas peripheral nerve injuries have been comparatively more frequent, yet spinal injuries have been by no means rare. The student interested in the complexities of the numerous problems arising from spinal cord injuries of war are referred to Schaller's excellent critical review.[1]

The chief symptoms of acute transverse lesion are expressed in the table Fig. 285 and diagrams according to the site of the lesion.

Lesions of the upper cervical segments usually cause instant death from respiratory paralysis. Marked hyperthermia often characterizes upper cervical lesions. Injury lower down is very frequent, occurring in workmen from objects falling upon the bent neck or in reckless diving. The symptoms are charted. Many of these patients live for some time, even years, dying usually from bladder and kidney complications. An inferior cervical syndrome is described by Holmes,[2] which is characterized by hypothermia, lowered arterial tension, slow pulse, diminished urinary secretion, and general stupor, in addition to the usual motor and sensory losses.

Dorsal lesions are comparatively rare, and often show the Brown-Séquard syndrome, which latter may however, develop from lesions at all levels above the conus. Localized lesions at D_1 D_4 often show decided increase in the pulse rate as an additional localizing sign. Mid-dorsal lesions frequently show persistent vomiting. This is a vegetative pathway disturbance. Recent studies of dorsal lesions by Lhermitte, Head and Riddoch and others have contributed greatly to a acknowledge of dorsal cord lesions. Lhermitte[3] has shown that total section of the dorsal cord shows two phases: (1) Phase of shock;

[1] Archives of Neurology and Psychiatry, vol. **1**, No. 4, 471.
[2] British Med. Jour., 1915, **2**, 769 et seq.
[3] La section totale de la moelle dorsale, Bourges, 1919.

(2) phase of spinal automatism. (Mass reflex phase of Head and Riddoch.) In (1) the phase of shock there is complete loss of motility below. Complete loss of sensation of all qualities below the segment. Obscure, unprecise and non-localizable subjective sensibility disturbances result from radicular complications. Tendon reflexes are abolished. Cutaneous reflexes variable. Sympathetic reflexes not lost. Plantar reflex usually flexor in type (very characteristic). Urinary retention, incontinence of feces. Pilomotor and scrotal reflexes preserved. Anidrosis is usual. In (2) the phase of spinal automatism, setting in after a variable length of time, defensive movements are marked. Loss of bony sensibility to tuning fork, may be acoustic retention. Vesical power returns, automatic in character. Visceral sensibility lost. Muscle tonus lost, or hypertonus. Tendon reflexes, may return. Skin reflexes, may be augmented. Babinski reflex may be present. Rectal incontinence may continue or be spasmodic like the bladder.

Lower dorsal and upper lumbar lesions are the most frequent of all. Their symptoms are indicated in the chart. Lesions from D_5 D_8 often present polyuria.

Injury to the lower lumbar vertebræ causes cauda equina lesions, as the cord proper terminates at about the first lumbar. And Collier has shown that in intradural hemorrhage a tendency for the blood to collect in the cauda often gives rise to cauda equina syndromes, the injury often being higher up.

Intramedullary lesions—*hematomyelia*—have a symptomatology all their own.

Careful sensory testing shows that in an intramedullary lesion there is a tendency toward a complete separation of the impulses underlying the appreciation of posture, the discrimination of two points, and their correlated faculties from those of other sensory groups.

All painful and thermal impulses coming from the periphery undergo regrouping after entering the spinal cord, and, whether they arise in the skin or in deeper structures, become arranged according to functional similarity. Then, after a longer or shorter course, they pass away to the opposite side of the spinal cord. (See Plate XIII.)

This process of filtration leaves all the impulses associated with postural and spatial recognition to continue their course unaltered in the posterior columns; they are the survivors of peripheral groups broken up by the passing away of certain components into secondary afferent systems. At any point in the spinal cord, these columns transmit not only impulses from the periphery which are on their way, after a shorter or longer passage, to regrouping and transformation, but at the same time they form the path for impulses, arising both in the cutaneous and deep afferent systems, which undergo no regrouping until they reach the nuclei of the medulla oblongata.

Thus, a lesion confined to one-half of the spinal cord, even at its highest segment, may interfere with the passage of sensory impulses, some of which are travelling in secondary paths, while others are still

within the primary level of the nervous system. All impulses concerned with painful and thermal sensations from distant parts, disturbed by such a lesion, will be travelling in secondary paths and will have come

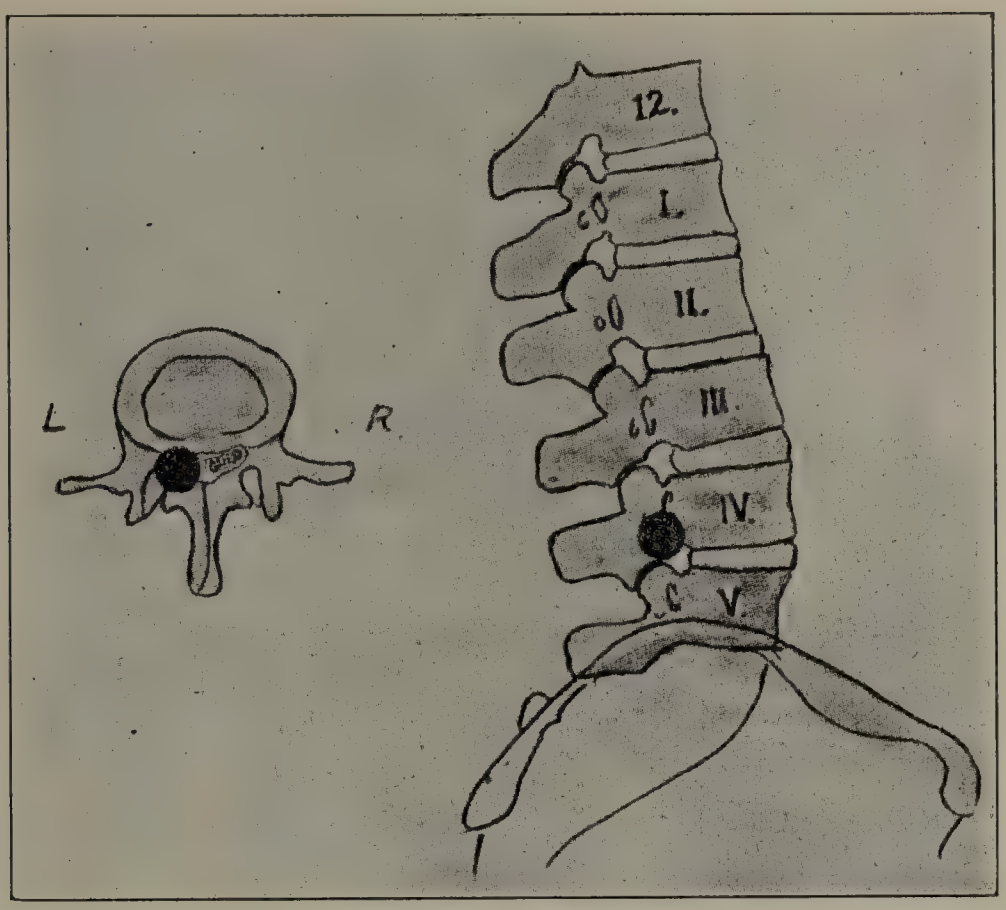


FIG. 286

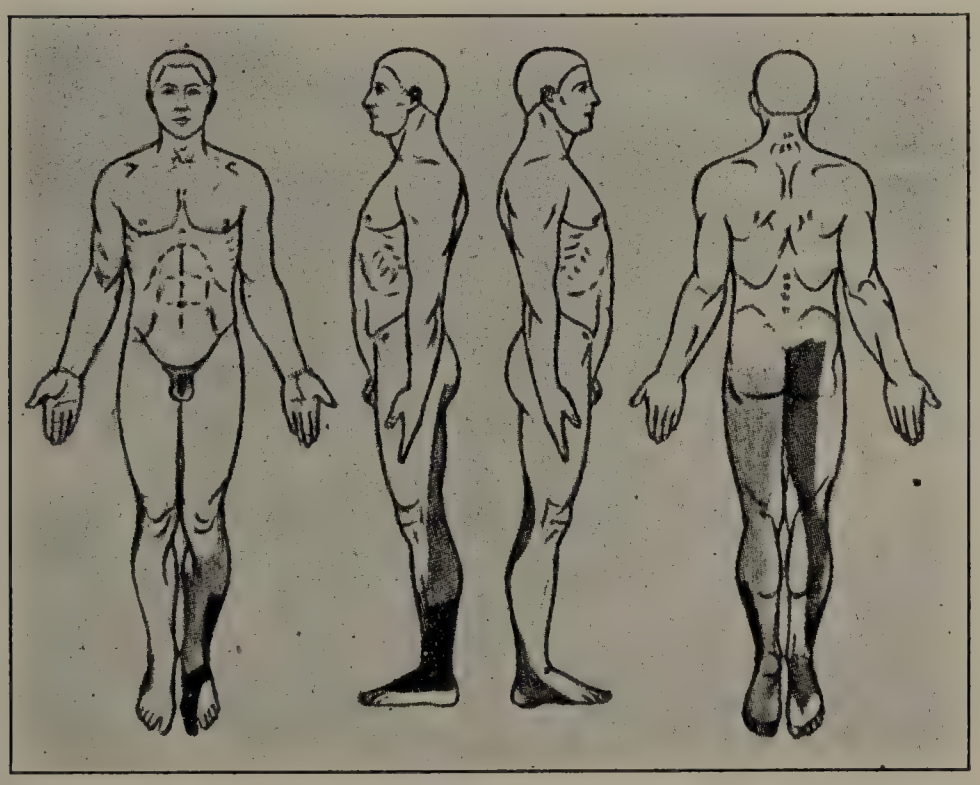


FIG. 287

FIGS. 286 and 287.—Partial lesion of fourth lumbar segment from bullet wound. (Veraguth.)

from the opposite half of the body; for, after regrouping, they have passed across the spinal cord. But those impulses underlying the appreciation of posture, the compass test, size, shape, form, weight,

consistence, vibration, will be affected on the same half of the body as the lesion. They still remain in paths of the primary level and have undergone no regrouping. (See Plates XII and XIII.)

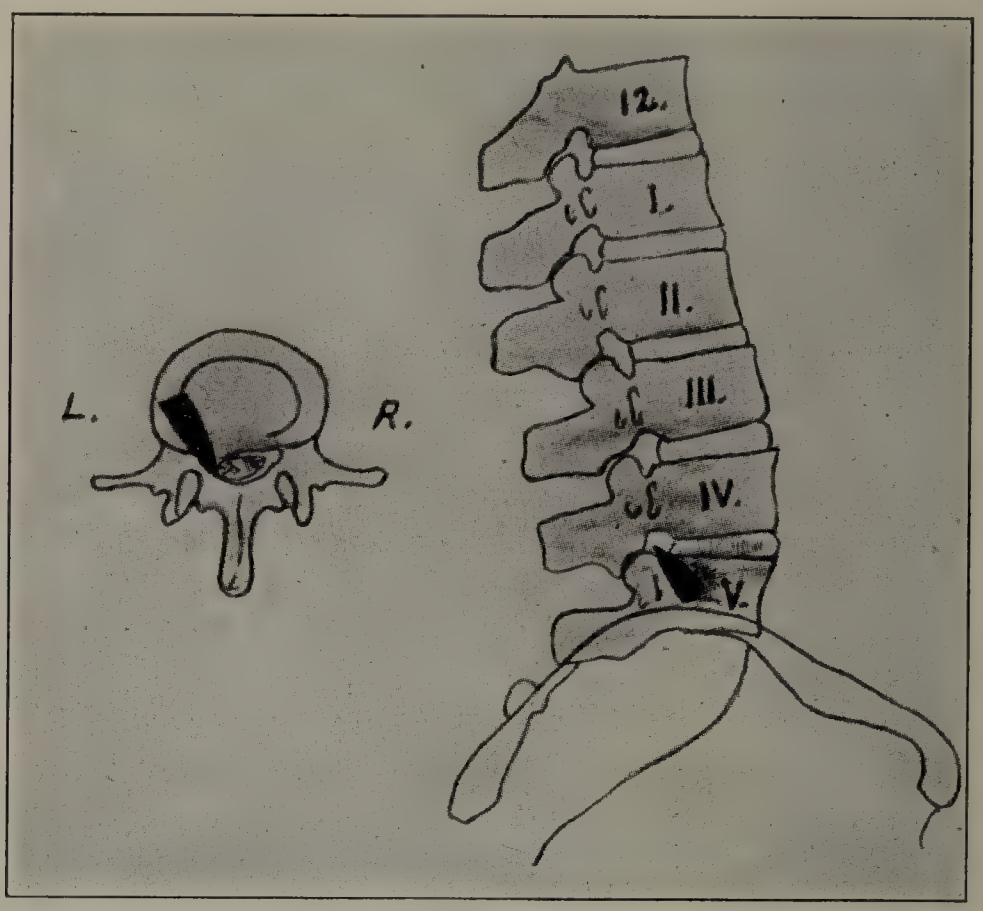


FIG. 288

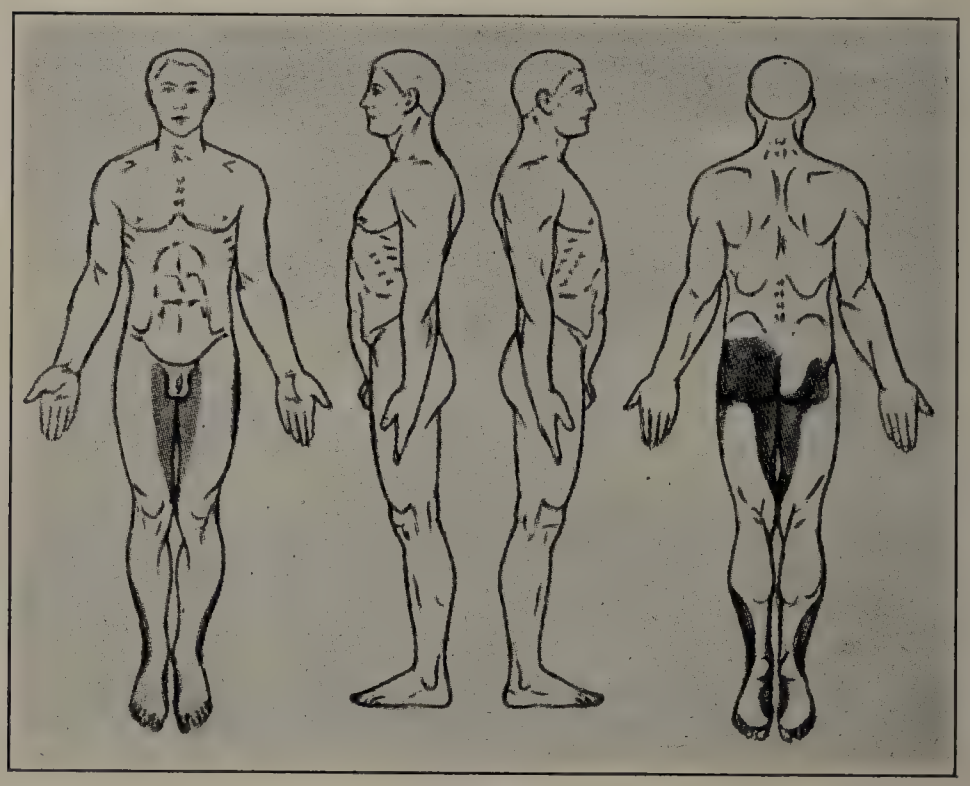


FIG. 289

FIGS. 288 and 289.—Partial lesion of fifth lumbar segment from bullet wound. (Veraguth.)

In such a case the parts on the side opposed to the lesion may be insensitive to pain, heat and cold; but all the postural and spacial aspects of sensation will be perfectly maintained. Yet, all power of recognizing position, of estimating size, shape, form, and weight, or of

discriminating the two compass points, will be lost in the limbs which lie on the side of the lesion, although tactile sensibility and localization of the spot stimulated may be perfectly preserved.

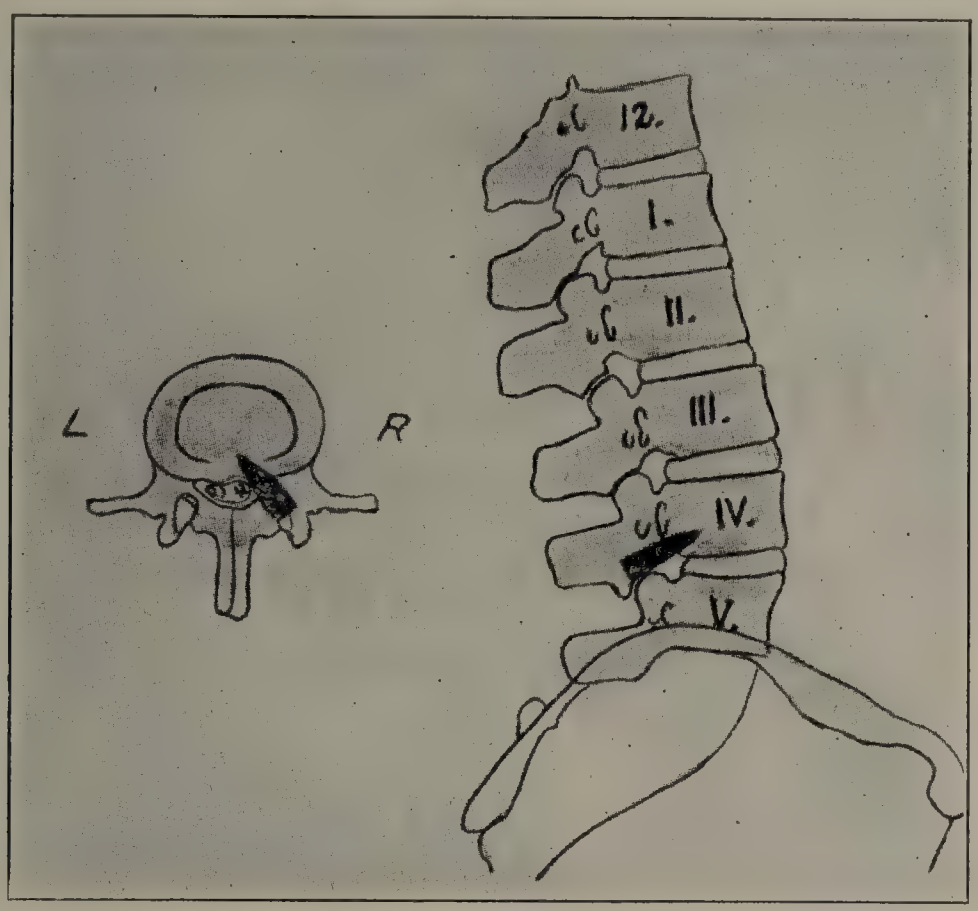


FIG. 290

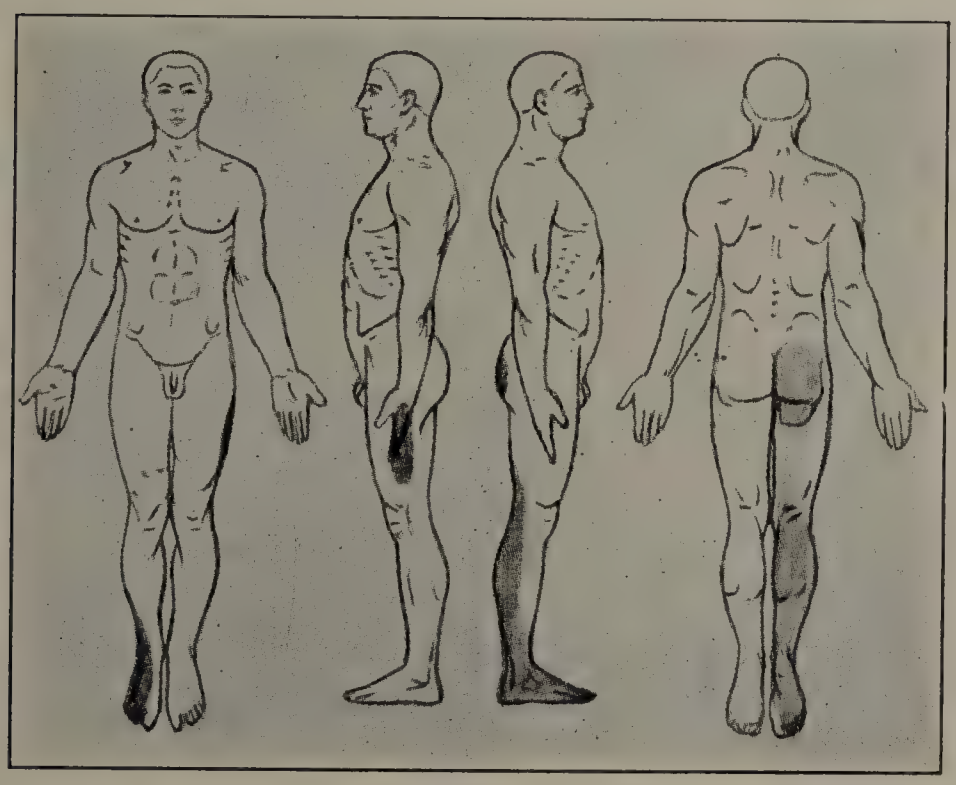


FIG. 291

FIGS. 290 and 291.—Partial lesion of fourth lumbar vertebra from bullet. (Veraguth.)

This remarkable arrangement enables one to analyze, as Head has pointed out, the nature of the peripheral impulses upon which depends the power of postural and spacial recognition.[1] Obviously even at

[1] Head and Riddoch: Studies in Neurology, London, 1920.

the periphery, they must be independent of touch and pressure. The power to distinguish two points applied simultaneously and to recognize as such size and shape, requires as a preliminary the existence of sensations of touch; but the patient may be deprived of all such powers of spacial recognition without any discoverable loss of tactile sensibility. In the same way our power to appreciate the position of a limb, or to estimate the weight of an object, is based upon impulses which, even at the periphery, exist apart from those of touch and pressure, called into simultaneous being by the same external stimulus.

This long delay of the postural and spatial elements in reaching secondary paths enables them to give off afferent impulses into the spinal and cerebellar coördinating mechanisms, which lie in the same half of the spinal cord. The impulses which pass away in this direction are never destined to enter consciousness directly. They influence coördination, unconscious posture and muscular tone, and, although arising from the same afferent end-organs, they never become the basis of a sensation.

Finally, the last survivors of these impulses from the periphery become regrouped in the nuclei of the posterior columns and cross to the opposite half of the medulla oblongata in paths of the secondary level. So they pass to the optic thalamus and thence to the cortex, to underlie those sensations upon which are based the recognition of posture and spatial discrimination.[1]

Diagnosis.—Accurate testing of the motor, sensory, pilomotor, tendon, and also the segmental vegetative functions leaves no doubt as to the presence of a spinal cord injury of a major grade. The results of minute lesions may escape recognition. Total severance of the cord is unusual, most, even severe injuries, leave some pathways undivided, which, after the immediate effects of the injury have subsided, give some sensory response. Total, complete severance causes absolute anesthesia to all forms of stimuli, flaccid paraplegia, with loss of all reflexes and all visceral reactions below the site of the lesion. This has been termed Bastian's (1882) law and has been widely accepted. The recent war experiences have tended to modify it very radically. Riddoch and Lhermitte have shown in complete section, particularly for dorsal lesions, there is a stage of flaccidity, then a stage of reflex activity, when the bladder and rectum may act automatically (vegetative pathways) and muscle tonus may pick up and then a stage of gradual failure of all reflex activities.

Half-sided lesions produce the typical Brown-Séquard syndromes, which vary according to the segments, as seen in the accompanying scheme (Fig. 293). Recent war studies of the sensibility crossing tend to show that in the mid-dorsal regions pain and temperature fibers tend to decussate almost within one segment, in the superior dorsal regions two segments are used and in the cervical cord pain fibers traverse

[1] See Report of Medical Research Council: Injuries of Spinal Cord, London, 1924, and Treatises by Elsberg and Taylor on Spinal Cord, and Roussy and Lhermitte: Blessures de la Moelle, Paris, 1918.

five to six segments, temperature four to five. There are reasons to suppose that below D_{12} the sensibility fibers do not cross at all, hence the Brown-Séquard dissociation syndrome occurs in lesions only above L_1.

Certain other signs are found in incomplete section. Among them are slower relaxation phases in the knee-jerks; no marked difference in tonus between flexor and extensor groups, presence of postural reflexes of the nature of defence—due to integrity of some proprioceptive spinal pathways.

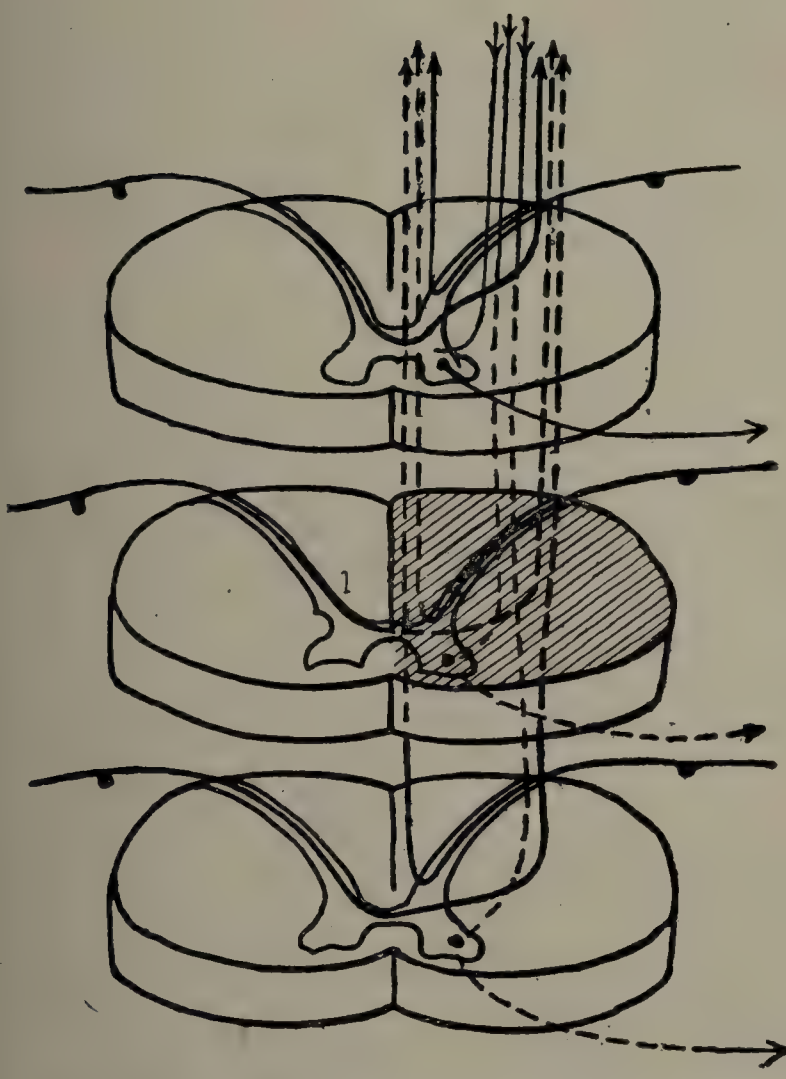


FIG. 292.—Lesion of the cord producing a Brown-Séquard syndrome. (Veraguth.)



FIG. 293.—Brown-Séquard syndrome. Black represents superficial anesthesia and joint anesthesia; – – – passing hyperesthesia and paralysis. (Veraguth.)

Prognosis.—This is bad in practically all spinal injuries save small hemorrhages or limited cauda equina lesions. High lesions are almost immediately fatal. Lower-lying ones—fourth cervical and down—vary in their immediate and remote results, according to the site of the lesion and its extent.

Collier[1] advises careful study of the plantar reflexes as offering some

[1] Lancet, 1916, 1, 700.

clues as to prognosis. A progressive change from no reflex to reduced reflex or response to extensor response is indicative of some recovery and argues for incomplete section—the reverse progression indicates a bad prognosis.

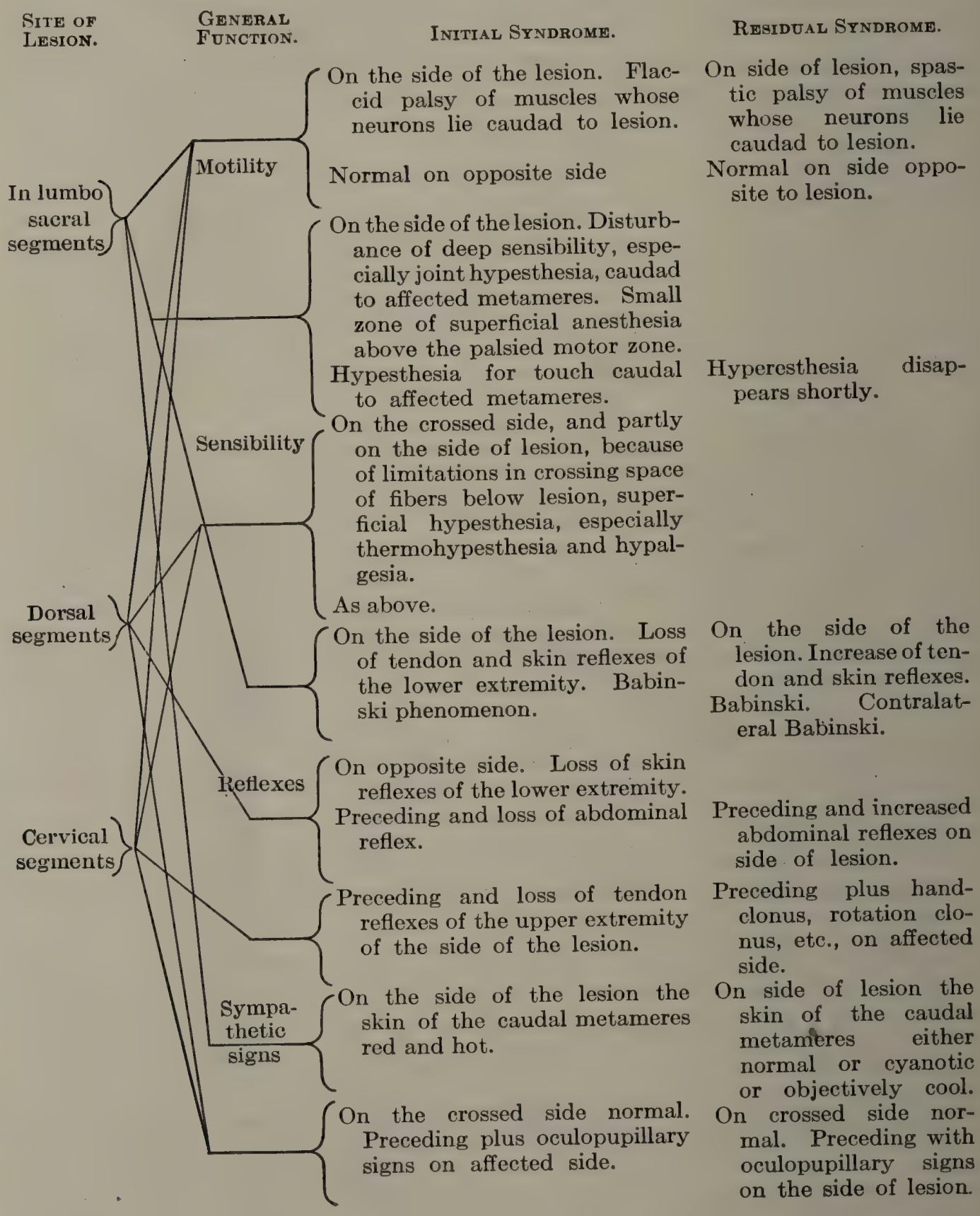


Fig. 294.—Scheme showing localization and symptoms in Brown-Séquard syndromes. (Veraguth.)

The question of regeneration of spinal neurons has not as yet been entirely settled experimentally, but the preponderance of practical evidence is against it.[1] Pathways once destroyed remain so. Just

[1] See Jacob, Girou et Ferrand: Bull. de l'Acad. de méd. de Paris, 1915, **74**, 423, for report of so-called successful cabling of spinal cord. Morat: Lyon Chir., 1914–15, **12**, 761. Collica: Riforma med., 1917, **23**, 583.

at what period it may be said that the residual symptoms will be permanent is largely a matter of the extent of the lesion. As a rule the findings at three months are apt to be those of permanence, still occasionally marked improvement, particularly in bladder and rectal functions, may take place after a year or more of total loss. Many spinal lesions, especially those dependent chiefly on hemorrhage, will show widespread symptoms early, and later these will be reduced to a minimum. The chief bugbear as to life is the bladder. Great care should be taken of it in the early stages, and every available measure taken to keep it sterile.

Treatment.—Surgical treatment is indicated in most spinal injuries even though the prognosis is not at all reassuring. The persistence of some form of sensibility (all types should be tested) is an indication that the cord is not completely severed. Operation during shock is not advisable, yet too long a delay is unwarranted, even if all sensibility seems abolished below the lesion. The functional loss practically always exceeds the anatomical defect. Roentgen-ray examination should be made immediately for diagnosis, and if there is evidence of compression, operation is advisable as early as is compatible with the patient's condition. In the absence of evidence of compression, earlier surgeons advised against operation as dangerous and futile. Since in the hands of competent surgeons most of the dangers of spinal cord surgery have disappeared, there is a growing tendency to operate more freely and, at times, fortunately. More often there is little gain.

Operations, after spinal injury, are often futile, yet with care rarely do any harm, and not infrequently, if the cord itself is only being pressed upon by hemorrhage, may be distinctly advantageous. Evident surgical indications (fracture, dislocation) should be met. The patient is rarely benefited but is entitled to the doubt.

The general treatment of the patient is of great importance. It should be directed toward giving a maximum of relief from distress; avoidance of bladder infection, preventing bed-sores, and careful bowel attention. Antispasmodics and analgesics may have to be used for a long time, but morphine and its derivatives are to be avoided if possible, save in those agonizing cases where it gives the only relief from torture. Electricity is largely a placebo.

Concussion Syndromes.—A mass of material from the war has contributed to a knowledge of severe spinal cord injuries without direct impact of bullets or spinal fragments, but from mass mechanical factors which have produced disseminated hemorrhage, hematomyelias, vacuum pressure changes as in caisson accidents and a host of irregular forms giving rise to direct and indirect forms of severe spinal cord injury with somatic syndromes. Edematous swelling, focal necroses, localized disseminated hemorrhages, with secondary cavity formations and extensive fiber and cell degenerations have been observed. The resulting syndromes have been various. Severe quadriplegias, hemiplegias, monoplegias, diplegias, cerebello-spastic types and anomalous mixtures of these have been described. In fact a great multiplicity of

types shading from the most severe persisting quadriplegias to the cases not definitely to be separated from the more psychogenic war neuroses of the spinal cord are abundant in the literature to which the student must turn as they cannot all be discussed here.[1]

COMPRESSION OF THE CORD.

Compression of the cord, as a slow chronic process results from (1) bony hypertrophies, (2) tuberculosis, (3) tumors, (4) syphilis, (5) aneurisms, (6) meningeal disease.

Bony Hypertrophies (Osteitis, Osteitis Deformans, Osteoarthritis, Spondylosis Rhizomelique).—Under these various names one finds patients who present signs of more or less spinal cord compression, either with or without stiff backs or deformities.

They show gradually increasing weakness, going on to paresis, or complete paraplegia with spasticity and increased reflexes. Frequently there is severe pain and, when the bony disease impinges upon the intervertebral foramina, neuritic pains and symptoms of peripheral nerve palsy develop. (See Plexus Palsies.) Roentgen-ray examinations reveal the nature of the bony changes and the location of the pressure.

Tuberculosis (Caries).—Pott's disease is a most widespread cause of spinal cord compression. From the days of Avicenna, of Guy de Chauliac, of Wedel (1671), who located the process precisely, to Pott, who recognized the relation between a paraplegia and the vertebral disease, down to the very complete monograph, "Paraplegie Pottiques,"[2] by the gifted daughter of brilliant parents, Mme. Dr. Sorrel Dejerine, the study of this disorder has ever been active. In Mme. Sorrel Dejerine's thesis a complete résumé may be found and accurate observations recorded.

Paraplegia, the usual syndrome, is found in children as well as adults. Thoracic spinal disease is the chief producing lesion. Spinal pressure, rarely radicular, causes it. Two periods of evolution are usual. A precocious one, usually within a year, which causes rapid and within a few weeks a complete paraplegia with a serious clinical picture or a slower process extending over a number of years, insidious and less complete than in the precocious type.

These paraplegias are due either to: (1) Rarely, bony compression; (2) very frequently to intraspinal abscess formation; (3) pachymeningitis; (4) edemas without abscess formation. The transitory paraplegias are usually due to edema. The acute rapid ones are usually due to abscess and are most readily cured (eighteen to twenty-four months). The more chronic or incurable paraplegias are due to pachymeningitis, bony compression, or to grave vascular lesions (myelitis).

[1] See Schaller's Digest, loc. cit.

[2] Dejerine, Sorrel: Contribution a l'Étude des paraplegies pottiques, Masson, Paris, 1925.

Symptoms.—The chief symptoms are pain in the back, often reflected and complained of in knee (important to bear in mind in children), stiffness of muscles, rigidity and tenderness on motion in the early stages. With the development of a kyphosis, lordosis or scoliosis, deformities appear and symptoms of cord compression and plexus pressure begin. The plexus symptoms vary with the segments involved, as already discussed. (See Peripheral Plexus Neuritides.) The cord compression gives rise to increasing bilateral spasticity below the site of the lesion, slowly developing sensory impairment, which may develop into signs of a complete cutting off of the entire cord pathways below the site of the compression.

Lymphocytosis in the cerebrospinal fluid is a frequent sign. Roentgen-ray examination will early reveal the signs of tuberculous bony disease.

The symptoms usually show very gradually, as the tuberculous disease is usually a slow one. Occasionally they show a fulminating course, especially in childhood, or even in young adults (abscess).

The outcome depends upon the success obtained in combating the tuberculosis by orthopedic, surgical and general health measures. Early diagnosis is essential, and the spinal fluid examinations and roentgen-ray findings will aid in such an early sizing up of the situation, which will lead to the proper procedures. Absolute immobility at times is the sole valuable therapy which may be combined with marine or other heliotherapy and general tonic measures.

Spinal Cord Tumors.—The consideration of spinal cord tumors makes a large chapter in contemporary sensorimotor neurology, which can only be sketched here. They are comparatively rare, yet frequent enough to put one on one's guard in any spinal cord condition showing compression phenomena, *i. e.*, weakness, spasticity, and increased reflexes, *i. e.*, pressure symptoms of the spinal motor pathways.

Spinal cord tumors are as variable as those found within the cranial cavity. They are found extradurally, durally, intradurally and intramedullary. They are small and large, and located at any, sometimes at all, levels (multiple sarcomata) of the cord. The tumors of the spinal cord are identical in histological features with those of the brain (*q. v.*). Pseudotumors—cysts, angiomata, serous collections, enlarged veins, and many anomalies occur within the spinal canal and produce tumor symptoms. For the most part they lie laterally and posteriorly, and are thus more accessible to surgical removal. They occur at all ages, and in both sexes, and show a frequency comparable to those located in the cranial cavity.

Symptoms.—The chief symptoms of spinal cord tumor are pains, sensory signs in the skin, pilomotor, sudoral, and dermographic changes and evidences of motor paresis. The location of the tumor determines considerable variation in the syndromy.

Sharply localized pains at the site of the tumor are frequent, but may be absent; all pains may be absent, but this is infrequent.[1] The

[1] Bailey: Jour. Am. Med. Assn., 1914.

more usual pains are those of the root area or areas involved by the tumor. The pains are usually unilateral, but widespreading tumors cause bilateral pain later. Any so-called neuralgia, sciatica, etc., may be the initial pressure pain of a spinal cord tumor (see discussion on neuralgias). Under the popular misnomer, "rheumatism," many spinal cord tumors and neuralgic pains are hidden until it is too late to obtain relief.

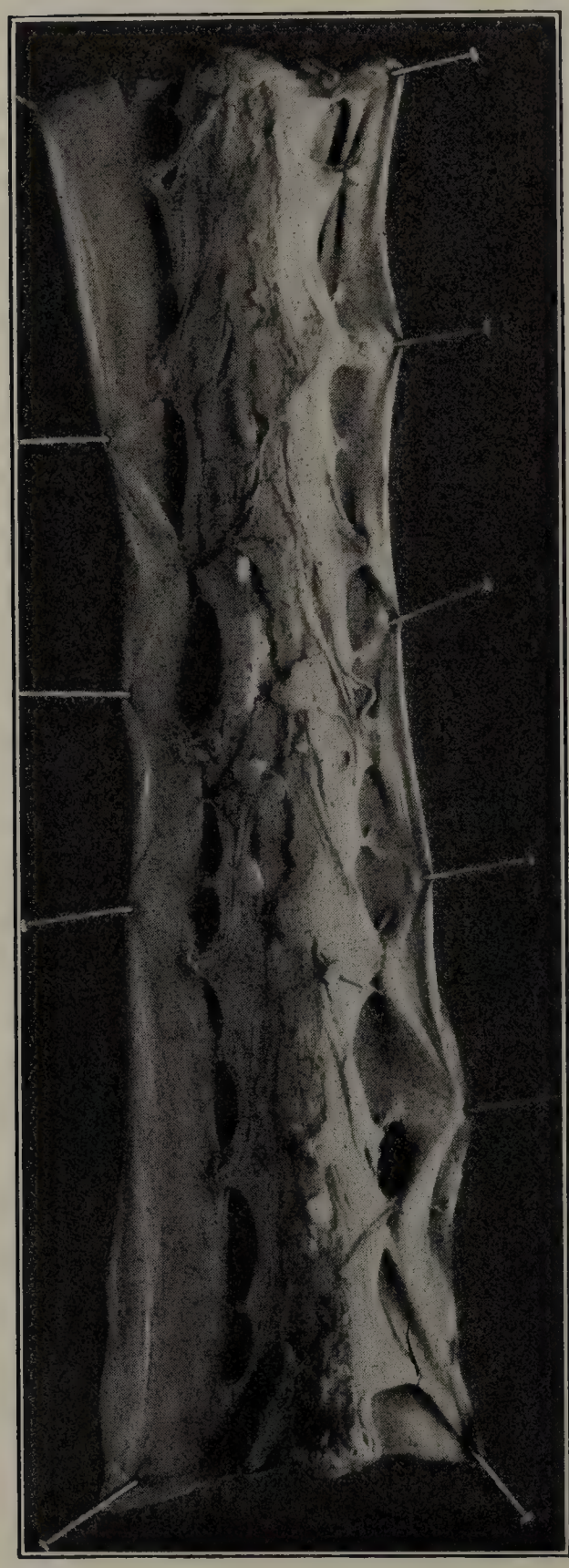


FIG. 295.—Osteophytes of spinal cord. (Larkin.)

The roentgen-ray picture is usually negative. The cell count of the cerebrospinal fluid often gives definite information relative to the subject of irritative meningeal signs (high cell count meaning acute inflammatory disease), and most tumors show a high globulin content in the cerebrospinal fluid. The Wassermann technic will rule out gummata as a rule. In some tumors there is an absence of fluid, or fluid under a very low pressure, below the site of the tumor.

The motor compression signs are variable, paresis advancing to paralysis (paraplegic), hypertonicity, spasticity, increased reflexes, Babinski, clonus, etc. At the level of the tumor there may be destructive lesions with signs of peripheral motor neuron disease, *i. e.*, atrophy, loss of reflexes in the diseased area, reaction of degeneration, trophic changes (bed-sores, etc.)

The sensory phenomena will vary also, one side often showing more markedly than the other. There is hyperesthesia at about the level of the lesion which is changed to various grades of anesthesia below the lesion. Slight loss of epicritic touch is apt to be an early sign. Greater sensory loss advances with increasing compression. Bladder and rectal disturbances are frequent. Small tumors (cysts, etc.), intramedullary, sometimes extramedullary, cause typical dissociation symptoms with retention of epicritic tactile sensibility and loss of protopathic pain and thermal sensibility.

The symptoms for localization of the tumor have already been discussed.

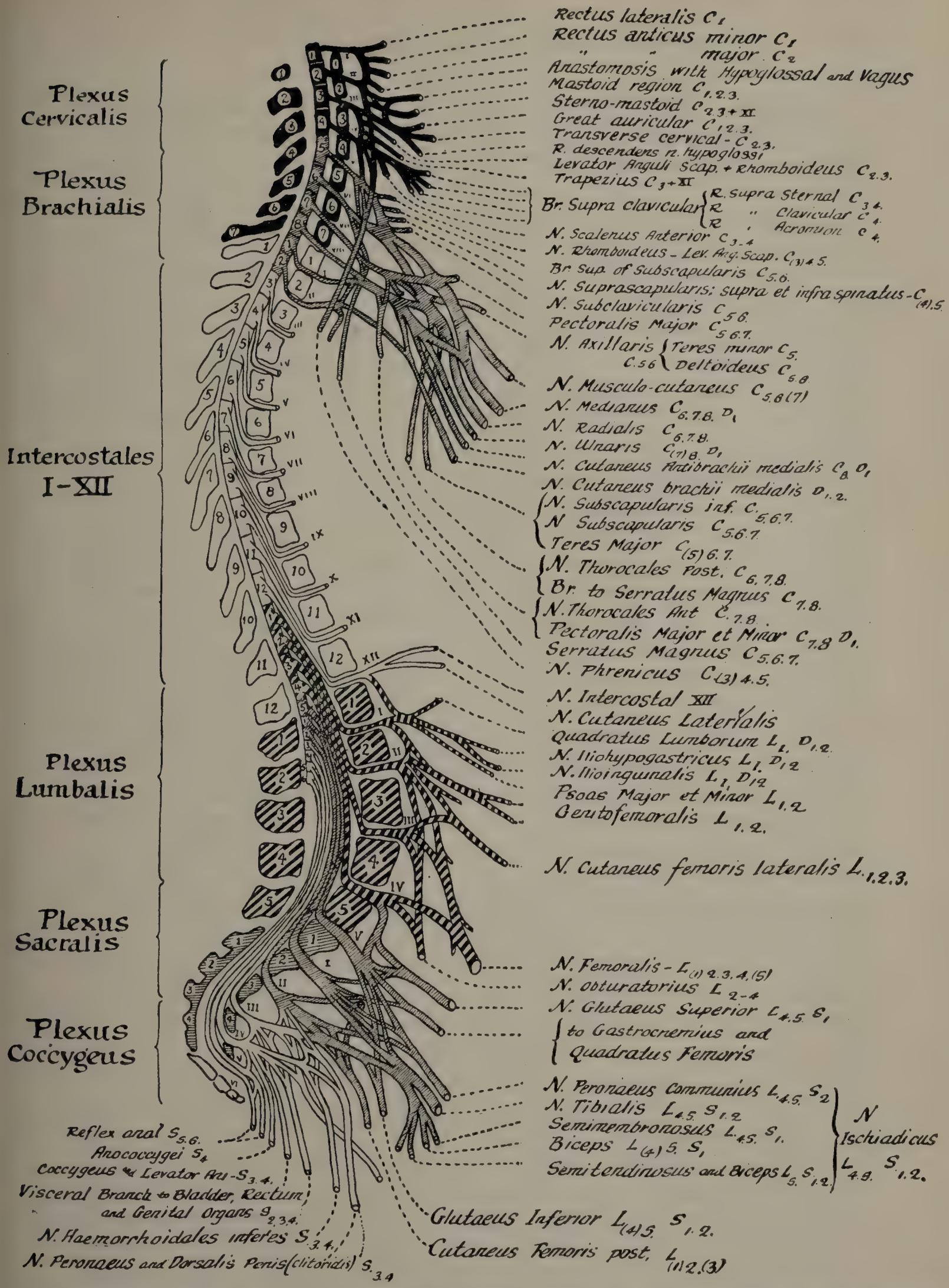


FIG. 296.—Showing the relationship of the segments of the spinal cord to the spinal nerves which emerge from them and to the bodies and spines of the vertebræ. It will be noted that all the cervical and first two thoracic spinal nerves emerge from the cord on practically the same level as the bodies of the corresponding vertebræ. The spinal nerves emerging from the thoracic segments, third to seventh inclusive, emerge from the spinal column about one vertebræ below their origin in the cord. From the eighth downward, the nerves emerge gradually farther and farther below the point of origin in the cord in such a manner that the fifth lumbar nerve emerges four vertebræ below its origin and the sixth sacral nerve, which originates in the cord on a level with the body of the third lumbar vertebræ, emerges from the spinal column at the coccyx. (After Dejerine.)

Diagnosis.—Roentgen-ray, lipiodol, spinal-fluid examination, and a complete neurological status should enable one to arrive at a satisfactory diagnosis. The chief diagnostic problem is syphilitic meningomyelitis. Bony disease is usually excluded by the roentgen-rays. Multiple sclerosis is not infrequently ushered in with sensory signs, also syringomyelia. They present real difficulties in diagnosis. The application of proper neurological and psychoanalytic procedures will determine the conversion mechanisms of hysteria.

Intramedullary or extramedullary diagnosis is practically unimportant since spinal cord surgery has become so satisfactory. Theoretically employed, the problem is of interest; practically, less so. For localization symptoms consult Figs. 282, 283, 284, 291, 292, 295, and Plates XII and XIII.

Treatment.—Apart from syphilis the treatment is surgical and it should be employed early and for practically any or all tumors; not necessarily with the promise of cure but as a routine exploratory procedure. In skilled hands the danger is slight, and apart from special contraindications, the risk is very frequently worth while, as very unexpected things turn up within the spinal cavity.[1]

The localization is the most difficult problem. Most tumors are found a couple of inches above the site usually sought for as determined by the level of the anesthesia. Surgical progress relative to spinal cord tumors is rapidly advancing and cannot be discussed here. The general prognosis has improved both with reference to the finding, as well as the successful removal of the tumors. There may be recurrences, and many patients are not benefited. Little is to be expected in those long-standing tumor cases with marked signs of spinal cord compression degeneration. Yet every patient is entitled to the benefit of the doubt. Surgical failure is better than a let-alone policy covered up by morphinism. This latter even is not necessary, since careful division of the affected sensory roots in inoperable cases may give relief from pain, even if life cannot be saved.

Most patients die of the myelitis bed-sore and bladder complications after one or two years in inoperable cases.

LATERAL SCLEROSIS GROUP.

Historical.—These disorders, which clinically may resemble a number of spinal cord conditions, have been isolated from the mass of rachialgias of the eighteenth century, and from the paraplegias and myelitides of the nineteenth, more particularly by Seguin, Türck and Erb. Erb, in 1875,[2] posited a hypothetical degeneration of the pyramidal tracts in their spinal course, terming the disease primary spastic paralysis. Charcot accepted Erb's teachings and spoke of the disturbance as a spasmodic tabes dorsalis. The disorder, as understood by Erb, has

[1] Elsberg: Surgery of the Spinal Cord. Frazier: Surgery of the Nervous System.

[2] Virchow's Arch, vol. **70**; Deutsch. Ztschr. f. Nervenh., 1899, vol. **6**; Jendrassik: Arch. klin. Med., 1897.

been found to be much less frequent than was at first supposed, as the lateral sclerosis picture was found to be but one stage of a number of other cerebral and spinal affections, notably, as in multiple sclerosis, syringomyelia, hydrocephalus, tumor of the cord, anemia of the cord, diffuse myelitis, senile changes and amyotrophic lateral sclerosis, etc. Of recent summaries, those of Erb,[1] Spiller[2] and Specht[3] are the most valuable.

Pathology.—Ideally this consists of a simple degeneration of the pyramidal tract which rarely ascends to the cortex. A replacement glia infiltration is present. But few autopsies are recorded with the ideal lesion.

Symptoms.—These are exclusively motor, and usually of the lower extremities alone, although the disorder may show itself in the arms. The ordinary picture is that of a spastic paraplegia, *i. e.*, motor weakness, increased reflexes, clonus, Babinski, spasticity, with no sensory or visceral signs.

Gradually increasing weakness of one or both legs is first observed after a long walk, or dancing, or any prolonged muscular exertion. A certain stiffness develops, but very slowly, often only after several years. The tendon reflexes are exaggerated at a very early date.

Slight motor weakness supervenes, and some stiffness to passive motion, and then the patients note that their gait is more constrained; they do not bend their knees, but shuffle somewhat and stumble easily. The limbs become stiffer and stiffer, the extensor muscles being more involved, this brings about extreme rigidity at the joints.

In this stage, which may be arrived at in a few years, or more often after many, the patient walks with a stiff, stubbing tread, perhaps the knees overlap (scissors walk) or knock each other; the toes are not lifted, but rather shoved along, thus wearing the shoes markedly at the toe.

The cutaneous and tendon reflexes are exaggerated. There are markedly increased knee-jerk, patellar clonus, ankle-clonus, Babinski, and paradoxical and Bechterew-Mendel signs; Strümpell's tibialis phenomenon is apt to be marked. Spasms frequently occur in the affected limbs. In the latest stages flexor contractures usually result.

Sensory symptoms, save the muscular pains of spasm, are absent. The bladder and rectum are not implicated. In rare instances the upper extremities are involved, and even the bulbar motor tracts.

Forms and Varieties.—1. *Hereditary Familial Types* (Strümpell,[4] Newmark[5]).—Here the disorder shows itself either in childhood, or late in life, and many generations may be affected.

The symptoms are those of muscular hypertonia, spasticity, exag-

[1] Deutsch. Ztschr. f. Nervenh., 1903, No. 23.

[2] Osler's Modern Medicine, 3d ed., vol. **6**; Bond: Rev. de méd., March, 1908.

[3] Arch. f. Psychiat., vols. **10**, **17**, **34**: Deutsch. Ztschr. f. Nervenh., vols. **5**, **6**, **27**. Specht: Ztschr. f. d. ges. Neurol. u. Psychiat., 1925, **99**, 32.

[4] Deutsch. Ztschr. f. Nervenh., 1904, p. 27; 1906.

[5] American Journal of the Medical Sciences, 1893; Deutsch. Ztschr. f. Nervenh., 1904, p. 27; 1906.

gerated tendon reflexes, and later palsies and contractures. The skin and anal reflexes are less apt to be involved in the familial types. In some cases (Newmark, Lorrain) there are more widespread disorders, optic atrophy, feeble-mindedness, muscular atrophies, etc. Deep sensibility is occasionally involved with slight Romberg.

The changes in these patients tend to become more or less stationary after a certain length of time,[1] but the disease may extend to the upper part of the cerebrospinal axis.

2. *Infantile Types.*—Infantile types beginning from three to six years, are due to developmental defects in the pyramidal system. Here the lower extremities are most involved. The advent of the Wassermann technic is relegating the greater number of these to some intra-uterine or early syphilitic process.

3. *Unilateral Ascending and Descending Types.*[2]—The validity of this type is not yet established. It shows itself as a gradually progressive hemiplegia, supposedly due to primary degeneration of the pyramidal tracts.

4. *Mixed Types.*—These, previously described as due to syphilis, to lead, to lathyrus poisoning, anemia, etc., are more properly more or less irregular forms of myelitis, and are treated under that title.

5. *Congenital Type* (Little's Disease).—This will be considered under Diseases of the Brain. The pyramidal tract disease is secondary to other lesions.

Diagnosis.—Pure types of lateral sclerosis are rare. The underlying condition often develops after careful observation, sometimes extended over several years. The chief disorders to differentiate are multiple sclerosis, compression from tumor, amyotrophic lateral sclerosis, myelitis, old encephalitis, brain tumor, with or without hydrocephalus.

Hysteria can be readily excluded by the careful scrutiny of the reflexes (Babinski, Grasset, Hoover signs, etc.)

Treatment.—Foerster's operation, or posterior root section, may help the spasticities in some patients. At the present time, nothing is known that will stop the advance of the disorder. A Wassermann examination should be made in all cases, as in some an unknown syphilitic element has been revealed. Motor training (Mensendieck, Franz methods) will help the patient to control the spasticities.

COMBINED SCLEROSES. COMBINED DEGENERATIONS.

One speaks didactically of a combined sclerosis as a system disease, in which the lateral and posterior columns are involved; clinically a combination of tabes and lateral sclerosis. It is a doubtful question whether any one disease exists which may be designated as a combined sclerosis, but a great number of conditions are found in which both lateral and posterior columns are degenerated to a greater or less

[1] Bono: Rev. de méd., March, 1908, for summary of all cases to 1908. Specht: Ztschr. f. d. ges. Neurol. u. Psychiat., 1925, **99**, 32, for later study and literature.

[2] Mills and Spiller: Bull. Univ. Penn., 1906.

extent, and as a result varying symptom-pictures are present as the one or the other is more or less implicated.

Thus in true tabes, degeneration of the lateral columns not infrequently occurs; in general paresis there are typical combined scleroses; in many cases of syphilitic meningomyelitis, degenerations of posterior and lateral columns occur, likewise in severe anemias, in poisoning from lead, ergot, pellagra, lathyrus, various bacterial and protozoan infections; in the senile cord similar changes are found. In fact, a great variety of degenerations are found in the cord involving both sets of columns to a greater or less extent. Sano proposes to divide this group into the pseudosystem diseases and the polysystem diseases.[1]

Whether, as Westphal first maintained (1867), there also exists in this motley medley a true system disease of these columns is extremely doubtful. Among this large group, however, certain clinical types stand out, which permit of more or less clear-cut description. In practice they are separated one from another only with great difficulty. By Leyden and his school, most of these disorders were classed with the chronic myelitides. Henneberg[2] used the term funicular myelitis for one group of non-system combined degenerations. These latter are usually due to blood changes, and should be classed with the myelitides rather than with the combined scleroses.

The more fixed of the combined scleroses types which are here considered are: (1) combined sclerosis (ataxic paraplegia (Westphal)), and spastic paraplegia (Strümpell) forms; (2) general paresis forms (see under General Paresis); (3) toxic, anemic forms (Putnam-Dana); (4) senile forms. There are many intermediary forms.

1. **Combined Sclerosis** (Ataxic Paraplegia (Westphal) Types).—Here the characteristic features are those of a spinal tabes, with some signs of spasticity, *i. e.*, Babinski's reflex, and a crawling rather than a typical tabetic gait. Ataxia, pains, bladder disturbances, sometimes pupillary stiffness, etc., indicate that the meningo-neuritic element is predominant, the pyramidal tract involvement of less marked extent. Certain patients start with typical ataxic signs, then gradually develop spasticities and the spastic element finally becomes predominant.

Spastic Ataxia Type.—Here the spastic element enters predominantly into the picture. Weakness precedes, the gait then becomes stiff, and the toes drag; there are increased skin and tendon reflexes, just as in atypical lateral sclerosis; then pains develop; radicular sensory disturbances commence; the knee-jerks become diminished; hypotonia gradually takes the place of hypertonia; one leg may be hypotonic, the other hypertonic; visceral disorders are added, and ataxia and Romberg are present. Possibly there is added optic atrophy, or pupillary stiffness. Nystagmus is not infrequent. The Babinski reflex is apt to persist.

The course in both forms, of which there are all possible gradations, is chronic. Complete disuse of the lower limbs follows. The patients

[1] Bull. de l'Acad. Roy. de Méd. de Belg., 1903.
[2] Arch. f. Psychiat., 1905, **40.**

are bed-ridden with contracted, drawn-up limbs. Twitchings, spasms, and bed-sores supervene.

2. **Combined Sclerosis in Paresis.**—(See Paresis.)

3. **Toxic Anemic Forms.**—Here one may group a motley array of combined scleroses. These are due to poisoning from the lepra bacillus, to diabetes, to lathyrus, to pellagra, to ergot, to alcohol, carcinosis, malaria, chronic septic states, pathogenic protozoa, tuberculosis, Addison's disease, anemia, pernicious anemia, leukemia, etc.[1]

The anemic types offer special features, especially those of pernicious anemia in 75 per cent. of which cord symptoms are present.[2] The anemias may be of manifold pathogeny. They are infrequent. The patients complain early of paresthesiæ sometimes, hyperesthesiæ, of the extremities, there is slight paresis and frequently some ataxia which latter is prone to increase with loss or increase of tendon reflexes, deep sensibility loss (Dejerine) and loss of vibration and position sense (Woltmann). Association of these latter may be present (Petrén). Superficial sensibility may also be involved though less radically. In the spastic types the knee-jerks are increased with clonus, Babinski, and increased knee-jerks. In the ataxic types there is a trend toward loss of reflexes, etc. The peripheral nerves are often involved as well and optic neuritis occasionally is found. The spinal disorder in the pernicious anemic type is progressive, and the patient dies in a year or two with great emaciation, cachexia, and prostration. The precise relationship between the spinal changes and the pernicious anemia is not definitely understood, some patients with bettered blood condition showing progressing spinal changes (Siemerling). Achylia gastrica and glossitis are frequent forerunners. Henneberg speaks of them as funicular myeloses. Four main types which intermingle and vary are distinguishable. Spastic spinal ataxic (pseudotabes), cerebellar ataxia, and cases with marked paresthesiæ.

4. **Senile Forms.**—Slowly progressing weakness of the limbs, with numbness, palsies, and stiffness is frequent in many old people. There develops a shuffling gait, and gradually a more or less complete paraplegia with increased reflexes, Babinski and clonus. The upper extremities share somewhat in the feebleness, tremor and spasticity. These senile myelopathies are very diverse in their nature. Lhermitte[3] finds (1) perivascular sclerosis, (2) marginal scleroses, and (3) combined scleroses of the pyramidal and posterior tracts. Crouzon has found

[1] For literature to 1903, see Sano: loc. cit.

[2] Lube: Deutsch. Ztschr. f. Nervenh., 1914, xlvi, 299. Lichtheim, 1887. Putnam and Dana: Jour. Nerv. and Ment. Dis., 1891. Minnich, 1892. See Nonne for different types, Deutsch. Ztschr. f. Nervenh., 1895. Summaries to 1921 see Gerhardt: Deutsch. Ztschr. f. Nervenh., 1921, **71**, 202. Hamilton and Mixon: Am. Arch. of Neur. and Psych. 1921, **6**, 1. Dejerine: Comptes rend. Soc. Biol., 1913, **75**, 554. Rev. Neur., 1914, **22**, 271, 382. Woltmann: Am. Jour. Med. Sci., 1919, **197**, 400 (from Mayo Clinic 80 per cent in 150 cases of pernicious anemia). Schaller: Siemerling: Arch. f. Psychiat., 1909, **45**, 567. Lauterbach: Ztschr. f. d. ges. Neurol. u. Psychiat., 1924, **95**, 1. Henneberg: Klin. Wchnschr., 1924, No. 22; Lewandowsky's Handbuch. Hurst: British Med. Jour., 1924. Seyderhelm: Ergebn. d. inn. Med. u. Kinderh., 1922. Mathieu, 1925.

[3] Thèse de Paris, 1907. Van Gieson: Jour. Nerv. and Ment. Dis., 1889. Wichmann, Stuttgart, 1887.

in this last group, paretospasmodic, ataxo-spasmodic, and ataxo-cerebello-spasmodic types. Thus it may be seen that the senile cord offers a great variety of pathological changes with a large series of closely related clinical pictures.

SYRINGOENCEPHALOMYELIA.

Historical.—As long ago as 1564, Etienne described cavity formation in the spinal cord; it was further recorded a hundred years later by Bonet (1688) in his celebrated *Sepulchretum*. Morgagni (1740) and Portal (1800) saw and described cases, and Ollivier of Angiers (1834), first gave the name syringomyelia to what had been taught by Etienne to be a persisting central canal, but which Ollivier claimed was a pathological formation.

The studies of Gull, of Landau, and Nonat, with those of Stilling and Waldeyer, first gave the impetus to the correlation of the anatomical lesion, and the clinical symptoms. Duchenne (in 1853) then called attention to certain muscular atrophies with sensory anomalies, which differed markedly from the chronic muscular atrophies he was describing; but it was not until as late as 1882 that both Kahler and Schultze brought out the factors that permitted a diagnosis during life.

This marks the period of active neurological interest in the disease with a sudden growth in its symptomatology. Wichmann (1887) and Anna Bäumler (1888) published monographs, the latter collecting 112 cases. Van Gieson (1889) gave a valuable study.

In the six years following, up to the appearance of the first edition of Schlesinger's masterly monograph, contributions appeared from everywhere, and the related subjects of leprosy and Morvan's disease took on an active interest. Schlesinger's (1894) monograph fixed the lines of research, which are shown in his thorough and monumental second edition of 1902, a monograph of some 600 pages, since which time few striking additions have been made. Haenel's contribution to Lewandowsky's *Handbuch* gives the later literature to 1911,[1] while Bremer's monographic study collects the material to 1926, special emphasis being laid upon the etiology.

Etiology.—Men are more frequently affected than women. Later studies report more females (Creutzfeld). Most of the cases occur before the age of forty, the greatest age of incidence being between the ages of twenty and thirty. It is not known that occupation has any bearing on the etiology. Toxic factors are not universal; infections do play a role in causing cord hemorrhages, emboli or thrombi, with secondary cavity formation—Schlesinger believes that in such developments the cord was not previously normal. Syphilis may be such an infectious agent also other noxæ. Syringomyelia in mother and son, and in different members of the same family has been observed. Redlich, Sindelar, Wexberg, Barré and Reys, Finzi and others all

[1] Tannenberg (Ztschr. f. d. ges. Neurol. u. Psychiat., 1924, **92**, 119) discusses the pathology to 1924. Bremer: Deutsch. Ztschr. f. Nervenh., 1926, **95**, 1.

report familial cases and also constitutional factors. Definite hereditary situations are given in Bremer's monograph.

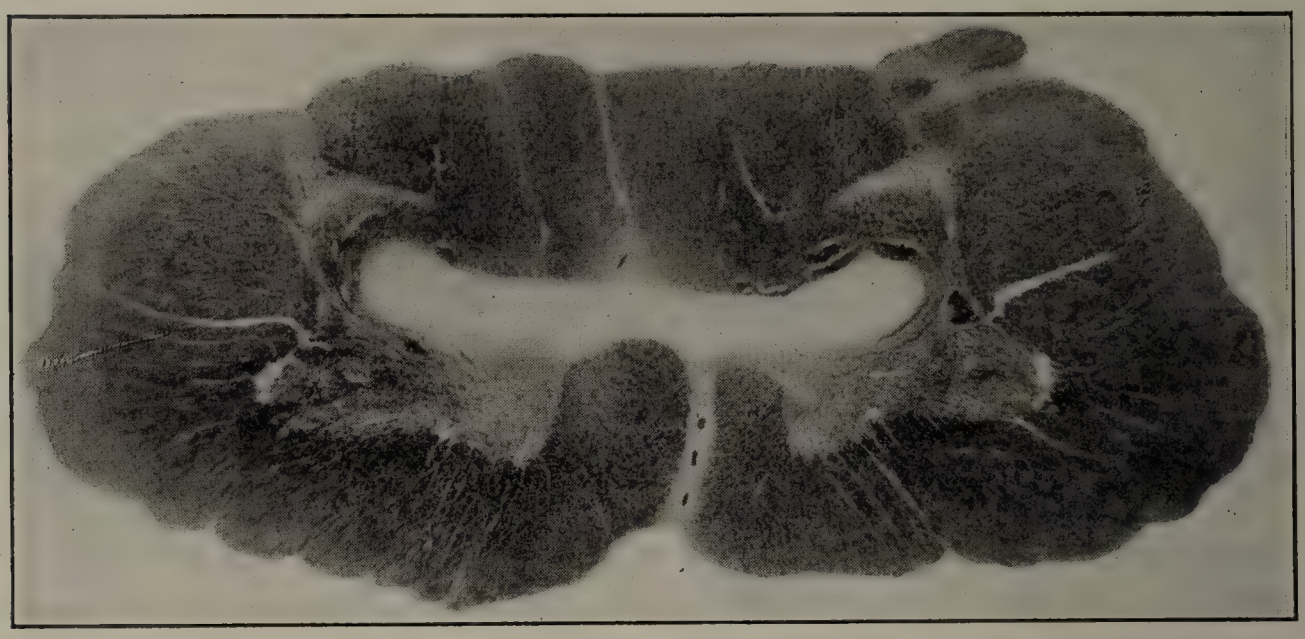


FIG. 297.—Syringomyelic cavity in the cord (cervical region.)

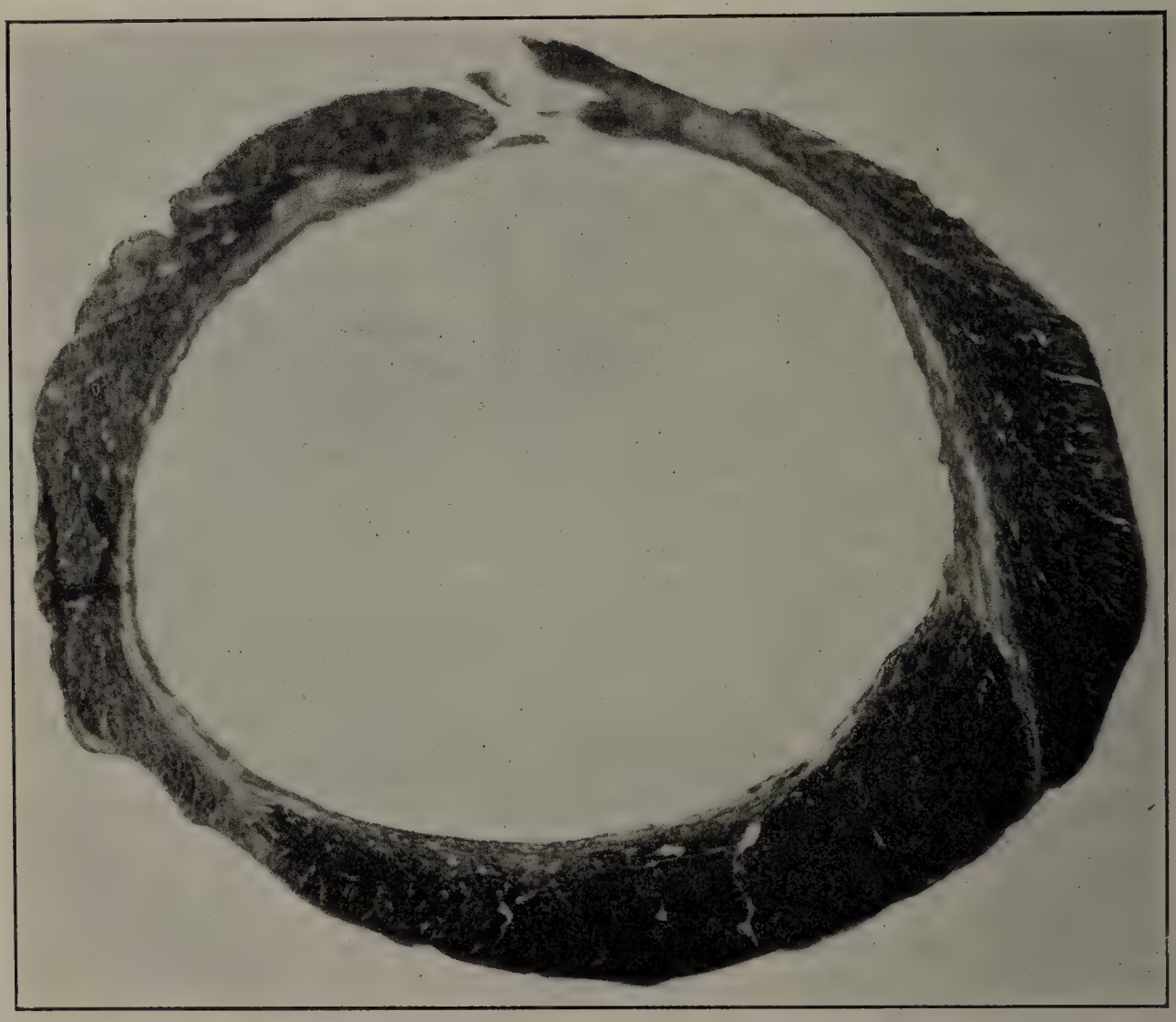


FIG. 298.—Another level of the cord, showing gliomatous tumor cavity.

Traumatism undoubtedly plays a role. Hematomyelia develops after spinal traumata, and then may give rise to secondary cavity formation; large numbers of which have been reported resulting from the Great War. Local traumata with ascending neuritis have possibly

given rise to later developing syringomyelia. Leprosy may also condition a syringomyelia. Vascular tumors with degeneration and proliferative glia changes are also causative.[1]

Symptoms.—Like multiple sclerosis, syringomyelia is characterized by its extremely rich and variable symptomatology; like this disorder also, almost anything is to be expected, since with the gradual increase in extension of the cavity formation in the cord, new areas become involved, and older boundaries enlarged. It is therefore a disorder which is apt to show a gradually developing symptomatology, and persisting as it does many years, a rich array of pictures may be presented. Again it is very irregular in its manifestations, although certain features form the background of the disorder. These chief features are: (1) a peculiar dissociation of the sensory impulses: (2) muscular atrophy and other trophic disturbances, affecting chiefly the skin and joints; (3) motor disturbances either irritative or paretic in character. Not all of the cases show these symptoms, but inasmuch as the cavity formation is apt to occupy certain portions of the cord more often than others, this grouping of symptoms occurs more often than other groupings.

1. *Sensory Dissociation* (Kahler, Schultze).—This consists in a loss of ability to recognize sensations of heat and cold and sensations of pain but without any loss of touch, especially of epicritic touch.

This dissociation varies considerably. It may be absent. In one patient or in one area the thermo-anesthesia is very profound, the loss of pain sense less so—again, in others, the reverse is true. In the majority of patients the loss is only a partial one, not an absolute one. Most cases if seen early enough will show only slight reductions—epicritic heat and cold are lost before protopathic heat and cold—sometimes the reverse is true, or thermo-anesthesia and analgesia may be present on one side only. Again heat or cold alone may be affected. Epicritic touch may be involved, but it is not usual.

These sensory anomalies may be distributed over very small areas, rarely bulbar (mucous surfaces included), most often cervical; or they may extend almost throughout the entire spinal axis from the trigeminus to the cauda (loss of testicle pains, and analgesia of the bladder, etc.).

The areas of diminished or lost sensibility to heat and pain are usually bilaterally asymmetrical, not infrequently they are unilateral for a time, then spread to the opposite side. They show the most unique distributions. The distribution may be exquisitely radicular; again, it is predominantly segmental or metameric. Schlesinger's most recent researches speak for the segmental type of distribution for the majority of the cases (Fig. 298).

In the beginning, one frequently encounters the glove and stocking type of sensory changes. Later a whole limb will be involved; there

[1] Weitz: Beitrag zur Aetiologie der Syringomyelie, Deutsch. Ztschr. f. Nervenh., 1924, **82**, 65. Tannenberg: Ztschr. f. d. ges. Neurol. u. Psychiat., 1924, **92**, 119, with full bibliography. Bremer: Loc. cit.

may be girdle sensation. A hemianalgesia, or hemithermo-anesthesia may be present. Allochiria is usually absent.

The sensory changes usually take years to develop. This sensory dissociation is frequently preceded by paresthesiæ—burning pains, cold spots, neuralgia-like pains, etc. Position sense is rarely involved, even in the advanced cases. Tactile agnosia (astereognosis) is frequent. The bony sensibility is very frequently involved (Egger). It may also be, although rarely, an initial sign. Deep pressure sense is often involved. Romberg and ataxia are naturally not infrequent.

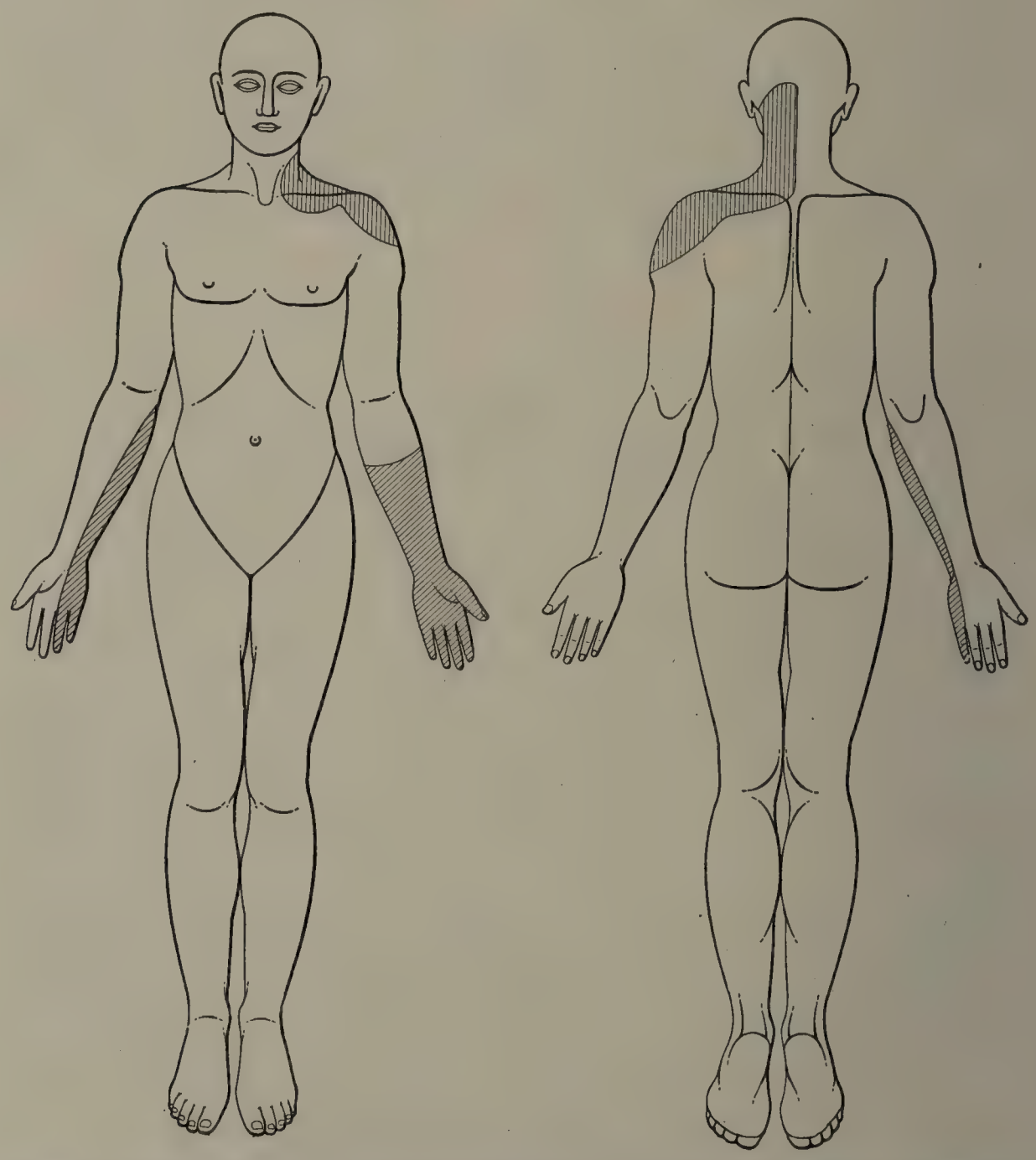


FIG. 299.—Syringomyelia, showing the dissociated loss of sensibility. Areas of loss of sensibility to pain.

2. *Muscular Atrophies and Other Trophic Changes.*—These changes occur most often in the small muscles of the hand (ulnar distribution), and of the arms. Thus are produced typical claw-hands, which develop slowly and insidiously. Here, again, no absolute rule is followed. The interossei may atrophy first, or the muscles of the thenar or hypothenar eminences. A (median) typical monkey atrophic hand may be present, or the "preacher-hand." Or the atrophy in the hands may be

skipped or combined with that of the shoulder-girdle (Aran-Duchenne types), the wings of the scapula stand out, etc. Atrophy of the trunk muscles results in various distortions of the spinal column. Pes equinus, pes valgus, etc., occur in the lower extremity, though less frequently. The bulbar nuclei may be involved, causing speech disturbances; and these may be initial symptoms, though infrequent. Cerebral extension is even recorded (Spiller). Argyll-Robertson pupil has been observed (Wilson).



FIG. 300.—Syringomyelia ("preacher's hands").

These atrophies are usually progressive; like the sensory changes, they are usually bilaterally asymmetrical, and show much variability. Thus one may find a claw-hand on one side, and a monkey-hand on the other, etc.

Reaction of degeneration varies, being present in some and not in others. Increase of electrical excitability is present in others.

Fibrillary twitchings are common in the degenerating muscles, and even more active movements are observed, consisting in static tremors, choreic-like movements, intention tremors, or paralysis-agitans-like tremors. Cramps are not infrequent, and peculiar myotonic contractions. These irritative motor phenomena belong more to the early stages.

The motor power is uniformly diminished with the muscular atrophy, and spasm or contractions and rigidity may be present. Spasticity is not infrequent in the lower extremities. This only argues for pressure upon or involvement of the pyramidal tracts by the lesion.

The gait is not infrequently involved. The patients tire easily; they commence to walk more slowly; hemiplegic types, paraplegic types, patients bent to one side, or bent strongly forward are seen

in the later stages. Ataxic and tumbling gaits belong to the curiosities with rare bulbar localizations. Schlesinger reports a large variety of rare anomalies.

Trophic Disturbances.—These may be many. They may vary in number and extent in strict accordance with the involvement of the trophic cells in the cord.[1]

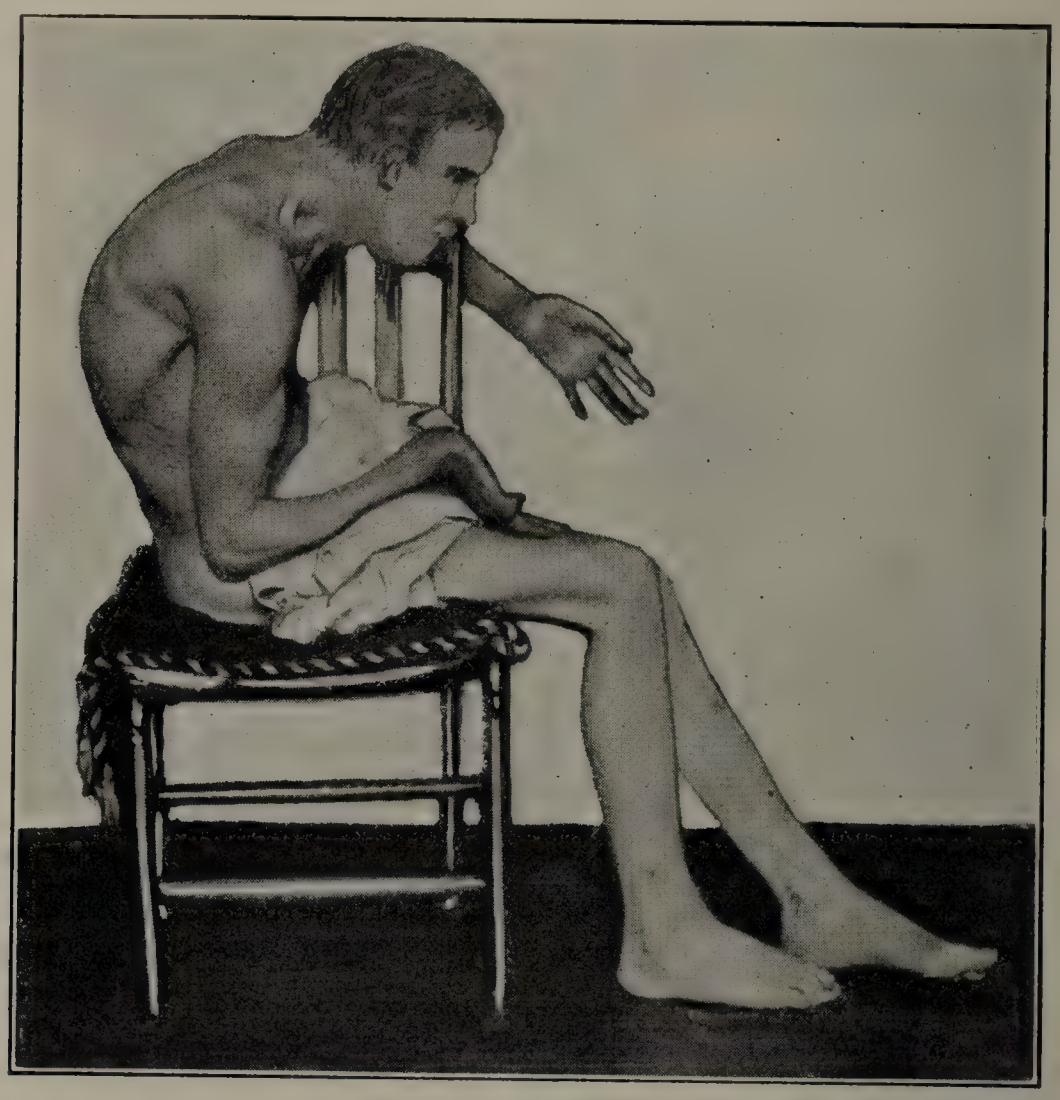


FIG. 301.—Syringomyelia, showing the curvature of the back and the atrophy of the muscles of the hands.

In the skin one finds hyperemias, either active—idiopathic congestive erythemas—or passive, with the formation of dark red patches or various patches of a dark blue color—cyanotic. These changes are usually associated with others of the muscles or of the bones. Cutaneous anemias and cutaneous edemas (succulent hand), with or without distinct Raynaud symptoms, may occur. The glandular activities of the skin also suffer. The perspiration may be absent, or excessive, or one encounters curious anomalies, such as increased perspiration to cold stimuli, or islets of increased perspiration in normal skin, or perspiration-absent areas. These areas of altered perspiration show anomalous distributions quite comparable to those seen in the sensory sphere. Changes in the fatty secretions may be looked for.

Chronic dermatoses are also encountered with exudative phenomena; urticarias, angioneurotic manifestations, phlyctenulæ, dermatitis

[1] Curschmann: Handb. d. inn. Med. (Mohr-Staehelin, 1925).

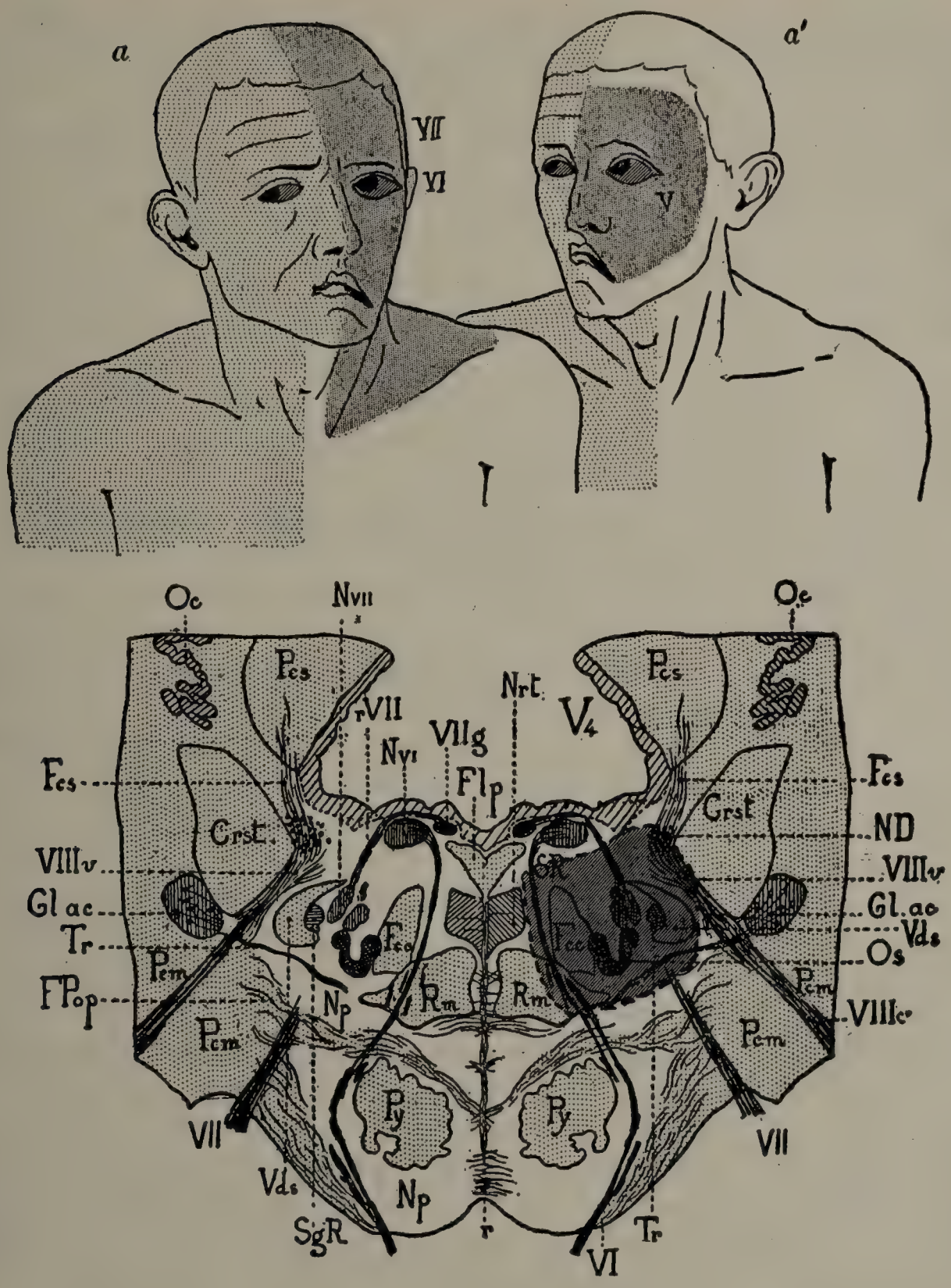


FIG. 302.—Pontine syndrome, with eye palsies of central origin and syringomyelic dissociation. There is here a crossed hemianesthesia with alternating paralysis of the VI and VII cranial nerves, anesthesia of the V nerve due to hemorrhage in the lateral and lower portion of the pontine tegmentum of the left side. The *right-hand figure* shows the hemianesthesia, dissociated as in *syringomyelia* (hemianalgesia and hemithermanesthesia due to lesion of the crossed sensory pathways of the lateral portion of the reticular formation). There is preservation of the tactile and postural sensibilities and of the stereognostic sense, because of the incomplete extension of the lesion to the median lemniscus (*Rm*). The *left-hand figure* shows (1) atrophic paralysis of the VII nerve with reaction of degeneration, lagophthalmia, drooping of the lips, loss of facial mimicry, paralysis of the entire left facial (VII) indicated (Fig. *a*); (2) anesthesia of the face, following involvement of the descending root of the trigeminus (see V on *a'*); (3) paralysis of the external rectus with convergent strabismus by reason of the overaction of the antagonists. Furthermore, there is a paralysis of the lateral movements of the eyeballs toward the left notwithstanding the integrity of the posterior longitudinal fasciculus (*Flp.*), of the nucleus of the VI and of the adjacent reticular formation. The lesion of Deiters's nucleus, and of the labyrinthine oculorotary fibers which unite Deiters's nucleus (*ND*) to the nuclei of the III and VI causes this. By reason of the overaction of the antagonists the patient looks to the right. (After Dejerine.) For abbreviations on the anatomical sketch see section on Midbrain and Plate X.

bullosa. Hemorrhagic infiltrations, or even ulcer and gangrene of the skin are known. True hypertrophies, sclerodermas and changes in the nails are recorded.

The bony structures also suffer. Arthropathies are common (10 per cent.—Sokoloff; 25 per cent.—Schlesinger), but more often in the upper extremities, in sharp contrast to the lower limb distribution of tabes. They usually occur late in the disease and persist for many years. The onset is usually acute, with pain, swelling, and destruction. Mild cases cause little or no deformity. Both atrophic and hypertrophic changes occur, with fixation of the joints, dislocations or fractures. The sensory, secretory and trophic disturbances are usually in closely related areas, and bony sensory anomalies are to be expected. Occasionally suppurations occur.

Bony changes in the vertebræ, with thorax deformities, occur either as a consequence of arthropathies, or as muscular atrophies. These scolioses occur usually in the upper dorsal, and give rise to compensatory curvatures.

The bony structures of an entire limb may be involved—a hand or a foot—with atrophies of the muscles. Here acromegaly is to be ruled out usually by the deformed, contracted nature of the syringomyelic hand or foot, the marked muscular atrophy, and the isolated character of the limb involved. Syringobulbia may cause a facial hemiatrophy or hemihypertrophy.

Reflexes.—Great variability and changeability is present. The skin reflexes vary from complete loss to exaggeration. The Babinski reflex may or may not be present, as well as the abdominal, epigastric and cremasteric reflexes.

The tendon reflexes are often striking in that one reflex in the arm for instance will be lost, the others present, or even exaggerated. The same holds true in the lower extremity, but is less striking. Increased reflexes are here more apt to be found. Increased knee-jerk, Achilles-jerk, and even ankle-clonus are not infrequent, particularly in high-lying syringomyelias—bulbar, cervical and upper dorsal. Lost knee-jerks, unilateral or bilateral, may also occur in syringomyelia, either as an early or a late symptom, and may be associated with increased skin reflexes (Babinski).

Rare cases are encountered with increased jaw reflexes.

Visceral Symptoms.—Bladder disturbances are not the rule, although at times appearing, usually late in the disease. When appearing early they are apt to be transitory. They may be sensory or motor, irritative or paralytic. Cystitis is not infrequent in the later stages of the disease. André Thomas has described a persistent galactorrhea in one patient. In syringobulbias one finds anomalies of secretion, polyuria, glycosuria, diabetes insipidus, pollakiuria.

Obstipation is not uncommon; incontinentia alvi less so. Loss of sexual power and of desire also occur. Persistent priapism is one of the curiosities of this disorder; also analgesia of the testicles.

Bulbar Symptoms.—These are frequent, when isolated being then termed syringobulbia. They are usually more benign than when found in other affections (chronic bulbar palsy), have a very chronic course, are usually unilateral, and often implicate the vagus. They occur in about one-third of all cases. Certain affections of cranial nerves have been recorded for many years. Smell and hearing are involved rarely. Vestibular vertigo is also rare. A dulling of the sense of taste is not infrequent, and shows irregularities of disturbance—half-tongue (Dejerine); dissociation (Hitzig, Simon)—antero-posterior changes.

Optic nerve atrophy or swelling of the disk occurs in advanced bulbar cases; a few cases show restriction of the color fields. The eye muscles may show nystagmus or nystagmoid movements, paralyses and sympathetic complications. H. Simon reports an interesting case of nystagmoid movements with both eyes, absent when only one was in use. Schlesinger reports transitory diplopias in 11 per cent. of the cases; they are not infrequently early signs, and do not necessarily recur. The abducens is frequently affected.

Ptosis is not infrequent, and is often an important early sign. Its relations to the sympathetic are intimate. Myosis is found in a small percentage of cases, and differences in the pupil are frequent, at least 25 per cent. (Schlesinger). They are mostly due to sympathetic palsies. Argyll-Robertson pupil has been observed, but chiefly in pre-Wassermann days but also later (Wilson).

The Klumpke syndrome is frequently met with and points to the upper dorsal localization of the lesion. Myosis, diminished lid aperture, retraction of the bulbi, and anomalous pupillary reactions (loss of cocaine dilatation, etc.) are signs of sympathetic involvement.

The trigeminus distribution shows sensory anomalies, either as paresthesiæ and pains (tic douloureux) or as analgesiæ, with loss of the trigeminal reflexes and corneal trophic changes. Dissociation in the trigeminal distribution is also observed. Pain and temperature are mostly affected. Deep sensibility is less often involved.

The facial is involved in but few cases (3 per cent.) but impairment of ability to swallow is not infrequent. The tongue is involved, projects to the paretic side, shows fibrillary twitching, and hemiatrophy. The soft palate and laryngeal musculature is often involved at the same time, which with the loss of sensation often causes deglutition pneumonia.

Laryngeal palsies are infrequent. They are characterized by the palsy of one recurrent nerve, and the usually accompanying homolateral palsies of the pharynx and soft palate. Posticus palsy and homolateral bulbar palsies go hand in hand. From 15 to 20 per cent. of all syringomyelias have laryngeal symptoms which not infrequently antedate the other bulbar or cranial nerve symptoms.

Speech disturbances are common, as one or other of the related mechanisms are apt to be involved. Recurrent palsies give the high, rasping voice, tongue involvement, the thick, hot-potato speech of

the bulbar paretic; palatal palsies cause a "nasal" voice; occasionally a scanning-like speech is observed.

Tachycardias, bradycardias, and dyspnea are among the rarer symptoms.

Apoplectiform or epileptiform attacks have been described, and are sufficiently frequent to attract attention. They are often accompanied by signs of vestibular involvement—rotatory vertigo, nystagmus, vomiting, etc.[1]

Mental Symptoms.—Syringomyelia is often associated with chronic hydrocephalus which two conditions arise simultaneously. Here defect symptoms are present either in the form of debility or imbecility. Other patients are reticent, surly and vindictive. Many patients come to develop a totally perverted feeling about their anesthesiæ, analgesiæ, etc., and cannot be convinced regarding its true character.

Course and Progress.—The great multiplicity of symptoms, and the almost fortuitous situation of the tumor and cavity formation make it inadvisable to generalize too sharply about the symptom groupings, but in general one can outline four more or less classical types. These are the bulbar, the cervical, the dorsolumbar, and the sacrolumbar.

1. *Bulbar Types.—Syringobulbia.*—If restricted solely to the medullary lesions, this is the least common of the types (Schlesinger). Bulbar symptoms, however, are very often found in the other types. In this type the lesion is limited to the cranial nerves. The laryngeal palsies are prominent; difficulties in swallowing are present, either transitory or persistently, and cause death. Atrophy of the tongue, and paresis of the ocular muscles are frequent. Sensory disturbances in the region of the trigeminus are frequently found.

Initial syringobulbias, in contrast to those developing with or following spinal signs are not as dangerous to life *per se.*

2. *Cervical Type.*—This is the commonest and best known. The patients first complain of weakness in the small muscles of the hand with clumsiness for finer movements. Paresthesiæ and pains are frequent in the hands and arms. The patients frequently have severe sores on the hands from being wounded or burned, and then note the advancing analgesia, muscular wasting, and loss of ability to tell hot from cold, analgesiæ, total or partial, with intact touch sense. The difficulties are first unilateral, and later spread asymmetrically. The knee-jerks are apt to be exaggerated. Advancing disease shows itself in the greater atrophy, with claw-hand, preacher-hand, monkey-hand, and the whole shoulder-girdle may show involvement. The knee-jerks are increased, clonus and Babinski may be present. There may be beginning scoliosis or kyphosis. Trophic disturbances in the upper extremities appear.

3. *Dorsolumbar Type.*—Strictly localized dorsolumbar types are rare. Here the typical syringomyelic sensory anomalies are met with. Paresthesiæ, pains followed by analgesia, thermo-anesthesia, preserva-

[1] Martin, J. P.: Fits in Syringomyelia, Jour. Neurol. and Psychopath., 1924, **5**, 227.

tion of touch, kypho-scoliosis are frequent. The muscles of the pelvic girdle become involved and the lower extremities show atrophies and deformities. The Klumpke type of sympathetic involvement is seen here as well as in the preceding type, *i. e.*, unequal pupils, inequality in the palpebral fissure, recession of the eyeball. The gait becomes spastic paretic, the tendon reflexes usually increased—at times lost. Girdle sensations and involvement of the bladder and rectum are not infrequent. Secretory and trophic anomalies, already noted, point to the localization of the process. Many of these cases closely resemble tumors of the cord.

4. *Sacro-lumbar Types.*—These are rare types, and are characterized by muscle atrophies, especially of the lower extremities, and the smaller muscles of the feet. The glutei may also be involved. Segmental sensory disturbances of the perineum and genital regions of the dissociated type referred to are present. Trophic disturbances are usually profound and extensive, such as fractures, ulcerations, running sores, etc. Vasomotor disturbances are present. The tendon reflexes are usually increased. Babinski may be present. Bladder and rectum are usually involved. Contractures are common, and kyphoses and scolioses occur.

Characteristic Groupings.—Not only do certain types stand out, conditioned by the topography of the intramedullary lesion, but certain cases show predominant forms of lesion, such as motor, sensory, trophic, or secretory. Thus certain cases resemble amyotrophic lateral sclerosis very closely; others again have the general features of a spastic spinal paraplegia, others again show a characteristic Aran-Duchenne scapulo-humeral atrophy. A few cases of general anesthesia are on record, and certain sensory types may be confused with hysteria. Trophic cases with an isolated picture of Morvan's disease are striking, and tabetic-like forms are likewise puzzling. Schlesinger also calls attention to a pachymeningitic type.

The illustrative case of Schmitt and Baraban, which is not unique, shows the striking variability that may be present in the symptomatology of this disorder. At various times this patient was diagnosed by competent authorities as tabes, chronic diffuse myelitis, amyotrophic lateral sclerosis, and spastic paraplegia of unknown causation.

Differential Diagnosis.—The chief disorders that come in review are atypical multiple sclerosis, amyotrophic lateral sclerosis, tumor of cord with spastic paraplegia, diffuse sclerosis, progressive muscular atrophy, central myelitis, syphilitic meningomyelitis, polyneuritis, leprosy, pellagra and ergotism.

The *course* of the disease is very chronic. Large cavity formations involving areas controlling the functions of the intestines and bladder soon lead to death in from four to six years; whereas if the lesion does not compress the entire cord and lies outside of the more vital nuclei the patients may live thirty to forty years. Dejerine has reported a case of fifty years' development.

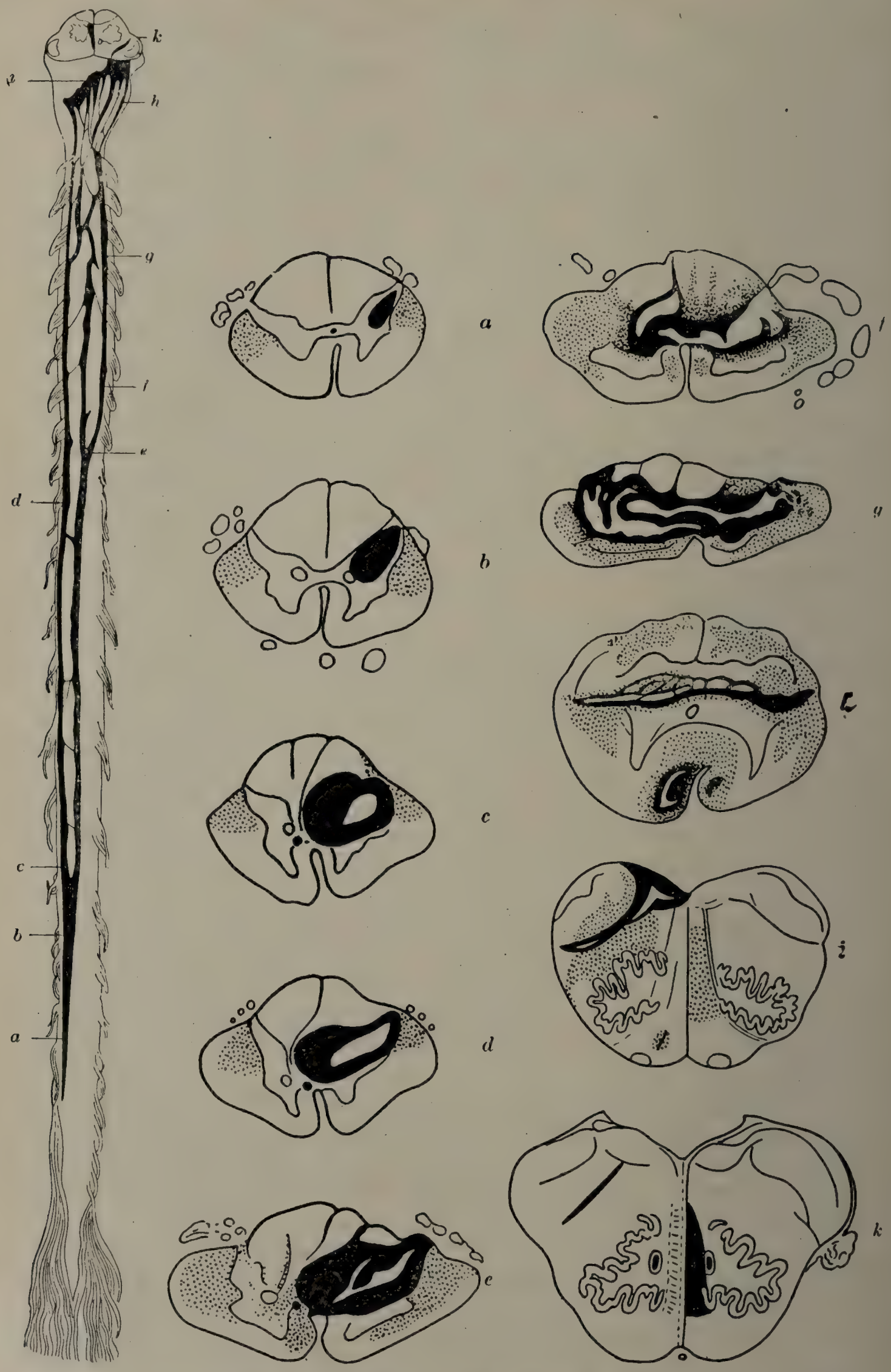


FIG. 303.—Partly schematic representation of a widespread syringomyelia, showing the lesion throughout the cord and at various cross levels of the same. (J. Hoffmann.)

The patients die more often of intercurrent disease; tuberculosis in particular. Bladder sepsis, with kidney complications, is also frequent.

The patients frequently show an up-and-down course, quite analogous to the course seen in multiple sclerosis.

Pathology.—Syringomyelia, pathologically speaking, is due to a cavity in the cerebrospinal axis not in genetic relation to the central canal—dilatations of this latter structure are better termed hydromyelias. Cavities due to hemorrhage are recognized as being different from those in syringomyelia proper and are classed with the hematomyelias. On opening the spinal canal, which is rendered difficult by reason of the vertebral deformities, the dura is usually found to be normal; occasionally a pachymeningitis complicates the picture. The pia is usually thickened, and evidences of swelling or of some internal irregularity are seen either as paths of grayish degeneration, or cystic-like irregularities, with flattening in places. The posterior fissure frequently appears deeply sunken in the cord, causing, as Thomas says, a double-barrelled-shotgun appearance. The cord collapses on cutting and clear fluid escapes. Repeated section, best made after hardening of the cord, shows the existence of one or several cavities, of variable length and diameter, and occupying various situations in the cord. Its most frequent site is behind the neighborhood of the central canal, usually involving the posterior commissure, and with a tendency to reach backward more than forward. The cavity seems to have a special fibrous wall, which is well limited, smooth or papillated; often a gliomatous mass fills the lower end of the canal. In the hydromyelic type the cavity is round and usually occupies the center of the cord. The cavity is lined first by a layer of epithelium, and is surrounded by a gliomatous wall.

In the syringomyelic type there are also some ependymal cells, but they are less regularly arranged. These are interspersed with neuroglia cells and rest upon a solid wall of glia cells, many of which are in process of disintegration. Small vessels are frequent, among which may be found many undergoing hyaline degeneration. The picture is different at every level, and in many sections the cavity has no lining at all in places, bordering directly upon the nervous tissues. Sections through the glioma show perhaps no cavity at all. Fresh hemorrhagic remains of old hemorrhagic foci are frequent findings.

The glioma may be sharply delimited—central gliosis or the neurogliar tissue may infiltrate the cord in all directions—diffuse gliosis.

Secondary degeneration in the parts impinged upon or invaded takes place by process of atrophy and then of tissue replacement with characteristic neurophagia. Regenerated fibers are also encountered.

In cases complicated with pachymeningitis one finds the lesions of this process, and in the traumatic forms one usually finds the remains of an ancient fracture, with inflammatory thickening of the meninges

and pia. The cavity is usually posterior, may traverse the entire cord, midbrain, and even be found in the cerebral hemispheres,[1] constituting a true syringomyeloencephalacia.

Pathogeny.—No unanimity of opinion has yet been reached. The present general trend is to regard the process as degenerative rather than one resulting from a productive inflammation.[2] The general hypotheses are: (1) it is due to a defect of development; (2) it is due to an intramedullary tumor formation which later breaks down; (3) it is the left-over remains of an inflammatory (chronic myelitis) or hemorrhagic process (hematomyelia); (4) it is due to a modification of pressure in the ependymal canal, brought about by a trauma, a compression. In general it seems that no one hypothesis can explain all of the cases.

Thus, according to Schlesinger, one divides the cavity formations that may occur in the spinal cord as follows:

1. Cystic formation, after
 - (*a*) Traumatic destruction of tissue,
 - (*b*) Traumatic hematomyelia,
 - (*c*) Non-traumatic hematomyelia.
2. Softening of an inflammatory or non-inflammatory nature with short course.
3. Syringomyelias:
 - (*a*) True hydromyelia (as malformation),
 - (*b*) True tumor with cavity formation,
 - (*c*) Syringomyelic gliosis,
 - (*d*) Syringomyelia from vessel disease without gliosis,
 - (*e*) Pachymeningitis and leptomeningitis with cavity formation.

In a similar manner the cavity formations of the medulla may be classified as follows:

1. Cysts following softening or hemorrhage.
2. Softening of inflammatory nature with acute course.
3. Cavity formation following degeneration of tumors.
4. True syringobulbias and syringoencephalias (in typical localities).
 - (*a*) Embryonal, lying in the center, and in combination with hydromyelia or syringomyelia.
 - (*b*) Without combination with hydromyelia or syringomyelia.
 - (*c*) Arising in later life and lying laterally as continuation of a syringomyelia due to circumscribed bulbar or cerebral changes.

Treatment.—Since hemorrhage into the glial cavities is a frequent occurrence in syringomyelia, it is advisable to avoid hard, muscular work, since such seems to favor bleeding. Severe muscular effort also aids in spontaneous fractures. Occupations involving heat are to be avoided because of the danger from burning.

[1] Spielmeyer: Ztschr. f. Neurol. u. Psychiat., **25**, 543.

[2] Spiller: Jour. Nerv. and Ment. Dis., 1916. Bremer (loc. cit, 1926) emphasizes the view that "essential" syringomyelia is a dysraphia (Bielschowsky, Henneberg), *i. e.*, a failure in the closure of the embryonal neural tube (Hypothesis I).

For the pains, the treatment is symptomatic. Here the various analgesic antipyretics may be employed. Aspirin, acetanilid, antipyrine in 5-grain doses are sufficient.

Specific therapy is as yet unknown. One is justified in recommending a spinal cord operation in the case of large hemorrhages with sudden increase in pressure symptoms. Here the principle of open expression of the clot can be practised to advantage (Elsberg).

Certain advantageous results have been reported from the use of high-frequency currents. These, however, have not been employed long enough to determine their absolute efficiency. Beaugard and Lhermitte[1] recommend weekly applications of penetration rays, 79 (radio chronometer of Benoist), dose of 3 H, at a distance of 15 per cent. between anticathode and the skin. Twenty-six séances are recommended.

MULTIPLE SCLEROSIS SYNDROMES.

Historical.—Because of the striking variability in the symptomatology of this syndromy, it is not altogether surprising, historically considered, that the first clues to its partial isolation should have been gained from the pathological rather than from the clinical side.

Under the older nosological schemes one finds these patients grouped as epilepsy, paraplegia, tremor, chorea, dance of St. Guy chronica, and a host of other conditions. Although Sylvius de la Böe recognized an intention tremor as different from other types, it cannot be said that the clinical conception assumed its modern form until the work of Vulpian (1862), Ordenstein (1868) and Charcot. The initial pathological notion was given, however, by Cruveilhier (1832–1845) in his famous case of Darges (in 1840), a cook in the Salpêtrière, a sketch of whose cord is here reproduced. Carswell's picture, appearing in 1838, is probably the very first illustration of this condition on record. From this time on, brain and spinal cord sclerosis became an active subject of discussion, in which the work of Frerichs[2] and Valentiner[3] stand out prominently. The former made a diagnosis of brain sclerosis during life and found multiple sclerotic patches at an autopsy, and tried to erect a clinical entity with differential pathological diagnosis, which later was amplified by his student, Valentiner, who collected a series of cases resembling those of Frerichs, and who also gave a remarkable summary of the symptoms. When these reports are read it may be seen that they would not be included within the modern concept, yet Valentiner picked out the facts of involvement in early years, the marked motor disturbances, hemipareses, tremors, speech disturbances, the remissions, the unilateral onset, the greater involvement of motor than sensory function, the involvement of the cranial nerves, the long course and bad prognosis. The mental

[1] Sem. méd., 1907.
[2] Ueber Hirnsclerose, Arch. f. d. ges. Med. Haeser, 1849, p. 334.
[3] Ueber die Sclerose des Gehirns und Rückenmark, Deutsch. Klinik, 1856, p. 147.

symptoms as outlined by Valentiner are not as characteristic. A diplegic idiot, for instance, is included in his series of 15 cases.

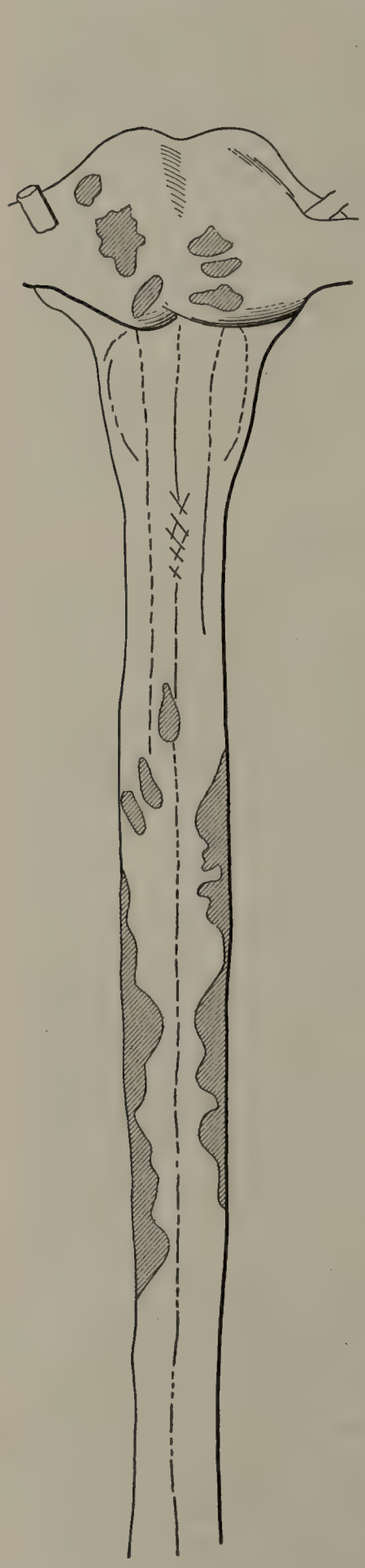


FIG. 304

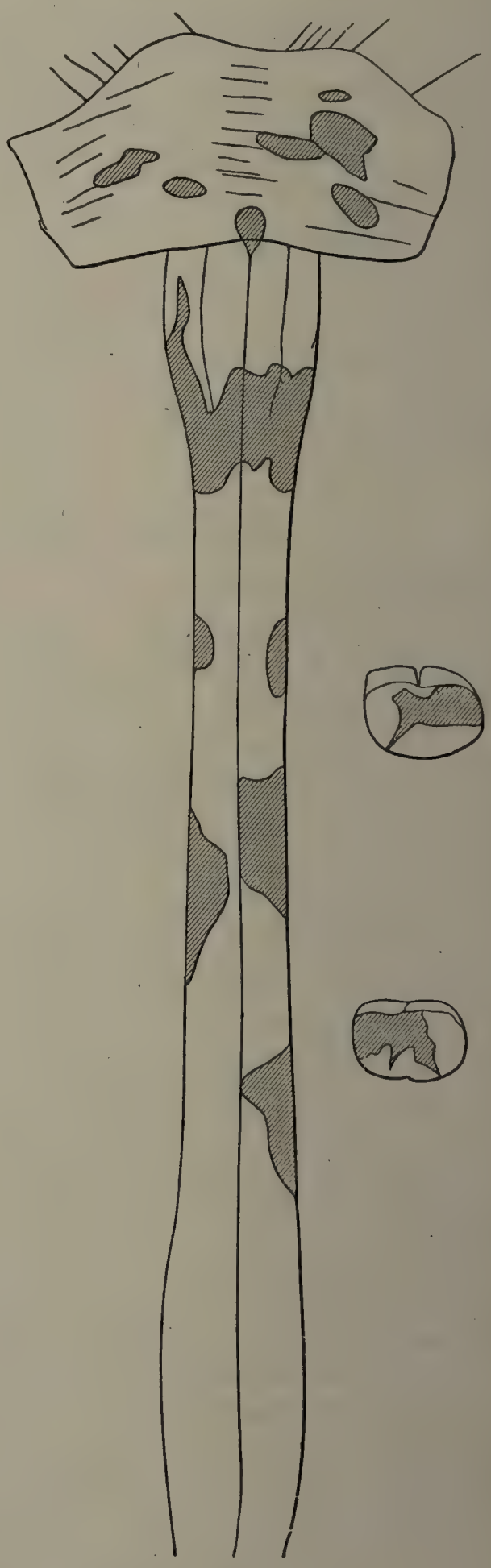


FIG. 305

FIGS. 304 and 305.—Early sketches by Carswell and Cruveilhier (1838 and 1840) of the sclerotic patches in primary multiple sclerosis.

The first real foundation of the symptomatology may be said to have been laid down by these two authors, and little practical progress

was made until the work of Vulpian, Ordenstein and Charcot (1862-1869) and his pupils, as summarized in the monograph of Bourneville et Guerard (1869), when the multiple sclerosis of the present day assumed definite form. As early as 1862 Vulpian and Charcot brought the classical triad, *scanning speech*, *nystagmus*, and *intention tremor*, into prominence as diagnostic of the condition, and in the later studies of the Charcot school the clinical and pathological foundations were laid much as they were held up to within comparatively recent times.

It was becoming more and more evident, however, that the picture insisted upon by Charcot was not a satisfactory one. Sensory pictures, neglected by him, had to be reckoned with in the later studies; also interference with the bladder. Decubitus as a symptom occurs. Then cerebellar symptoms were recognized; then Erb's spastic paralysis fell largely into the multiple sclerosis group; then this same author's chronic dorsal myelitis with optic changes. Hemiparesis not due to embolism or thrombi became recognized. Alternating Millard-Gubler types were described. Then many bulbar palsies passed into its confines; pontine encephalic pictures, pure ophthalmoplegias and occasional forms with painful attacks, resembling syphilitic meningomyelitis also. Amyotrophic lateral sclerosis (Dejerine) and pseudoparesis were later diagnostic possibilities, showing the extreme multiplicity of forms under which this syndromy may show itself. A large number of neurosyphilis cases show multiple sclerosis syndromes and epidemic encephalitis left a number of multiple sclerosis pictures in its wake, as has also poliomyelitis, and influenza.

One of the best documented monographs is that of Müller, 1904. The literature to 1913 is gathered by Wolhwill[1] and Marburg[2] and by Brown for the Society of Neuropsychiatric Research, N. Y., 1921, and the Fifth International Reunion of the Paris Neurological Society of 1924[3] presented extensive reports (Veraguth and Guillain and numerous studies). The histology has been exhaustively treated by Dawson,[4] Klingman, Spiller, and Hassin.[5]

The problem narrows itself to a question whether there exists a multiple sclerosis due to one etiological factor, with a uniform pathology, and other clinical types resembling the former so clearly as to be clinically indistinguishable, but in which one finds multiple tumors, multiple endarteritic lesions (syphilitic, arteriosclerotic), multiple encephalomyelitic inflammatory processes, etc., as etiological factors. The present description follows this conception: namely, there is a primary endogenous picture of unknown etiology and fairly consistent pathology, and a number of secondary syndromes of exogenous multi-

[1] Ztschr. f. N. u. P., ref., 1913, vol. **7**.
[2] Lewandowsky's Handbuch, 1911, vol. **2**.
[3] Rev. neurol., 1924, **31**, 629.
[4] Proc. Royal Soc., Edinburgh, 1916. Also Review of Neurology and Psychiatry.
[5] Am. Archives of Neur. and Psychiat., 1919, **1**, 193 and 210. Also see this Journal for some papers published of the Society for Neuropsychiatric Research, May, 1922, and Jour. Nerv. and Mental Dis., May, 1922 issue.

form etiology and diverse pathologies. The symptoms are purely matters of localization then. This will be discussed with the pathology.

Primary multiple sclerosis thus delimited is a disorder of adolescent or young adults, for the most part, beginning very gradually, advancing slowly, initially very varied, but ultimately assuming a very characteristic, almost stereotyped character. It is characterized by beginning muscular weakness, with spasticity, by disturbances in speech, nystagmus, intention tremor, by forced laughing or crying, and by temporal pallor of the retina. Sensory symptoms may be present but are not usually prominent, bladder symptoms are not uncommon, while vasomotor and trophic disturbances are comparatively infrequent. The process persists for many years, shows striking remissions—noted by Valentiner in 1856—and the patients usually die of exhaustion or intercurrent disorder. Occasionally the process comes to a standstill. In rare instances the disorder is characterized by running a very rapid course—acute disseminated sclerosis of Marburg. Of late years there has been a tendency to relegate this acute syndrome to the encephalitides.[1]

Multiple sclerosis in this limited sense is a relatively infrequent disease. Of 18,000 cases of nervous disease at Vanderbilt Clinic it was diagnosed as occurring 27 times, *i. e.*, a percentage of 0.001 + per cent. In European clinics it would appear to be more frequent. Bramwell-Williamson show 2 per cent. Possibly better diagnoses account for these differences, since the American statistics are obtained from polyclinic material. These are not checked by autopsy findings, and are suggestive rather than conclusive of its frequency. Personal figures show 1 patient in 200 of somatic nervous disorder, 0.5 per cent. Veraguth[2] estimates from a collective study of Swiss material that at least 1 in 3000 of the inhabitants have this syndrome. Thus it is more frequent than neurosyphilis.

Etiology.—The etiology in the *primary* endogenous type, if it exists, is unknown. The *secondary* types show a multiform etiology. Veraguth[3] terms it one of the great enigmas of neurology. Practically all of the infections have been held responsible. Metallic poisons, Zn, Cu, Mn, give rise to clinical pictures at times closely resembling multiple sclerosis, also barbitol. Trauma has been held responsible; so also have sexual excesses, cold and wet and child-bearing.

Heredity may be an important factor, according to many (Pelizeus and others), and Strümpell contends that the essential feature in the primary type is a congenital factor, which develops after any one of the exogenous factors. Müller's critical summary would seem to exclude practically all the exogenous causes, since they occur in but a very small proportion of his cases. He concedes that a secondary multiple sclerosis, in the sense already outlined, may possibly follow infections, but that multiple sclerosis, in the narrow sense, develops only on the basis of a congenital predisposition. The recent encepha-

[1] Bill: Deutsch. Ztschr. f. Nervenh., 1922, **73**, 26.
[2] Loc. cit. [3] Loc. cit., 1924.

litis epidemic left a number of multiple sclerosis syndromes in its wake, thus widening the conception of the secondary types. The newer findings of Kühn and Steiner[1] add another etiological factor to the exogenous group. The many negative findings only serve to show that we are dealing with a polyetiological picture. To claim "multiple sclerosis" to be solely *due* to a "spirochete" is as logical as to assume that all "spastic paraplegias" are due to syphilis.

Sex plays practically no part. Both the results of Charcot's and Whitoff's studies, which spoke for greater frequency in female and male material respectively are due to their particular clientele. Polyclinic statistics nearly always show a greater frequency of women, because they are freer to visit dispensaries, and usually go to all of them in a big city. The chief reliable statistics are those of Müller, which were controlled by autopsy. They show practically no difference.

Age.—Three-fourths of the carefully observed cases occur between the years of twenty and forty. In our own statistics[2] two-thirds of the patients were under forty. Cases have been reported in children as young as five months, and in adults as old as seventy-five to eighty years, but for both extremes diagnostic mistakes are not ruled out. Marie's view that multiple sclerosis is common in children has not withstood the severe critique of autopsy material, and Müller takes the stand that the primary disease is found extremely seldom in very early youth. The initial stages, however, may be traced very often to the years of adolescence or early adult life. Occupation plays no role that is yet known.

Symptoms.—Here is described the general symptomatology of the different multiple sclerosis syndromes. The fact that the numerous sclerotic patches are fortuitously distributed in various individuals makes it possible for almost any combination of neurological signs, thus the greater the number of cases studied the richer has become the symptomatology. Certain patients show comparatively few symptoms for years; others show additions almost from month to month, until they become veritable neurological museums, with signs of involvement from the frontal poles to the tip of the cauda equina. No two patients are alike, yet most seem to attain the same level in the end and in the so-called primary endogenous form come to present a stereotyped picture.

In this picture the most striking feature is the involvement of the motor system, giving rise to muscular weakness, hypertonus and general spasticity in facial, arm, trunk and leg segments. The extreme range is the feature that characterizes multiple sclerosis in its fully

[1] Kühn v. Steiner (Med. Klin., 1917, No. 3) and Siemerling (Berl. klin. Wchnschr., 1918, No. 12), claimed to have found spirochetes in the multiple slcerosis nodules. Other observers do not find them. See Noguchi and Collins: Am. Jour. Med. Sci., 1923. This only shows we are dealing with a syndrome and not a "disease" in the sense of measles. Strümpell (Neurol. Centralbl., 1918) among them. See Abstract, Jour. Nerv. and Ment. Dis., July, 1919. Also complete discussion by Da Fano: Jour. Nerv. and Ment. Dis., 1920, **51**, 428.

[2] Jelliffe: Jour. of Nerv. and Ment. Dis., 1904, p. 446.

developed form. One finds isolated or group phenomena all over the body just in the same proportion as the fortuitous distribution of the sclerotic patches is isolated or diffuse. For this reason it has been thought advisable to begin the description of the symptoms with those of the cranial nerves, and proceed systematically throughout the nervous system. The older and classical triad of Charcot is only partially true, and today a multiple sclerosis may be diagnosed from a temporal pallor of the optic disks combined with bladder incontinence in the absence of nystagmus, intention tremor, or scanning speech.

The classical picture of Charcot may be found in not more than 15 per cent. of all the cases, at a period when the presence of other symptoms permits one to make a diagnosis of multiple sclerosis. If one waits for the "classical" picture, one may have to wait for years. Many patients with true multiple sclerosis never develop the Charcot "triad."

A general chart of the clinical signs in 141 cases is here represented.[1]

SIGNS AND SYMPTOMS OF MULTIPLE SCLEROSIS IN THE ORDER OF THEIR DIAGNOSTIC IMPORTANCE BASED UPON THE STUDY OF 141 CASES.

	Per cent
1. Easy fatigue, weakness and stiffness of one or both upper or lower extremities culminating in spastic paraplegia	81.7
associated with:	
(*a*) Increase of deep reflexes	90.0
(*b*) Positive Babinski sign	78.3
2. Nystagmus, generally horizontal, slight at the beginning, gradually becoming more marked	70.0
3. Ataxic tremor of upper extremities and tremor of the head	55.3
4. Marked diminution or loss of abdominal reflexes	83.7
5. Spastic ataxic or ataxic gait and station (including the Romberg symptom)	43.2
6. Scanning speech or some form of dysarthria	36.0
7. Pallor of optic disks, especially of the temporal halves	32.6
8. Disturbance in facial innervation, often very slight	32.6
Deviation of tongue	10.0
Disturbance of deglutition	3.5
9. Explosive laughter and emotional instability	17.0
10. Unusual remissions often leading to disappearance of symptoms	42.0
11. Transitory ocular palsies with diplopia	29.0
12. Vague objective and subjective sensory disturbances:	
(*a*) Objective (1) Posterior column disturbance	17.0
(2) Pain, touch and temperature disturbance	16.3
(*b*) Subjective: Numbness, tingling and pain	30.0
13. Disturbances in vesical (not rectal) reflexes	40.0
Hasty or delayed micturition; incontinence in 21 cases	14.0
14. Tenderness of the spine, chiefly mid-dorsal	12.0
15. Dizziness (vestibular vertigo)	8.25
16. Mental changes	15.6
17. Auditory nerve involvement	2.0

Important Negative Signs and Symptoms.

1. Pupils generally active with occasional hippus.
2. Biological tests negative except for the occasional presence of globulin and slight increase in the number of cells in the spinal fluid in a few instances.

Colloidal-gold curve reported as paretic by some writers. Not sufficiently studied in this series.

[1] Sachs and Friedmann: Jour. Nerv. and Ment. Dis., May, 1922; Association for Research, New York, Hoeber, 1922.

Olfactory.—Hallucinations of smell are occasionally found but belong to the rarer and infrequently recorded psychical signs. Bilateral anosmia has been recorded.

Optic.—The researches of Uhthoff have emphasized the frequent occurrence of changes in the optic disks. About 50 per cent. of the patients show changes in the papillæ, and it is striking that these changes occur early. They are in the nature of partial atrophies, and show themselves for the most part as a simple atrophic pallor of the disk—usually most marked temporally. True optic atrophy is found in some of the cases, and again in still fewer a true papillo-edema or choked disk. This usually recedes and either leaves no sign or an atrophy. A great number of variations are known.

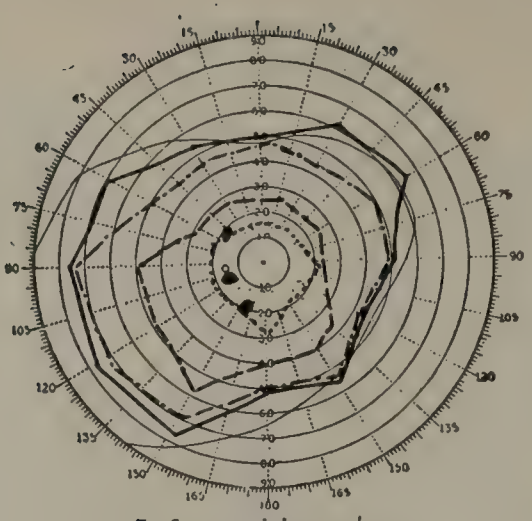
The sight is frequently affected, sometimes blindness, partial or complete, unilateral or bilateral, is an initial symptom. This not infrequently clears up. Complete blindness with double-sided atrophy is extremely rare (Gnanck-Uhthoff). Parinaud has made three classical types, but later studies have shown that there is no uniformity, and the variations in sector blindness and dimness are very many. The loss or diminution in sight in multiple sclerosis is characterized by its advancing and receding character. As a rule, the ophthalmoscopic picture is usually more pronounced than the symptoms. Brouwer has offered a phylogenetic hypothesis for the early involvement of the temporal atrophies.[1]

The fields of vision more often show central scotomata with peripheral clearness than peripheral scotomata. Unilateral scotomata, quadrant scotomata, and a great variety of other forms are known as well as a certain amount of variability from time to time in the individual case. The color fields show similar variations and variabilities. Optic hallucinations also occur. (See Figs. 306 to 309.)

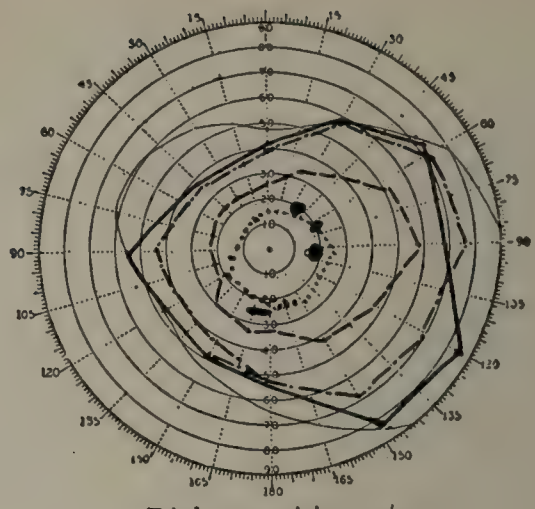
Ocular Symptoms.—The characteristic phenomenon here is nystagmus. Its occurrence as a symptom of "brain sclerosis" was first pointed out by Valentiner (1856) and Charcot overvalued its diagnostic importance. True central nystagmus is rare, but nystagmoid movements on lateral motion of the eye occurs in from 70 to 80 per cent. of the cases, especially in the later stages of the disease. Vertical, oblique, rotary nystagmus is included with the other forms. In cases with pronounced cerebellar involvement the nystagmus is of this type, and rotation of objects with subjective rotation and forced positions are to be expected. These eye movements may be summarized as: (1) continued rhythmical oscillations (true central nystagmus) analogous to the continuous movements of the head and body; (2) rhythmic oscillations set up on movement of the bulb in any direction, analogous to the intention tremor of the hands; (3) nystagmoid movements on extreme lateral or vertical movements, analogous to fatigue (paresis) movements; (4) ataxic movements. Of these

[1] Brouwer (Jour. Nerv. and Ment. Dis., 1924) contributes an important paper concerning the involvement of late acquired functions in the nervous system. Temporal pallor, abdominal reflexes, pyramidal signs, etc.

FIG. 306

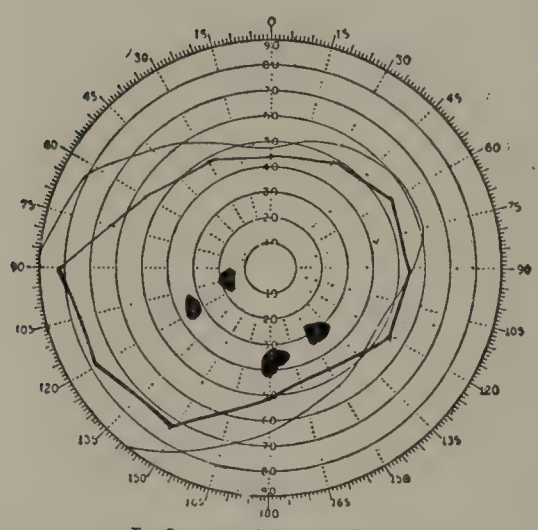


Left eye vision 20/30

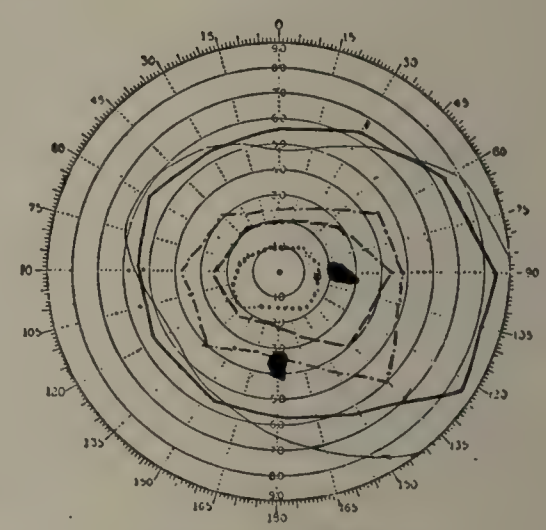


Right eye vision 20/30

FIG. 307

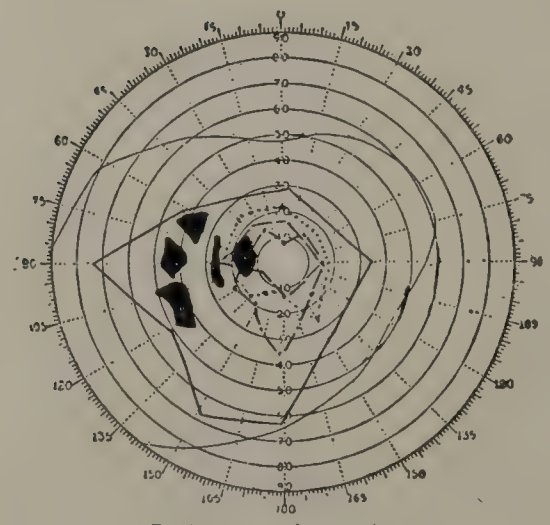


Left eye vision 15/40

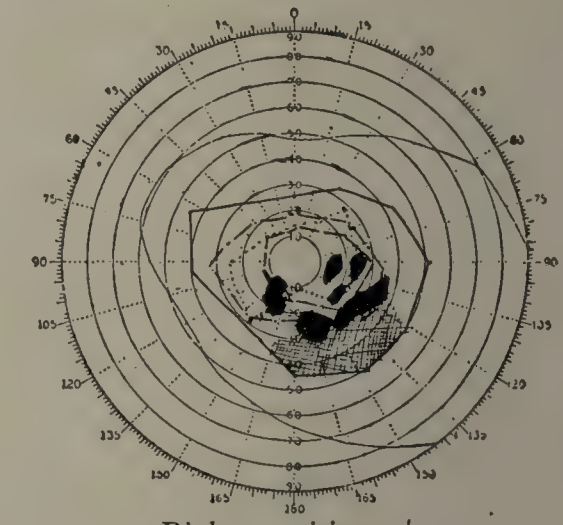


Right eye vision 15/40

FIG. 308

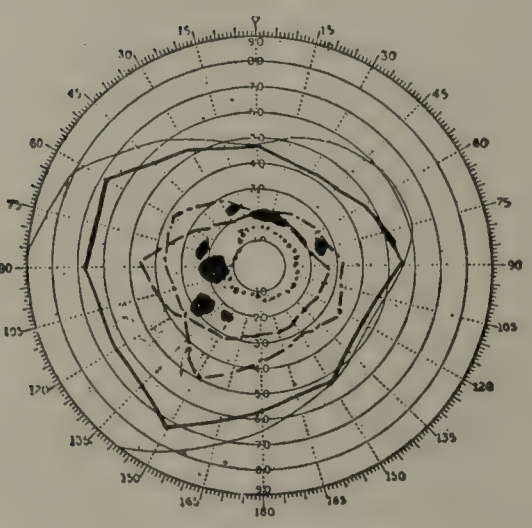


Left eye vision 15/30

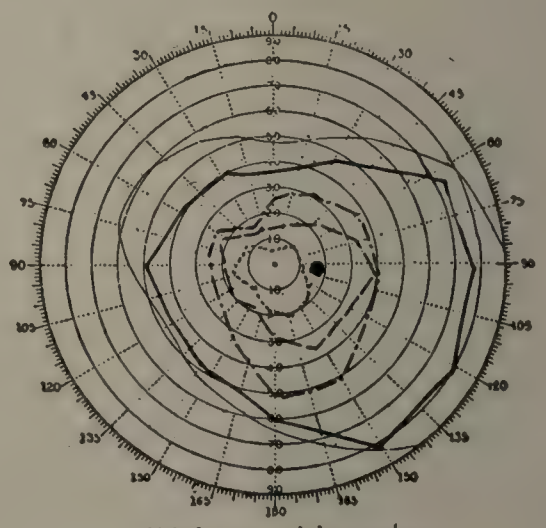


Right eye vision 15/50

FIG. 309



Left eye vision 20/40



Right eye vision 20/30

Field of vision ——— White -.-.-.-. Blue

Green for 5 mm. □

Relative scotoma ▨

FIGS. 306 to 309.—Irregular scotomata pictures seen in multiple sclerotic patients. (Klingmann.[1])

[1] Jour. Nerv. and Mental Disease, 1910.

(3) is the most frequent; practically all are conditioned by central disease, although peripheral involvements are described.

Eye Muscle Palsies.—These are important in diagnosis, and present themselves infrequently as isolated or complete palsies, transitory or persisting. Paralysis of convergence is more often found. In general, from 17 to 46 per cent. of the patients show palsies, the statistics varying according to the more or less strict interpretation of paralysis. Uhthoff demands "double vision" as a criterion, which symptom is not an infrequent early occurrence in multiple sclerosis. Müller, on the other hand, admits milder palsies to his statistics (46 per cent.)

Ptosis is less frequently found (8 to 15 per cent.), usually one-sided, occasionally double, usually incomplete and ephemeral. One also meets with external ophthalmoplegia, abducens and trochlearis palsies. Divergent palsy has been noted by Bielschowsky. Complete internal ophthalmoplegia has not yet been recorded.

Pupillary inequalities are not infrequent (24 per cent.—Müller), and these are changeable and frequently early. They may represent spasms or paresis of muscles. Miosis is frequent in the later stage (Parinaud), but the reflexes are usually normal. Pupillary unrest (hippus) is found (Frankl-Hochwart). Argyll-Robertson pupil was found only 4 times in 364 cases. One case of Uhthoff was controlled by autopsy; 2 cases by Rad,[1] Wassermann control.

V. Trigeminus.—Facial neuralgia has been observed as the first symptom of a multiple sclerosis (Oppenheim), but involvement of both the sensory and the motor branches is rare.

VII. Facial.—Facial palsies, usually associated with other central (hemiplegic) pontine (alternating palsy), or pressure symptoms, occasionally coming on very acutely, are not infrequent (15 per cent.); as an isolated palsy it is rare. Like other palsies their fluctuating character is striking, the severe permanent types being rare (Bouchaud), as are also bilateral palsies.

VIII.—Hearing is transitorily affected. Truffert, in Guillain's report, found the cochlea intact. Complete or partial deafness has been recorded, as well as the occurrence of noises. Auditory hallucinations are possible, but not anatomically proved. Hyperesthesiæ are frequent.

The vestibular nerve of the eighth pair is not infrequently involved in its cerebellar connections. An explanation of some of the more severe nystagmus movements may be found here. Giddiness and dizziness are very prominent symptoms, and occur either from involvement of this nerve or other sensory cerebral or spinal tracts. Rotatory giddiness with tendency to fall, revolving of objects, etc., is present in a few cases of multiple sclerosis, and certain Ménière cases belong here.

[1] Neurol. Centralbl., 1911, **30**, 584.

X.—Pneumogastric involvement is rare. Müller reports a case of paroxysmal tachycardia in multiple sclerosis. Dyspnea is also known, but is infrequent.

The *taste* has been modified in a few cases.

Speech Disturbances.—Charcot's typical speech disturbance was of a slow, monotonous, and scanning character. This special type is present, however, in only a small proportion of the cases, and then usually only in the later stages. Müller's autopsy-controlled material (81 cases) gave only 25 per cent. of this character, whereas patients with other types of speech disturbance run higher—25 to 30 per cent. —making about half of all multiple sclerosis cases with some disorder of speech. Guillain allies the scanning speech disturbance to a cerebellar asynergia and finds it relatively infrequent. Friedmann and Sachs[1] find it in 36 per cent. of their 141 cases. Easy fatigability, with increasing unsteadiness; stuttering, with accentuated mouth movements, are other types.

Singers soon notice these alterations, especially the fatigability, and an increasing inability to modulate the tones. Dysarthria and explosive speech are obtained in later stages. Articulatory disturbances with repetition of syllables, or words, is frequent in later stages —*r*, *l*, *p* and *g* are particularly difficult. Aphonias or other laryngeal complications are occasionally met with (Rethi). The tongue occasionally shows mild transitory palsies, with mild atrophies, usually unilateral and fibrillary twitchings. Ataxic movements of the tongue are frequent in the later stages. Occasionally subjective sensations of fulness, thickness and difficulty in movement are observed.

Chewing and swallowing are involved, the former rarely, the latter not infrequently, less often transitorily as an early symptom, more often as a permanent terminal sign. Excessive salivation seems very infrequent.

Motor Disturbances.—The most characteristic are the intention tremor, ataxias, and palsies with later developing atrophies and contractures. Dysmetrias and asynergias, are infrequent, adiadocokinesias frequent.

Ataxia.—By the finger-nose test or the finger-finger test, and by the static position one can demonstrate an ataxia in the upper extremities, separate from an intention tremor, in a large number of the cases (Müller, 70 per cent.). A similar ataxia may be shown in the lower extremities in even a more striking manner in testing by the knee-heel test. The ataxic movements usually precede the development of the intention tremor, and are later often covered up by the same. Occasionally the ataxias are increased on closure of the eyes, occasionally not, showing that at least two types are to be observed. Recent findings in the sphere of deep sensibility, position, sense, etc., afford an interpretation of these ataxias. They vary clinically from the tabetic ataxia, particularly in the increased tempo of the individual move-

[1] Loc. cit.

ments and the irregular contraction of the agonist muscles.[1] The usual hypertonus of the multiple sclerosis patient is in marked contrast to the hypotonus of the tabetic. Crossed hemiataxias are recorded.

Intention Tremor.—Present in from 50 to 75 per cent. of the cases, this symptom is one of the most striking in well-advanced multiple sclerosis. It may be absent for a number of years, and yet a diagnosis may be made on other grounds. Considerable care must be exercised in not confusing an intention tremor with an ataxia, and *vice versa.* Certain shell-shock tremors observed in warfare have closely resembled this intention tremor. Müller has laid considerable stress upon this point, and because of his more rigid criteria regarding the differences, states that real intention tremor was present in only 25 per cent. of his cases. Marquézy, in 14 per cent., Friedmann and Sachs (loc. cit.), in 99 per cent., Guillain (loc. cit.) counts it among the infrequent symptoms. This intention tremor is increased by prolonged effort and by emotional disturbances. It is not markedly increased by shutting the eyes.

It develops gradually, occasionally suddenly after an apoplectic attack, is more often bilateral, corresponding closely with the pareses. The arms are more frequently involved, then the trunk, and then the head; rarely the chin and muscles of the face.

Only exceptionally are the legs markedly affected by an intention tremor. In the trunk this disturbance gives rise to a type of continual balancing or rocking motion; it also involves the muscles supporting the head, causing a nodding and swaying series of movements. Passive tremors of the hands when at rest are also observable.

The precise anatomical reasons for the intention tremor are not completely analyzed. Spinal cases do not show it, and the greatest probability is that the lesions causing the intention tremor are those that cut off portions of the cerebello-rubro-thalamic tracts. A complete analysis of the fiber-tract involvements in multiple sclerosis has yet to be made before all questions relating to localization can be answered. (See discussions of striatum athetoid tremors, thalamic syndrome tremors, cerebellar tremors, paralysis agitans tremors in their respective chapters.)

Writing shows characteristic changes due to both the intention tremor and the ataxia. The strokes are irregular—too thick or too thin—and the excursions in forming the letters irregular.

Motor Power.—Motor weakness is usually the earliest symptom noted in multiple sclerosis. It may be the arms, oftener the legs, or occasionally in some special group of muscles; larynx in singers, hand and arm in painters or players, occasionally bladder or rectum. Marked fatigability is an important symptom, and may precede the other symptoms for years.

Spastic paresis, with hypertonus, then develops, and though varying considerably in intensity, often dominates the picture, leading

[1] Breitbach: Deutsch. Ztschr. f. Nervenh., 1921, **72**, 1.

later to the most severe forms of contractures. The type may be hemiplegic or paraplegic, and usually indicates involvement of the pyramidal tracts. The lower extremities are involved much oftener than the upper. Irregular Brown-Séquard paralysis is occasionally present. Oppenheim has recorded a bilateral Brown-Séquard syndrome.

Gait disturbances are common and variable. They may be purely spastic, or in the cerebellar cases, spastic ataxic. Pure ataxic gaits are very rare. A spastic paretic gait marks the pre-bed stage. Occasionally one finds the tumbling or reeling gait of cerebellar tract implication.

Certain motor anomalies are encountered as the patients rise or sit down. A Romberg, which is not much modified on closing the eyes, is not uncommon.

Muscular Atrophies.—These occur, but are rare.

Sensory Phenomena.—Marie's earlier contention that sensory disturbances do not belong to multiple sclerosis has been definitely disproved.[1] On the contrary, they are very frequently found, but largely by reason of their fluctuating character, both as to intensity and extent are overlooked or falsely interpreted. The frequently made false diagnosis, "hysteria," is usually founded on these fluctuating, sensory signs. Hoffman and Freund found sensory disturbances in from 70 to 90, Müller 76 and Keschner and Malamud[2] 80 per cent. of their cases. Pains are not infrequent; paresthesiæ, on the other hand, are very common. Anesthetic or hyperesthetic spots are frequent; the patients frequently complain of dead or numb fingers. Tactile anesthesia is not infrequent; occasional tactile agnosia is present. The sense of localization is frequently faulty, and bony sensibility is also often involved, with diminution of position sense and disturbance of thermal sensibilities. Thermal changes are also observed.

Skin Reflexes.—The abdominal and epigastric reflexes are almost invariably modified (80 per cent.). There may be unilateral diminution or loss, or more often bilateral diminution or loss (62 per cent.). Thus bilateral loss of these reflexes constitutes a very important differential for the diagnosis of not only an organic nervous disease, but especially for multiple sclerosis. Careful studies by Müller have shown that when correctly tested they are modified in only about 5 per cent. of normal individuals, and these occurred most often in women who had had children, or in very obese subjects. The cause is to be sought in an interruption of the sensory tracts, either in the cord, in the median lemniscus, in the thalamic end-stations, or in the post-Rolandic sensory distributions. As Brouwer[3] has emphasized

[1] Jeremias has collected the rich literature bearing on sensory disturbances in multiple sclerosis. Dissert., 1898; see also Müller.

[2] Am. Arch. Neurol. and Psychiat., 1924, **12**, 682.

[3] Brouwer: Significance of Phylogenetic Studies in Nervous Diseases, Jour. Nerv. and Ment. Dis., **51**, 1920, Herter Foundation Lectures, Baltimore, 1927.

the abdominal reflexes are phylogenetically recent and hence, more susceptible to various noxæ.

The cremasteric reflex is also often involved unilaterally, or bilaterally, but less often. Also the vomiting reflex, though not so often.

Babinski's phenomenon is very frequent; Müller says, almost constant. Crossed Babinski may even be observed. Oppenheim reflex is very often, though less frequently, found.

Like all of the symptoms of multiple sclerosis, the skin reflexes are subject to considerable variations. They infrequently return after showing a loss.

Tendon Reflexes.—In concordance with the spasticity and hypertonus, increased tendon reflexes are to be expected. The upper extremities show increased triceps-jerk and increased radio-periosteal reflexes. The Achilles-jerk is exaggerated, unilaterally or bilaterally, as also the knee-jerk, and usually there is unilateral or bilateral ankle-clonus. Patellar clonus is less often observed. Guillain, Girot and Marquézy describe a hyperspasmodicity of the abductor and adductors of the hip. Diffusion of reflex excitability is frequent. Dissociation of the mediopubic and abdominal reflexes is emphasized by Guillain, whose discussion at the Paris Society contains a large number of interesting observations on rare and little studied reflexes. Foix's defense reflexes are often elicitable. Loss of the palatal reflex is described by Guillain and Marquézy.

Vasomotor and Trophic Signs; Other Signs.—One finds a number of anomalies of this group in a large collection of multiple sclerosis cases, but, as a rule, they are sparse and isolated. Among these may be mentioned hyperidrosis, angiospasms (cyanosis), vessel palsies with edema, erythromelalgia-like affections, syringomyelia-like trophic disturbances, abnormal blushing. Hyperpyrexias are encountered with apoplectiform and epileptiform attacks. In the ordinary cases the patients not infrequently show lability of the body temperature. Veraguth[1] also finds this.

Pilomotor reflexes are frequently disturbed; thus dermographia is common, and may show unique isolated localizations.

Bladder.—The bladder is frequently involved (80 per cent.—Oppenheim; 75 per cent.—Müller), often transitorily, and also it may be an early symptom. The patients feel a sense of insufficiency and have to strain to pass their water. Complete paresis with incontinence is rare. There is a great variability, with many ups and downs in the bladder symptoms. Polyuria and glycosuria have been observed.

Obstipation is frequent; loss of control less so, but is present.

Menstruation, childbirth, parturition, etc., are not markedly affected. Genital hyperesthesia is at times pathologically increased and occasionally there is anesthesia with impotence.

Lumbar Puncture.—The experience thus far gained shows but slight changes in pressure. Cytologically, occasionally slight lymphocytosis

[1] Loc. cit.

has been observed, but the majority of the cases have been negative. Serologically a number of variants are found. A *gold curve* similar to that seen in paresis is not infrequent. Guillain's mastic test has been found positive, also the benzoin test. A correlation of all tests should be made.[1] (See tables of Ayers, Cerebrospinal Fluid Examination, pp. 128–131.)

Mental Symptoms.—These are not constant nor prominent, and are for the most part absent, yet careful analysis shows them to be much more frequent than is usually taught. Mild intellectual reduction in the form of a dementia, hallucinations of hearing, of sight,[2] hallucinatory states with mild confusion, passing ideas of reference and of persecution, difficulty in thinking, spasmodic intermittent alternations in the capacity for attention and concentration, lapses of memory, etc.; these have all been recorded. The latter anomalies are not infrequent, and often are colored by depression (melancholic) or excited (manic) states, or more frequently the emotional tone is one of indifference. Pseudoparetic states may be expected at times.

Involuntary Laughing and Crying.—These were described by Cruveilhier in his classical case, and correctly appreciated by Bourneville. They are chiefly neurological signs, forms of paramimia, for neither the laughing nor the crying are necessarily accompanied by their usual emotional states, nor do they necessarily betoken any intellectual reduction. They are very often found, forced laughing being the more frequent (40 per cent.—Müller). They are both to be referred to implication (in part) of the cortico-bulbo-cerebellar reflex paths. An analysis of all of the variations is not possible here.[3]

Apoplectiform and Epileptiform Attacks.—These occur but not as frequently as was held before one was in a position to rule out anomalous paretic attacks. But the frequent occurrence (20 to 25 per cent.) of mild attacks with transitory disturbances of consciousness, or attacks of giddiness or faintness, with unilateral or bilateral pareses, or sensory anomalies in the distal extremities, should be emphasized. Such mild attacks resembling arteriosclerotic attacks, often mark the initial stages, or accompanying the sudden progressions which are characteristic of this disorder. Epileptiform attacks are much less often observed and usually speak against a multiple sclerosis, but they do occur, and very infrequently show as Jacksonian attacks (Gussenbauer).

Characteristic Forms.—As already noted, the classical signs of Charcot, with nystagmus, intention tremor and scanning speech, in reality are found in only a comparatively small proportion of the cases in the earlier stages of the disease. In the interest of early

[1] Nonne: Deutsch. Ztschr. f. Nervenh., 1910, 1912. Ayer, J. B.: Arch. Neurol. and Psychiat., May, 1922; Jour. Nerv. and Ment. Dis., May, 1922, p. 395. Report of Research Association: Brown, Davis, Jelliffe: Multiple Sclerosis, New York, Hoeber, 1927. Fifth International Reunion, Paris Neurological Society: Discussion on Multiple Sclerosis, Rev. Neuro., 1924, **31**, 631.

[2] Nonne: Mitt. Hamb. Staatskrankenhaus, 1910. Lhermitte: Paris méd., October 18, 1924, with bibliography.

[3] Cottrell and Wilson: Affective Symptomatology of Disseminated Sclerosis, Jour. Neur. and Psychopath., 1926, **7**, 1.

diagnosis, this must be borne in mind, especially as true nystagmus is always rare, nystagmoid movements are to be judged with care, and the tremor and scanning speech are usually later symptoms. The "non-typical" cases in Charcot's sense (formes fruste), are really more frequently met with. Any attempt to state which special group of symptoms occurs with more or less frequency is apt to be misleading, especially in view of the great variability met with and the inconstant changes, the advance of certain symptoms and the retrogression of others.

For purely didactic purposes one can divide the more common symptom pictures as follows:

1. Cases that begin with isolated or prominent cerebral symptoms. These occur less frequently than the spinal cases, perhaps than the bulbar, but hence are also more likely to be overlooked. Here optic atrophies, with defects of vision, ocular palsies, transitory diplopias, crossed eyes, etc., muscular weaknesses are prominent and occasionally associated with giddiness, nausea and headache.

2. Cases with isolated or prominent bulbar symptoms. These begin as mild or severe bulbar palsies, and are rare.

3. Cases with irregular and prominent spinal symptoms, mostly involving the lower extremities. Here the patients complain of the legs becoming tired easily, and after a time they stumble or trip easily. They then become somewhat stiff and walking up stairs becomes increasingly difficult. Mild bladder disturbances may have preceded or accompany the weakness and stiffness in the legs. Paresthesiæ are also frequent. These patients soon show spasticity, increased knee-jerks, clonus at times, Babinski's sign. The abdominal and epigastric reflexes are diminished or absent unilaterally or bilaterally.

This is apparently the most frequent mode of onset, since very often the patients have paid no attention to the rapid passing of transitory diplopias, slight speech difficulties or weakness of the bladder, or mild attacks of giddiness, yet sharp questioning usually elicits some of these other signs as having preceded or accompanied these spastic paraplegic types. Whereas these signs usually come on very gradually, they may appear to have had a sudden onset, as after a long walk, or slight accident, or following childbirth, or other striking intercurrent event.

4. In the vast majority of the cases there is a gradual onset of both spinal and cerebral symptoms. Headaches, diplopia, difficulty in walking, slight changes in speech, giddiness, abnormal muscular tire, paresthesiæ, transitory bladder weakness, etc. These symptoms progress and then recede—usually attributed to the skill of the physician or thought of as hysterical—and then reappear in the same or in a slightly different order.

5. A small number of cases begin with a mild initial apoplectiform attack, and then either progress, or remain stationary for some years.

6. Marburg[1] has described an acute disseminated sclerosis which runs a rapidly fatal course terminating in from three to six months. These are probably epidemic encephalitis cases which are not infrequent.

Non-characteristic Forms.—These unusual forms may be summarized as (*a*) forms which run a more distinctly psychic course, with the picture of slowly advancing dementia and pseudoparetic forms;[2] (*b*) forms that resemble brain tumor—hydrocephalus; (*c*) Jacksonian attacks due to patches in the motor area; (*d*) hemiplegic forms due to patches in the cerebral course of the pyramidal tracts; (*e*) bulbar paralysis and pseudobulbar paralysis types; (*f*) sacral forms as described by Oppenheim, resembling tumor of the cauda equina; (*g*) forms that give an acute onset resembling pontine encephalitis; (*h*) cerebellar types, resembling paralysis agitans (not infrequently misnamed early paralysis agitans); (*i*) forms that give a picture like cerebrospinal syphilis; (*j*) progressive muscular atrophy and amyotrophic lateral sclerosis-like forms; (*k*) tabetic-like forms;[3] (*l*) transverse myelitis and combined sclerosis types, and finally (*m*) latent and recessive forms which are differentiated from irregular psychogenic edemas of the cord, or from pure psychological blockings only after many years of the most exact scrutiny.

Diagnosis.—Enough has been said to indicate how the multiple sclerosis symptom-complex may result from different etiological factors involving the brain and spinal cord. Neurosyphilis is the most important disorder to be ruled out.[4] The leading features in the diagnosis concern the age of the patients, young individuals, the usual lack of heredity, the failure of usual external causes to account for the symptoms, absence of Wassermann reactions and of spinal fluid findings. A fairly constant gold sol finding is present in a certain group of these cases. Of the more important objective findings for the early diagnosis one counts on the early feeling of motor weakness, feelings of giddiness, the onset of spasticity with increased reflexes, clonus and Babinski, the presence of the fundus changes, the loss of the abdominal reflexes, fine ataxic movements of the legs in the knee-heel test, and of the arms in the finger-finger and finger-nose tests. See chapter on Examination.

In the later stages the full pictures as already outlined appear and make a diagnosis certain. One feature of perhaps the most striking importance is the peculiar advancing and receding course, the remissions during which the entire picture seems to fade away and which permit a patient condemned to a chronic organic nervous lesion to turn up in the physician's consulting-room apparently well.

It is for this latter reason perhaps more than any other that the false diagnosis, hysteria, is so frequently made in these cases. Then there are in addition the almost daily fluctuations in the sensory sphere

[1] Loc. cit.
[2] Hermitte: Paris Méd., October 18, 1924.
[3] Breitbach: Deutsch. Ztschr. f. Nervenh., 1921, **72**, 1.
[4] Wolff, H. G.: Disseminated Sclerosis with Syphilis, Boston Med. and Surg. Jour., 1927, **196**, 723.

which always suggest hysteria, and which only a rigid analysis will exclude. Furthermore, it must not be forgotten that not only does one experience the changeability in symptomatology suggestive of hysteria, but a helpful optimistic psychotherapy can most markedly alter the symptoms as well, causing some of them to entirely disappear. The rise in the level of nervous energy by such psychic means seems to enable the patient to force a better control of the disturbances. In the failure of a typical hysterical character, as determinined by a psychoanalytic study of the unconscious symbolizations of the symptoms and their teleological function for the individual, this disorder should not be diagnosed. Finally, one has always to reckon with a symptomatic psychological reduction in the patient's resistance due to the presence of somatic lesion.

Further, diffuse sclerosis, general paresis, brain tumor, chronic hydrocephalus, arteriosclerosis, cerebral syphilis, infantile palsies, encephalitis of a diffuse disseminated type, bulbar palsies, chronic leptomeningitis, infantile ataxias, Friedreich's ataxia in particular, disseminated myelitis, meningomyelitis, Erb's spastic palsy, hereditary spastic palsies, syringomyelia, tabes, spinal cord tumor, paralysis agitans, chronic zinc poisoning, chronic manganese poisoning, chronic mercurial poisoning and spinal cord edemas all come in review in the differential diagnosis of multiple sclerosis. Any or all of these can look like some multiple scleroses at some part of their clinical course. In the majority of cases a diagnostic summary may be soon arrived at, but then the study has only begun.[1]

Pathology and Pathogenesis.—On autopsy in the cases of *primary multiple sclerosis* one finds, as a rule, a normal dura, the brain itself usually shows irregular atrophies with thinning of the cortex and some internal hydrocephalus. At times one can observe on the surface the irregular patch-like areas of atrophy. These usually show much more frequently on the surface of the pons, medulla and cord, as Carswell and Cruveilhier showed early. On section of the brain one finds few (five or six) or many (several hundred) irregular sclerotic patches, which are usually isolated one from another.

Microscopic examination invariably reveals many not seen by the naked eye. They vary in size from that of a pin-head to 5 or 6 cm., which larger patches are usually made up of several smaller ones. The general color tone is gray. The reddish patches usually belong to the secondary encephalomyelitides. The plaques either rise slightly from the surface or are level or show a slight depression; the tissues about are slightly edematous. At times, particularly in the optic nerves, there is distinct shrinkage in the tissues. The consistency is usually tough, or almost leathery. The soft plaques are apt to resemble disseminated myelitis, encephalomyelitis, etc. The patches are very sharply differentiated from the surrounding tissues. As to localization, they may be anywhere; in the brain, cord, medulla,

[1] Steiner, G.: Ueber die gegenwärtigen Stand der Erforschung der multiplen Sklerose, Ergeb. d. inn. Med., 1922, **21**, 251.

pons, cerebellum, the roots of the cranial or spinal nerves, within the optic nerve itself. As a rule they are found in both brain and spinal cord, and seem to have a special fondness for places particularly rich in neuroglia, and there is a certain grade of asymmetrical symmetry in their localization.

In the cord, the median line, and the pyramidal tract region are favorite localizations, and in the white matter more than the gray. Central gliosis seems rare. In the medulla the olivary region, the floor of the fourth ventricle, and the posterior aspects are favorite sites, while in the cerebellum the dentate nucleus is usually most often implicated.

Histologically the patches are made up of masses of glia fibers. There is no areolar composition and the glia nuclei do not appear prominent. Small holes may be found within the plaques, around which the glia fibers cluster as about a bloodvessel. Bielschowsky preparations show the partially intact nerve axones passing through the glia masses in much reduced numbers; the medullary sheaths are partly modified in their qualities. Charcot's idea that the axis cylinders were rarely altered is not confirmed by finer technical studies. They are more frequently involved than the earlier studies indicated. Edema is a frequent precursor of degeneration, and regeneration has not been observed. The immense mass of glia fibrils is an entirely new-growth, apparently not to take the place of destroyed nervous tissue, but a pure addition product, a proliferative inflammation, in which one finds the almost normal elements still present, for a time at least. Secondary degeneration outside of the plaques is not usually present.[1]

What relation the disease has to the vessels is still far from clear, but it seems certain that primary inflammation of the vessels is not a part of true multiple sclerosis. One of the present trends is to view the process as related to a non-purulent encephalomyelitis, allied to a poliomyelitis, rabies, Borna's disease, etc., the exciting agent being some specific organism as yet not isolated. (Spielmeyer, loc, cit.; Schol[2]), whereas another is to view it as a non-inflammatory destruction due to factors making for retrograde degeneration.[3]

Dawson's monumental study seems to show that the sequence of the pathological processes in the disease is somewhat as follows: (1) A commencing degeneration of the myelin sheath and a simultaneous reaction of the glia in the immediately adjacent perivascular tissue; (2) an increasing glia-cell proliferation and a commencing fat-granule-cell formation; (3) the stage of so-called "fat-granule-cell myelitis;" (4) a commencing glia-fibril formation; (5) an advancing and (6) a complete sclerosis. Histological study has given overwhelming evi-

[1] See Dawson's and Klingman's complete histological studies, loc. cit. Also paper by Adams, Blacklock, Dunlop and Scott: Quart. Jour. Med., January, 1924; also consult Steiner, *loc. cit.*

[2] Gesellsch. f. Naturf., Dresden, February 24, 1914, and citations already made.

[3] Hassin and Leiners Studies, Am. Arch. Neurol. and Psychiat., May, 1922.

dence that sclerotic areas in the disease arise on the basis of this evolution through a stage of fat-granule-cell formation, and Dawson concludes that the underlying process is a subacute disseminated encephalomyelitis which terminates in disseminated areas of actual and complete sclerosis. There is much to favor one view that primary disseminated sclerosis is due to a specific morbid agent; other diffuse affections of the central nervous system (arteriosclerosis, syphilis, acute encephalomyelitic processes) may give rise to a clinical symptom-complex very like that of disseminated sclerosis. According to Dawson, the anatomical expression of the remissions often seen must naturally be the gradual clearing up of the cell exudation and a sclerosing of the tissue with a retention of the axis-cylinders. There is no adequate evidence to distinguish between a microbic and a toxic agent. It is possibly the latter. The causal agent is possibly of the nature of a soluble toxin, which is conveyed to the nervous tissues probably through the blood channels.

The suggestion made is that the restriction is in some way related to the selective action of the toxin in certain areas of the blood supply, or that unknown factors determine an irregularly distributed paralytic dilatation, with an increased infiltration through the vessel walls. Remissions and relapses necessitate the assumption of the latent presence of the morbid agent in the body—either the intermittent evolution of a toxin, or its accumulation from deficient elimination. Even Dawson's studies fail to show a real inflammatory reaction, similarly those of Hassin. This whole picture suggests the possibility that vegetative control of the spinal cord metabolism may be at fault. This viewpoint might aid in the understanding of the possible contributory influence of psychogenic factors to certain types in this syndrome, a problem to which Jelliffe[1] has devoted some attention.

The *secondary* or symptomatic multiple scleroses show the pathology of the inducing process. Syphilis, encephalitis, multiple sarcoma, war traumata, etc.

Reference has been made to the researches of Kühn and Steiner and others of the finding of a spirochæte and the experimental work of Bulloch (Gye) and others. Petit has reported the finding of a spirochæte in cerebrospinal fluid. Guillain[2] has reviewed the evidence carefully *pro* and *con* and reported his own experiments. They were negative. Also those of Noguchi and Collins upon monkeys. The disease induced in a guinea-pig or rabbit by inoculation is very flimsy evidence and this whole type of research thus far lacks sound principles.

Prognosis.—In general the disorder is progressive, yet there are many stationary cases, and some few that apparently recover. Polyetiological considerations may explain these variations. Multiple sclerosis, seen as a whole, is not one disease, but a symptom-complex due to different etiological factors. Many patients extremely ill

[1] Jelliffe: Am. Jour. Med. Sci., May, 1921; Jour. Nerv. and Ment. Dis., May, 1922.
[2] Loc. cit., also see Steiner, *loc. cit.*

and bed-ridden, following one of the acute advances of the disease, recover almost completely; but usually there is a later acute advance and then others. The time between advances or the length of remissions cannot be stated; they have varied from six months to ten years; some very rare remissions show a period of twenty years.

A few patients die rapidly of the disorder (six weeks to six months) —acute multiple sclerosis (encephalitis) of Marburg, Frankl-Hochwart and others—but the majority live for many years, and die usually of intercurrent disorders, pyelitis, tuberculosis, pneumonia, etc. Müller's average was four years; Charcot's, six to ten years.

Therapy.—Specific therapy is not yet known. Prophylaxis also seems difficult to grasp since no definite exciting cause is known. Relative prophylaxis in the shape of advising against marriage for young affected individuals, or against child-bearing in the married is necessary. Childbirth would seem to be an exciting cause for an active exacerbation.

Rest in bed is more than desirable in acute stages, it is imperative. The rest must be absolute, and should include eye rest. Active remedial treatment—massage, hydrotherapy, electrotherapy—are all disadvantageous. Warm baths for short periods are not contra-indicated. Definite motor quiet and sensory quiet as well is desirable. Counter-irritation in those cases which show sharp myelitic symptoms is to be tried. Diathermy has seemed to help some cases.

Pharmacotherapy has not yet devised any specific remedies. Arsenic is used on empirical grounds and because of the possible relation of the toxin-producing agent to a spirochete (positive Wassermann in a few secondary cases; gold sol curve in many (Ayres), non-syphilitic) and in combination with quinine, ergot, strychnine and iron does some service. Germanin, a new synthetic, has been reported upon as of value. Protein therapy likewise and malarial therapy is being experimented with by numerous neuropsychiaters.[1] Salvarsan, silver salvarsan and other remedies have been tried. No positively good results are authenticated in the after history.

Great circumspection should be exercised in judging of the therapeutic results of any procedure, since it is so well known that most patients show remissions of shorter or longer periods and some even come to a standstill for as yet unknown reasons. In many of these unconscious psychical factors have as yet been almost entirely neglected.

Mild work in the open air, gardening, etc., is advisable. Psychotherapy is never to be forgotten, and a healthy optimism helps these patients enormously.

Some very anomalous and extraordinarily good results have followed the opening of the spinal canal (Elsberg).[2]

[1] Steiner, *loc. cit.* Dreyfus and Hanau: Ueber Fieber, in besondere Malariabehandlung bei der multiple Sklerose, Deutsch. med. Wchnschr., 1926, Nos. 9 and 10. In the Vienna Clinic Besredka's typhoid vaccine has been used. The Vienna experience is against malarial treatment especially in advanced cases. (Schilder).

[2] Surgery of the Spinal Cord, Saunders, 1916.

CHAPTER VII.

LESIONS AT THE LEVEL OF THE MEDULLA, PONS, BRAIN-STEM OR MIDBRAIN.

Lesions involving the medulla, pons, brain-stem or midbrain do not differ essentially in their kind from those already discussed as involving the spinal cord. Softening and hemorrhage, however, are more frequent from lesions of the vertebrals, basilar and circle of Willis's vessels and their branches. Anatomical knowledge of the vascular supply of this region is of much importance and unfortunately neglected. Stopford's recent studies should be consulted.[1] As has been pointed out, many of the syndromes, such as for instance those known as multiple sclerosis, as syringomyelia, polioencephalomyelitis, syphilis, may involve the entire cerebrospinal axis. This involvement may be more or less instantaneous. Such is the case in poliomyelitis, which in one and the same individual may within three or four days develop a meningitis (cerebral), a choreo-athetoid movement (striatum), an eye or other cranial nerve palsy (medulla), paralysis of some of the muscles of the arm (cervical spinal cord), atrophic changes in skin and bone of any extremity (sympathetic of spinal cord), and finally a palsy of one or more leg muscles (lumbar cord). On the other hand, in the case of a syringoencephalomyelia, a multiple sclerosis, or a cerebrospinal syphilis, the complete syndrome may need ten to twenty years to reach a complete development from brain to lumbar cord.

Furthermore, certain of these processes may limit themselves entirely to one group of structures. Thus a pontine, medullary, midbrain or fourth ventricle tumor, a midbrain arteriosclerosis, a medullary poliomyelitis, a quadrigeminal syphilis, or thalamic arteriosclerotic softening may limit the syndrome within narrower confines. In such an event the topical as well as the etiological factors become paramount.

As emphasized, a knowledge of the vascular supply is highly desirable. Stopford's results from injection experiments are here summarized and in a later page Greenacres' diagram is introduced.

According to Stopford:

1. The Anterior Spinal supplies:
 (*a*) In the cord:
 1. Anterior and base of posterior cornua.
 2. Gray matter around central canal.
 3. White matter of ventral columns and region of anterior roots.

[1] Stopford, J. B. S.: The Arteries of the Pons and Medulla, Jour. Anat., January-April, 1916; April, 1917. Dejerine's great classic, Semeiologie du Systeme Nerveux, Paris, 1926 (impression) should be consulted by the serious student; also Claude and Lévy-Valensi: Maladies du Cervelet et de l'Isthme de l'Encéphale, Paris, 1922. Lévy-Valensi: La Diagnostique Neurologique, Bailliére, Paris, 1925.

(*b*) In the medulla:

1. Pyramids and pyramidal decussation.
2. Medial lemniscus and tecto-spinal tract.
3. Dorsal longitudinal bundle.
4. Hypoglossal nucleus (except cephalically).
5. Nucleus and tractus solitarius (at decussation).
6. Gowers' tract (at decussation).
7. Dorsal nucleus of vagus (at calamus region).
8. Olivo-cerebellar fibers as they cross the middle line.
9. The internal and ventral external arcuate fibers with nucleus.

2. The Vertebral supplies:

1. Few pyramidal fibers at formation of lateral cerebro-spinal tract.
2. Pyramids at the lower border of pons, and a variable part of the lateral part below.
3. The whole of the ventral part, and the cephalic portion of the dorsal part, of the inferior olive.
4. The dorsal accessory olive.
5. Tegmento-olivary tract and olivo-cerebral fibers in formatio reticularis.
6. Generally the dorsal vago-glossopharyngeal nucleus above the calamus region.
7. Most cephalic part of hypoglossal nucleus.
8. Nucleus and tractus solitarius (at calamus).
9. Nucleus and tractus spinalis nervi trigemini (at decussation).

When it replaces the posterior-inferior cerebellar it also supplies: The spino-thalmic, rubro-spinal, and anterior and posterior spino-cerebellar tracts, nucleus ambiguus, descending root of the vestibular nerve, and nucleus and tractus spinalis of the trigeminal nerve, as well as part of the inferior cerebellar peduncle. In 8 per cent, owing to the high level of fusion to form the basilar, the vertebral supplies the abducens and facial nuclei as well as the other more medial structures in the lower part of the pons.

3. The Posterior-inferior Cerebellar supplies:

1. Spino-thalamic tract.
2. Rubro-spinal tract.
3. Olivo-cerebellar fibers as they passed to inferior cerebellar peduncle.
4. Dorsal vago-glossopharyngeal nucleus or the emerging fibers of the vagus and glossopharyngeal nerves.
5. Nucleus ambiguus.
6. Nucleus and tractus spinalis of trigeminal nerve.
7. Ventral part of inferior cerebellar peduncle.

4. The Posterior Spinal supplies:

1. Funiculi and nuclei gracilis and cuneatus.

2. Caudal and more dorsal part of the inferior cerebellar peduncle.
3. Occasionally part of the descending root of the vestibular nerve.

5. The Basilar supplies:
 (*a*) Medial set of pontine branches:
 1. Abducens nucleus and trochlear nucleus.
 2. Oculomotor nucleus (caudal part).
 3. Medial longitudinal bundle.
 4. Medial lemniscus.
 5. Tecto-spinal tract.
 6. Thalamo-olivary tract.
 7. Transverse pontine fibers.
 8. Medial part of corpus trapezoidum.
 (*b*) Transverse set of pontine branches:
 1. Brachium pontis.
 2. Lateral part of corpus trapezoidum.
 3. Superior olive.
 4. Facial nucleus.
 5. Remaining nuclei of eighth nerve.
 6. Remaining nuclei of fifth nerve (including motor nucleus).

When the origin of the basilar is abnormally low, (19 per cent. Stopford) it supplies the pyramids, medial lemniscus, tecto-spinal tract, dorsal longitudinal bundle, and most medial part of the hypoglossal nucleus, together with a variable part of the lateral portion of the upper medulla.

6. The Anterior-inferior Cerebellar supplies a variable part of the most caudal portion of the brachium pontis and upper and dorsolateral limits of the medulla.

7. The Superior Cerebellar supplies a variable part of the most cephalic portion of the brachium pontis.

The present chapter intends to deal with some of the more important problems chiefly with reference to localization. These localizing signs are extremely intricate and proper diagnosis can be arrived at only through a complete knowledge of the anatomy of these parts. Such an anatomical knowledge can be gained only by study of serial sections of these regions. By reason of the compactness of the structures the many complicated connections, and the as yet imperfectly analyzed dynamics of the mechanism within the pons, brain-stem and midbrain, the study of disturbance in these regions is particularly fascinating and fruitful. Here correlating and coördinating dynamic forces are represented at a maximum, so far as sensorimotor functions are concerned. While they are infinitely less complex than the adjusting symbolic dynamics handled by the cerebrum, yet they are sufficiently complex to make this field of sensorimotor neurology most bewildering.

The anatomical plotting of many of the fiber tracts and the chief synapses are known. They are given for the most part in various

chapters in this work and chiefly figured in Plates IX, XII, XIII, and in the group of figures given in the pages immediately following. All of the clinical pictures resulting from lesions in these regions cannot possibly be given in a work of this kind, hence only those most frequently met with in practice are taken up, while a tabular summary of these and a number of others is given at the end of this chapter.

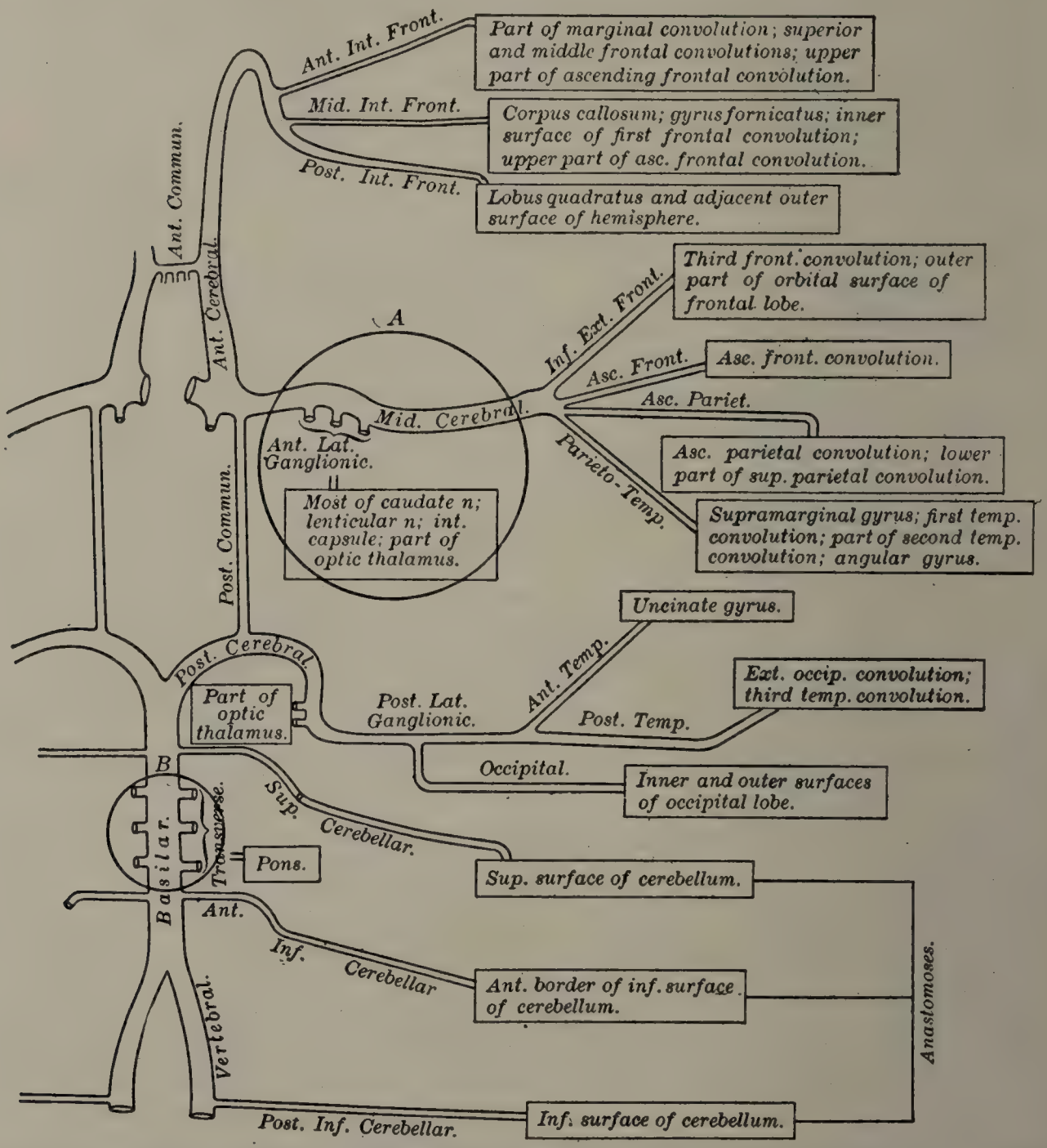


FIG. 310.—Circle of Willis and branches, with indication of distributions. Regions *A*, *B* most frequent sites for hemorrhages. (Greenacre, from Fursac.)

Medullary Syndromes.—Lesions of the medulla, usually depend upon vascular disorders of the branches of the vertebrals and basilar and produce marked initial symptoms of an apoplectiform nature and definite residual signs depending upon their location. They also occur in encephalitis, poliomyelitis, influenza, syphilis, in multiple sclerosis and a number of rarer conditions.

Among the *initial* signs are the usually rapid and severe onset of coma from cutting off of the cerebral pathways (diaschisis). Not infrequently there are generalized tonic convulsive movements of the extremities and often of the facial musculature with the exception of

those muscles whose nuclei may be implicated. Flaccidity of the lower extremities from diaschisis of the pyramidal tracts with initial loss of joint reflexes which later are increased. Involvement of vegetative pathways may cause vasomotor, respiratory, hepato-pancreatic, renal, pupillary and gastro-intestinal symptoms, vomiting, diarrhea, etc. Profound sensory loss also may be present.

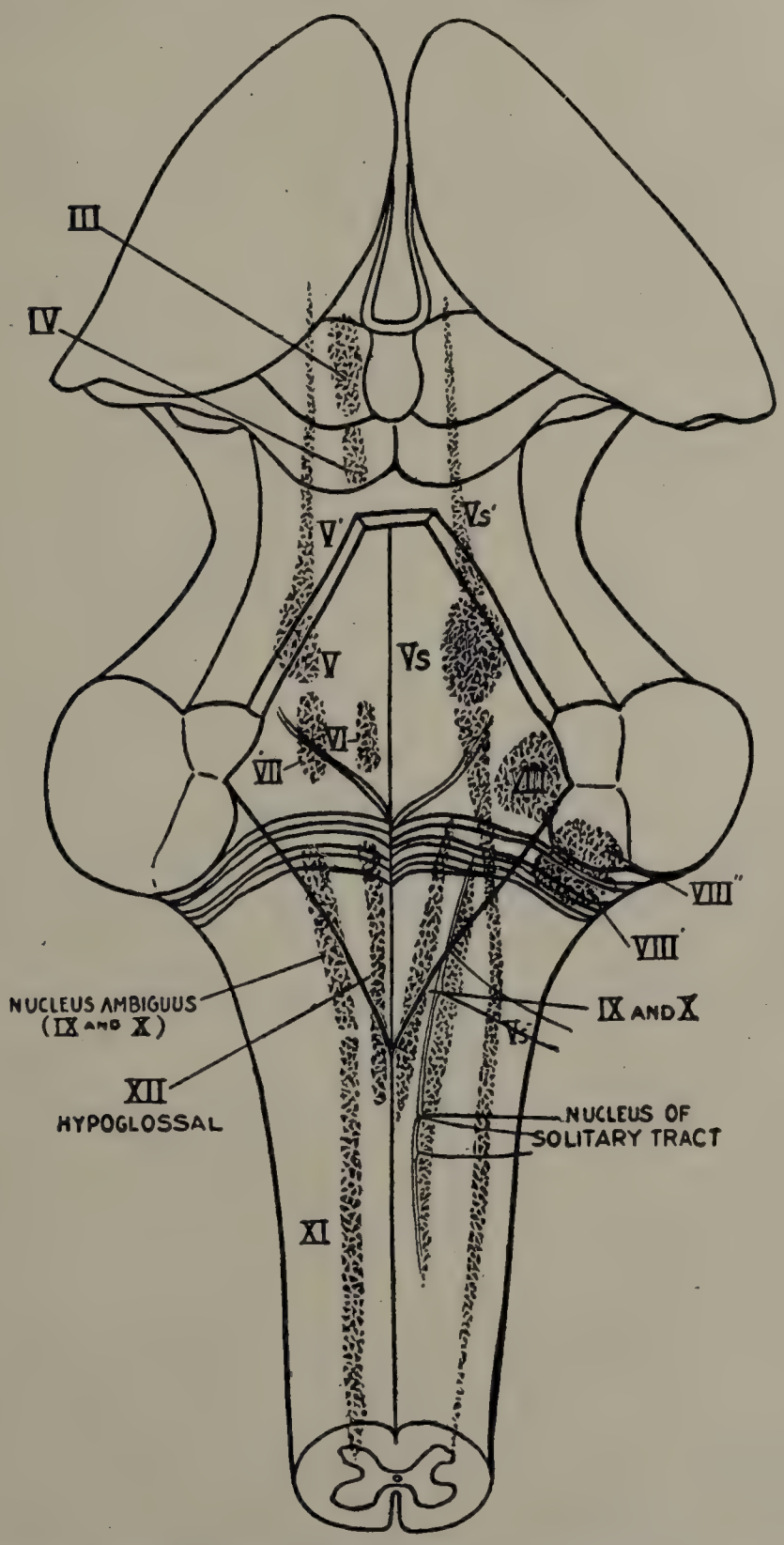


FIG. 311.—The cranial nerve nuclei schematically represented in schematically transparent brain-stem, dorsal view.

The *residual* symptoms will vary greatly according to the area destroyed. If recovery takes place, which is comparatively rare in lesions at the level of the pyramidal decussation, at least three separate clinical pictures are recognizable. If the lesion occupies the lower side of the medulla implicating the pyramidal tracts one observes a more or less continuous spastic paralysis of all four extremities usually

more marked on the side opposite the lesion; if it impinges further forward, involving the nucleus of the spinal accessory, there is added to the spastic diplegia a flaccid palsy of the muscles of the accessorius. Such clinical pictures result from arterial lesions, chiefly of the anterior spinal just below the juncture of the vertebral arteries to form the basilar.

A lesion at the side of the medulla here (meningitis, tumor, gumma, tuberculosis) involving the tractus cerebello-spinalis would cause a cerebellar ataxia.

Lesions slightly higher up in the medulla, at the level of the olive, for example and involving the lemniscus crossing fibers produce a dissociated disturbance of sensation of the entire half of the body omitting the trigeminus regions. If the lesion is below and impinging on the pyramidal region (Fig. 312) there is a spastic paresis of the opposite half of the body with atrophic paralysis of the same side of the tongue, which shows fibrillary twitching, deviates to the non-paralyzed side and gives reaction of degeneration.

Following lesions of the anterior spinal artery the lesion may implicate a long strip up and down the medulla. Thus in the patient (Fig. 312) inferior alternate hemiplegia from anterior pyramidal and interolivary lesions, the picture just described occurs, with addition of a hemianesthesia of the same side. There are no deep sensibility signs (hemianalgesia) nor hemithermo-anesthesia. Should the lesion cut off the circulation on both sides, as it not infrequently happens in anterior spinal artery occlusion from vertebral thrombi, the picture seen in Fig. 313 occurs. Here there is a double hemiplegia and hemianesthesia with alternate paralysis of the tongue, more marked on the left side. There is atrophy of the right half of the tongue with fibrillary twitching and reaction of degeneration.

GENERAL LEGEND ABBREVIATIONS OF MEDULLARY, PONTINE, PEDUNCULAR AND MIDBRAIN SYNDROMES. (Pp. 615–633.)

In all of these hemiplegia is indicated by oblique lines; hemianesthesiæ by dots and alternate paralyses by gray network. The lesions involve the cerebral peduncles and the pons at different levels.

ABBREVIATIONS. *Aq.*, aqueduct of Sylvius; *BrQp*, peduncle of posterior corpus quadrigeminum; *Crst*, corpus restiforme and inferior cerebellar peduncle; *Fcc*, central tegmental tract; *Fcs*, internal semicircular fibers of the cerebellum; *Flp*, posterior longitudinal fasciculus; *FPoa*, *FPop* anterior and posterior pontine fibers; *HC*, cerebellar hemisphere; *Lc*, locus ceruleus; *LN*, locus niger; *Lig*, lingula of superior vermis of cerebellum; *ND*, Deiters' nucleus; *Np*, nuclei of pons; *NR*, red nucleus; *NRl*, nucleus of lateral lemniscus; *Nrt*, reticular nucleus of the tegmentum; *NIII*, nuclei of third nerve; *NmV*, motor fifth nucleus; *NsV*, sensory fifth nucleus; *NVI*, nucleus of sixth nerve; *NVII*, nucleus of the facial nerve; *NVIII*, anterior nucleus of the acoustic (cochlear); *Oc*, cerebellar olive; *Os*, superior or pontine olive; *P*, lower stage of cerebral peduncle; *Pcm*, middle cerebellar peduncle; *Pcs*, superior cerebellar peduncle; *Py*, pyramidal tract in its pedunculopontine region; *Qa*, corpus quadrigeminum anterior; *r*, raphe; *Rm*, median lemniscus; *Rl*, lateral lemniscus; *SAq*, gray substance of the aqueduct of Sylvius; *SgR*, substance of Rolando; *Tpo*, tenia pontis; *Tr*, trapezoid body; V_4, fourth ventricle; *Vs*, superior vermis of the cerebellum; *VV*, valve of Vieussens; *III*, root fibers of the third nerve; *V*, trigeminus; *Vc*, descending motor root of the fifth; *Vds*, descending sensory root of the fifth; *VI*, root fibers of the sixth; *VII*, *rVII*, root fibers of the facial: *VIIg*, knee of the facial; *VIIIv*, vestibular.

Lesions of the vertebral (usually syphilitic thromboses) which involve the anterior spinal artery, and the posterior cerebellar arteries produce an involvement of most of one-half of the medulla (Fig. 314). This causes the so-called *Syndrome of Avellis*. The patient shows the usual apoplectic initial symptoms. The residuals show a hemiplegia and hemianesthesia on the side opposite to the lesion and a palsy of the



FIG. 312.—Antero-internal bulbar syndrome. Alternate hemiplegia of the hypoglossus by thrombosis of the left anterior spinal artery. The lesion involves the anterior pyramid, the reticular formation of the bulb, the root fibers of the hypoglossus, but not the *XII* nucleus

On the *right side* there is hemiplegia of the extremities and hemianesthesia especially of deep sensibility, without hemianalgesia or hemithermo-anesthesia.

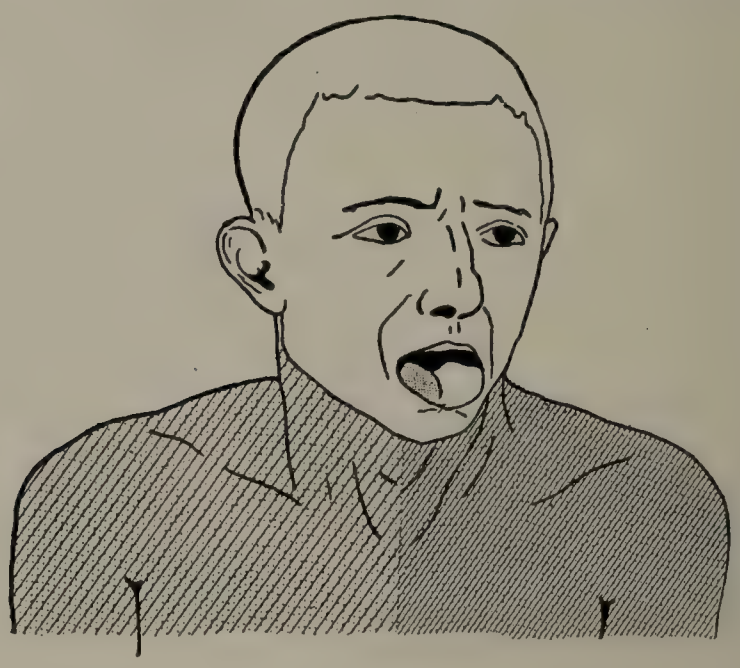
On the *left side* there is hemiatrophy of the tongue with R. D. (Dejerine.)

tongue on the side of the lesion. There is a trigeminal anesthesia of the involved side. Oculopupillary signs are present, narrowing of the palpebral fissure on the side of the lesion. There is also, from implication of the cerebellar pathways, a cerebellar asynergia. There is also a paralysis of the soft palate and vocal cord of the same side as the lesion.[1]

[1] Spiller: Jour. Nerv. and Ment. Dis., June, 1919.

This clinical picture may be encountered also in syringomyelia, multiple sclerosis or tabes where it is of gradual development, or in encephalitis when it develops suddenly.

Partial clinical pictures of the syndrome of Avellis have been termed the syndromes of Schmidt and of Jackson. In the *Syndrome of Schmidt* there is a unilateral paralysis of the soft palate, of the vocal



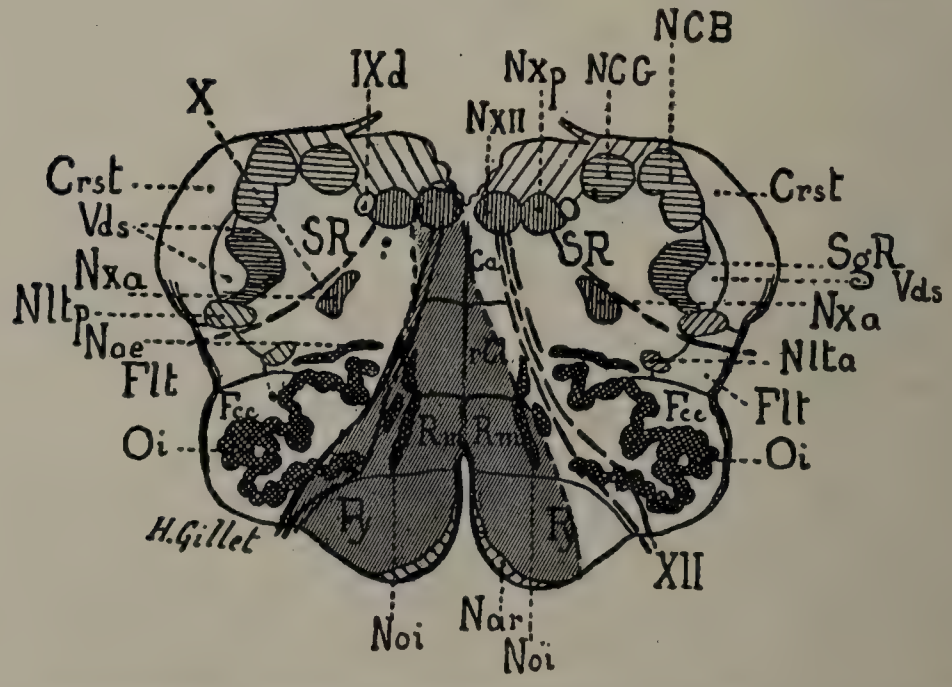


FIG. 313.—Antero-internal bulbar syndrome from occlusion of the anterior spin a artery from the right vertebra. There results a double hemiplegia with alternate paralysis of the hypoglossus. The lesion more marked on the right side involves both pyramidal tracts (*Py*) in the bulb, the interolivary paths (*Rm*) and the formatio reticularis (*rCl, Ca*). On the right side the lesion cuts off the root fibers of the hypoglossus. There is hemiplegia and hemianesthesia of all four limbs, more marked in the *left side* The sense of position is specially involved. On the *right side* there is hemilingual atrophy with R. D. (Dejerine.)

cord, of the sternocleidomastoid and of the trapezius. The spinal vagus nucleus and the spinal accessory nucleus or radicular fibers are involved.

In the *syndrome of Jackson* there is a hemiparesis of the soft palate, the vocal cord, the sternocleidomastoid, with hemiparesis of the tongue with atrophy.

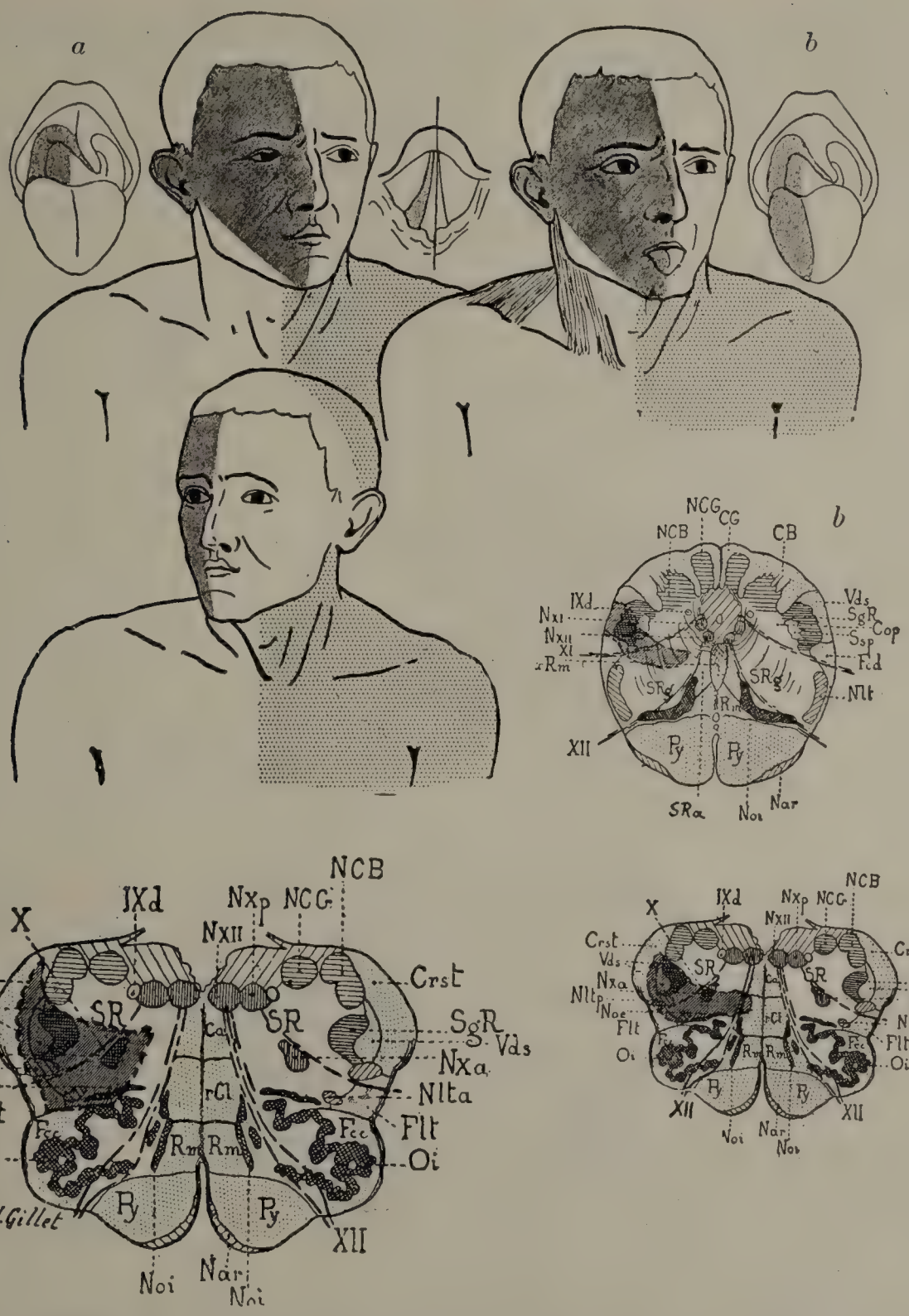


FIG. 314.—Retro-olivary bulbar syndromes. Hemianesthesia of syringomyelic type with alternate paralysis of the descending root of the trigeminus and of the spinal vagus (syndrome of Avellis) and hemiparesis of the pharyngo-laryngo-velo-palatine with (*a*) or without (*b*) oculopupillary vegetative signs; without (*a*) or with (*b*) concomitant alternate paralysis of the *XII*, and of the lower portions of the *XI* pairs (syndrome of Jackson). There is also lateropulsion, cerebellar hemiataxia and hemiasynergia from lesion of the bulboretro-olivary tegmentum of the right side supplied by the inferior and posterior cerebellar artery. There is no crossed hemiplegia of the limbs nor tactile nor deep hemianesthesia because of the integrity of the pyramids and of the reticular formation, and particularly of the interolivary paths.

On the *left side* there is analgesia and thermo-anesthesia of the extremities, of the neck and the back of the head from lesion of the crossed secondary sensory paths of the retro-olivary reticular formation.

On the *right* there is hemiataxia and hemiasynergia, more marked in the lower extremity with lateropulsion from extension of the lesion to the restiform body; pharyngo-laryngo-velo-palatine palsy from lesion of the spinal vagus; anesthesia of the face from lesion of the descending root of the trigeminus.

In *a* there are vegetative oculopupillary signs from involvement of the sympathetic fibers in the lateral reticular formation and trigeminus. In *b* the sympathetic fibers are not involved but the *XII* fibers are implicated, hence the paralysis and atrophy of the tongue; extending lower down in the medulla the inferior spinal accessory fibers being caught in the lesion there is paralysis of the external branch supplying the trapezius and sternocleidomastoid. (Dejerine.)

These bulbar palsies are observed in vascular lesions, in tabes, progressive bulbar palsy, multiple sclerosis, neurosyphilis, syringomyelia and occasionally in encephalitis and poliomyelitis.

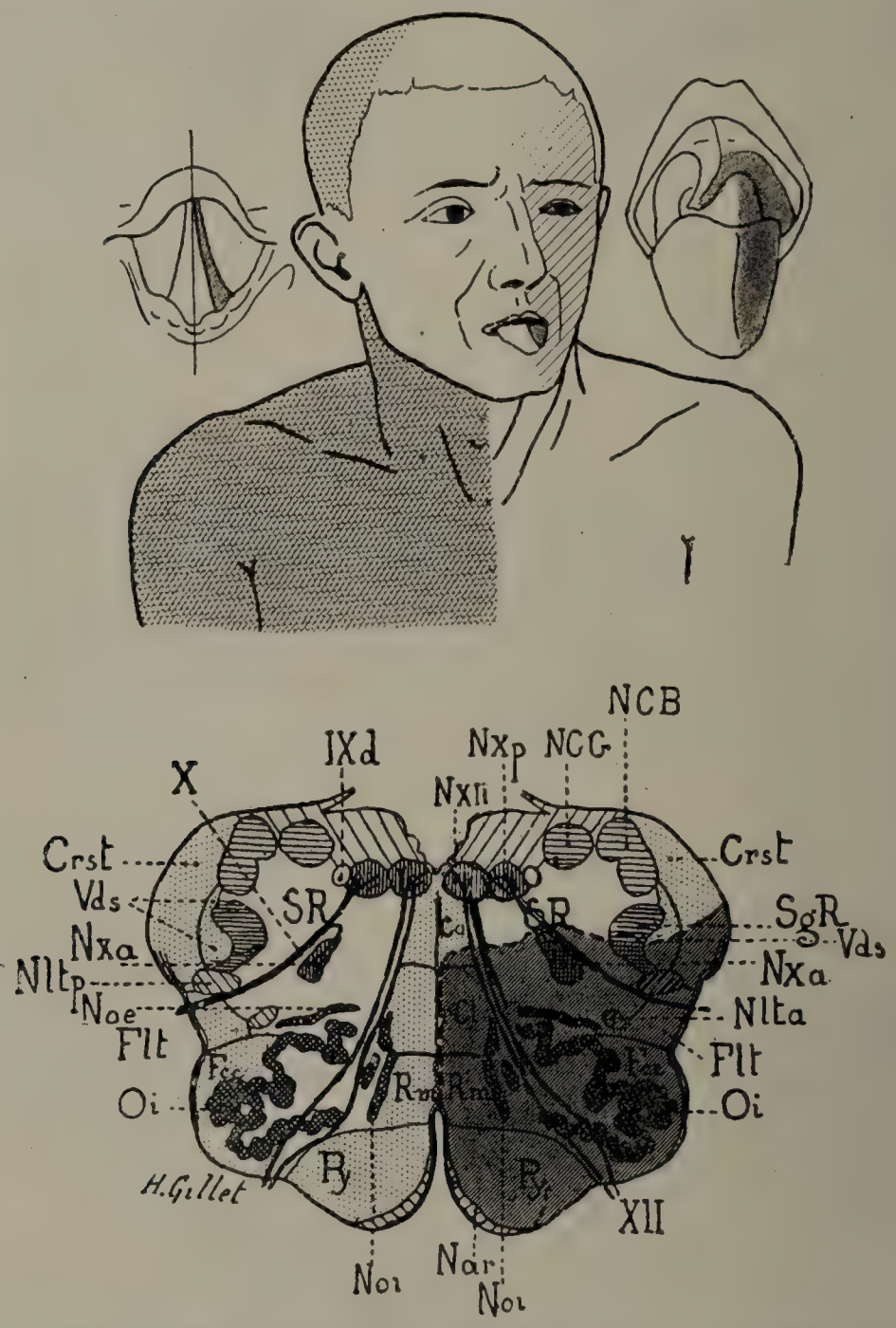


FIG. 315.—Antero-internal retro-olivary bulbar syndrome. Crossed hemiplegia and hemianesthesia with alternate paralysis of the hypoglossus, the descending fifth (syndrome of Avellis), oculopupillary vegetative signs, cerebellar hemiasynergia from thrombosis of the left vertebral before the giving off of the anterior spinal and inferior and posterior cerebellar arteries.

On the *right* side there is crossed hemiplegia, pontine-bulbar type, of the trunk and the extremities from pyramidal lesion; hemianesthesia, for all forms of sensibility, especially for pain and heat, of the trunk, neck, extremities and back of the head from lesion of the reticular formation (*Rm*, *rCl*) white and (*SR*) gray.

On the *left* there is cerebellar hemiasynergia with lateropulsion and hemiataxia. There is hemilingual atrophy from lesion of the root fibers of the hypoglossus, pharyngo-laryngo-velo-palatine palsy (syndrome of Avellis), with dysphagia, dysphonia, roughening and loss of voice from lesion of the root fibers and anterior nucleus of the spinal vagus (*NXa*), myosis and narrowing of the palpebral fissure from sympathetic oculopupillary fibers in the lateral reticular formation (*SR*); hemianesthesia of the face from descending fifth lesions. (Dejerine.)

A related syndrome due to lesions in this general region is that of Babinski and Nageotte.[1] These patients show acute or subacute

[1] Nouv. Iconog. de la Salpêtrière, 1902, **15**, 192.

involvement. If not comatose there may be dizziness. The residual picture is that of a crossed hemiparesis, hemianesthesia with deep sensory losses, and difficulty in swallowing (Fig. 301). These patients have to be supported when they walk with legs widespread. There is marked cerebellar asynergia with lateropulsion to the side of the lesion. The abdominal reflexes are not apt to be involved. The other reflexes follow the usual hemiplegic type. Irregularity of the pupils, myosis of affected side, enophthalmos, syringomyelic dissociation at times occur.

Cestan and Chenais have described a variant which is a combination of this syndrome with the palatovocal palsies of the syndrome of Avellis.

Lesions of the Pons.—Softenings, hemorrhages,[1] tumors, gummata, multiple sclerosis, syringomyelia, poliomyelitis, encephalitis, tabes produce isolated or complicating lesions of the pons, which varying with their location and size, give rise to a bewildering array of clinical pictures which merge one into another and almost defy analysis. A careful study of the motor nuclei and of the motor and sensory tracts will enable the student to differentiate these pontine syndromes, which when due to lesions implicating the seventh nerve nuclei or structures below or of its branches cause the various typical inferior alternate paralyses.

The most classical of these syndromes are those of Millard-Gubler, Foville and Gubler-Weber combinations.

Reference to Plate I (page 52) shows that the pontine fibers commence to cross about the upper level of the tenth and twelfth nerve nuclei. The syndromes just described—Schmidt, Avellis, Jackson, Bonnier, Babinski and Nageotte Cestan-Chenais, result from lesions just below this general area. The course of the cortico-nuclear fibers is extremely complex throughout the whole pontine levels and can best be traced from Plate IX, page 368, and from the series of cross-sections of the various syndromes of this chapter.

The sensory pathways are likewise extremely complicated.

So long as the sensory paths were in their spinal route they were capable of a certain amount of isolation either as they entered the cord and made their first synapses, or as they continued up the cord in primary or secondary paths. But as these paths converge to enter the brain-stem they become anatomically closer, disease processes are apt to overrun many paths, and thus the analysis becomes increasingly difficult up to the entering of these paths into the optic thalamus.

The most searching analyses of Head, Holmes, May, Rothmann and others and their researches tend to show that the impulses underlying sensations of pain, heat and cold seem alone to run unaltered, either directly or by intercalated fibers associated with the ganglion cells of the formatio reticularis, between the upper end of the spinal cord and the optic thalamus. Here are received the regrouped secondary

[1] Stopford, J. S. B.: Arteries of Pons and Medulla, Jour. Anat., January, 1916; April, 1917.

impulses from the face which cross, then join the specific paths for pain, for heat or for cold. These paths are so situated that they can be interrupted without disturbance of any other form of sensation on the

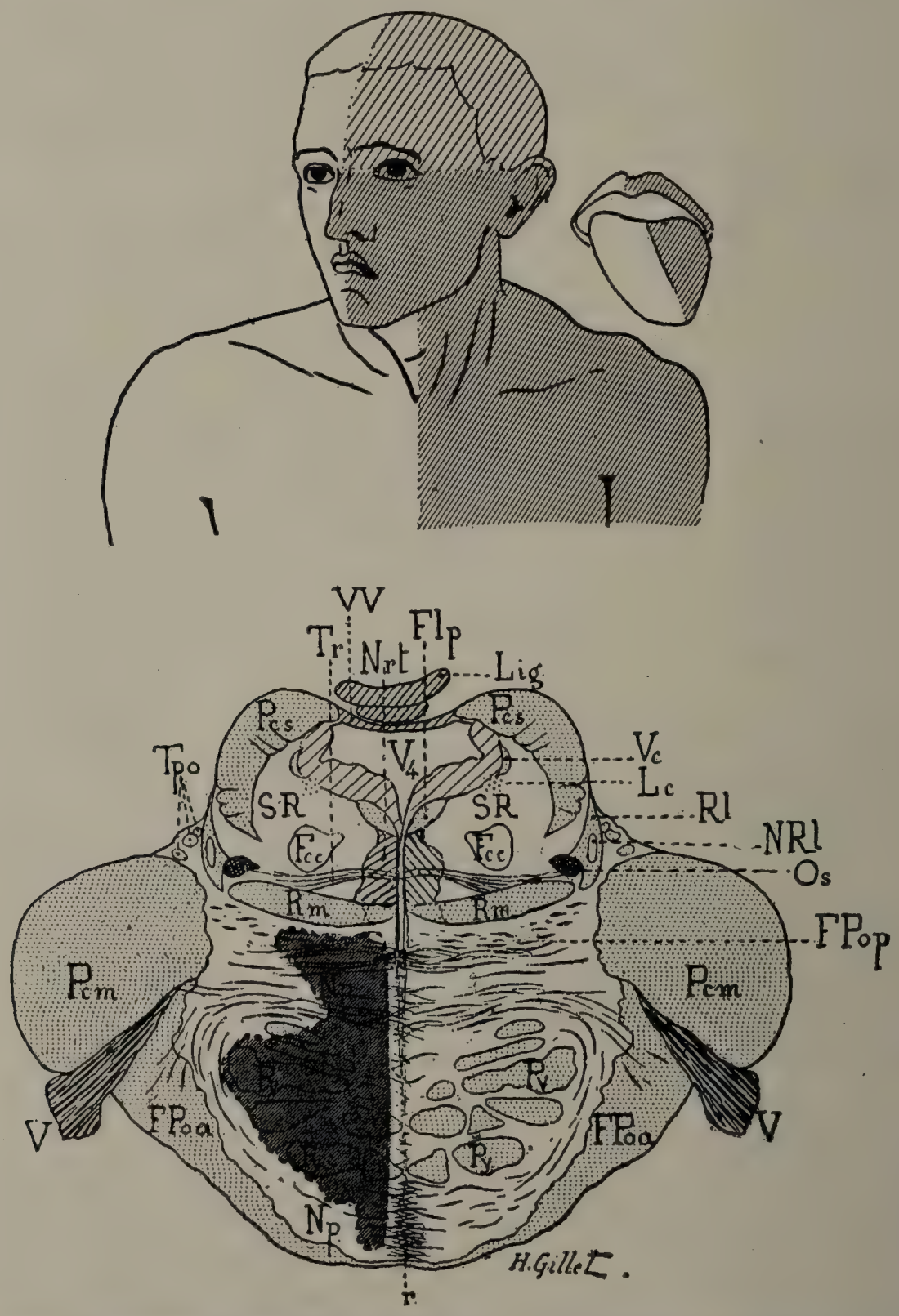


FIG. 316.—Anterior cephalic pontine syndrome. Hemiplegia of cerebral type due to thrombosis of the upper part of the basilar trunk. The lesion is unilateral, occupying the cephalad part of the pons, or the anterior portion of the right pons, destroying there the corticospinal pyramidal fibers, the corticonuclear fibers of the facial, the masticators, and the hypoglossal. It does not involve the tegmentum nor the root fibers of the cranial nerves.

On the left side there is a crossed hemiplegia of the trunk and extremities with contractures and exaggeration of the reflexes. Left inferior facial hemiplegia, mild superior hemifacial paresis, slight widening of the palpebral fissure, slight drooping of the external border of the left eyebrow, slight impairment of independent closure of the eyes; hemiparesis of the masticators, especially of the internal pterygoid. Hemiparesis of the tongue. Integrity of electrical excitability.

By reason of the predominance of the muscles on the sound side the lips and jaw are drawn to the right and on protrusion of the tongue it points to the paralyzed side, due to action of the right genioglossus. (Dejerine.)



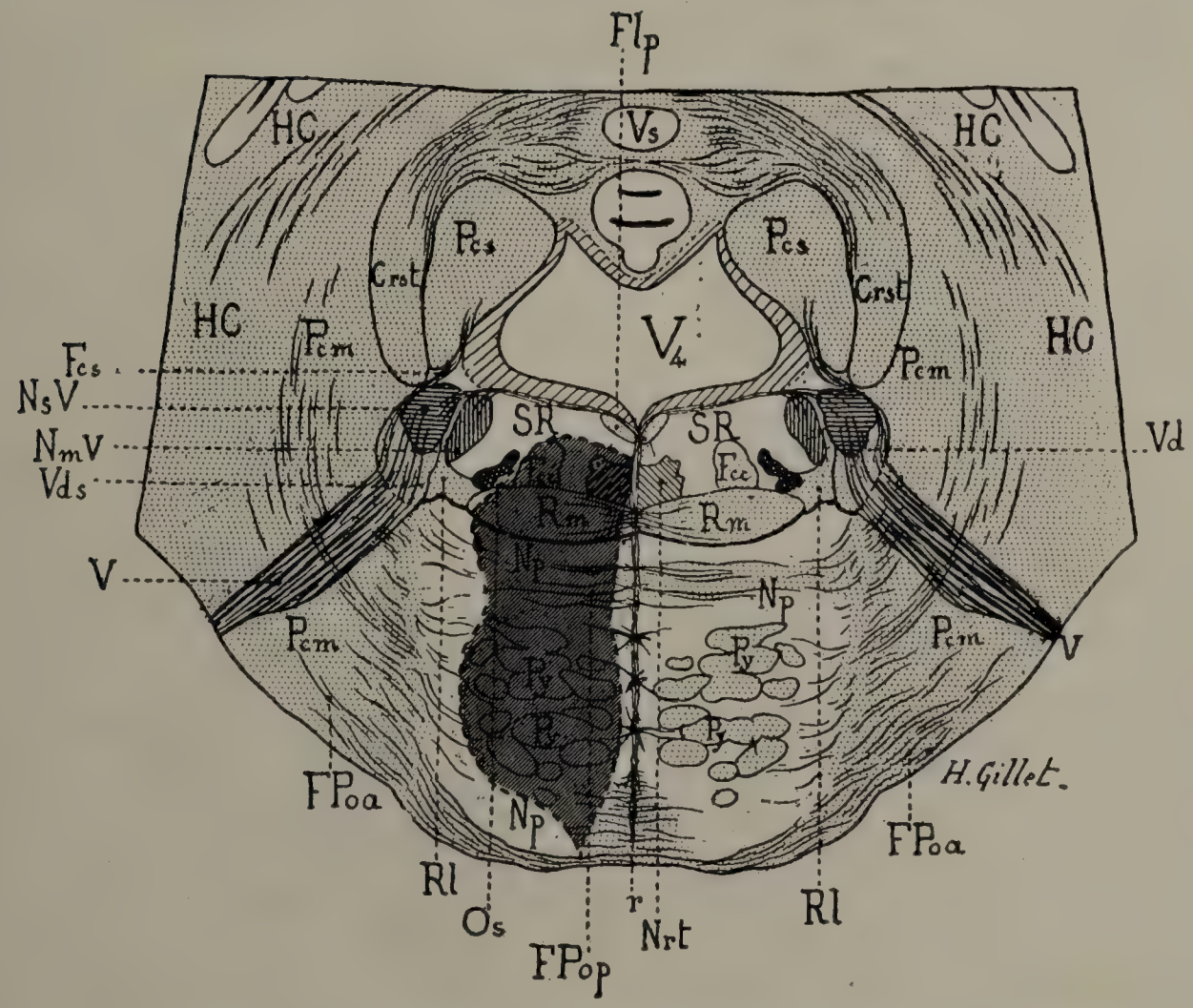


FIG. 317.—Anterior and posterior internal pontine syndrome. Thrombosis of the middle portion of the basilar artery involving the right anterior, mesial basotegmental portion; the lesion not going as far as the motor and sensory nuclei of the trigeminus and avoiding the posterior longitudinal fasciculus. There is contralateral hemiplegia, on the left side, of the cerebral type, of the trunk and extremities and of the inferior facial by reason of the lesion of the corticospinal pyramidal and facial fibers (*Py*); mild hemiparesis of the upper facial, hemilingual paresis and hemiparesis of the masticators (deviation of the chin to the sound side by implication of the aberrant pyramidal corticolingual, and corticotrigeminal (motor) fibers which travel in the median lemniscus (*Rm*) and the tegmentum (*Sr*)). (See Plate I.)

There is contralateral hemianesthesia for all kinds of sensibility but above all of the sense of position by lesion of the median lemniscus (*Rm*) and of the reticular formation of the tegmentum (*SR*). There is no alternate paralysis, the lesion not involving any of the sensory or motor cranial nerve nuclei. (Dejerine.)

body, and the analgesia and thermo-anesthesia so produced resemble in quality the loss of sensation to pain, heat and cold caused by a lesion in the spinal cord. (See Plates XII and XIII.)

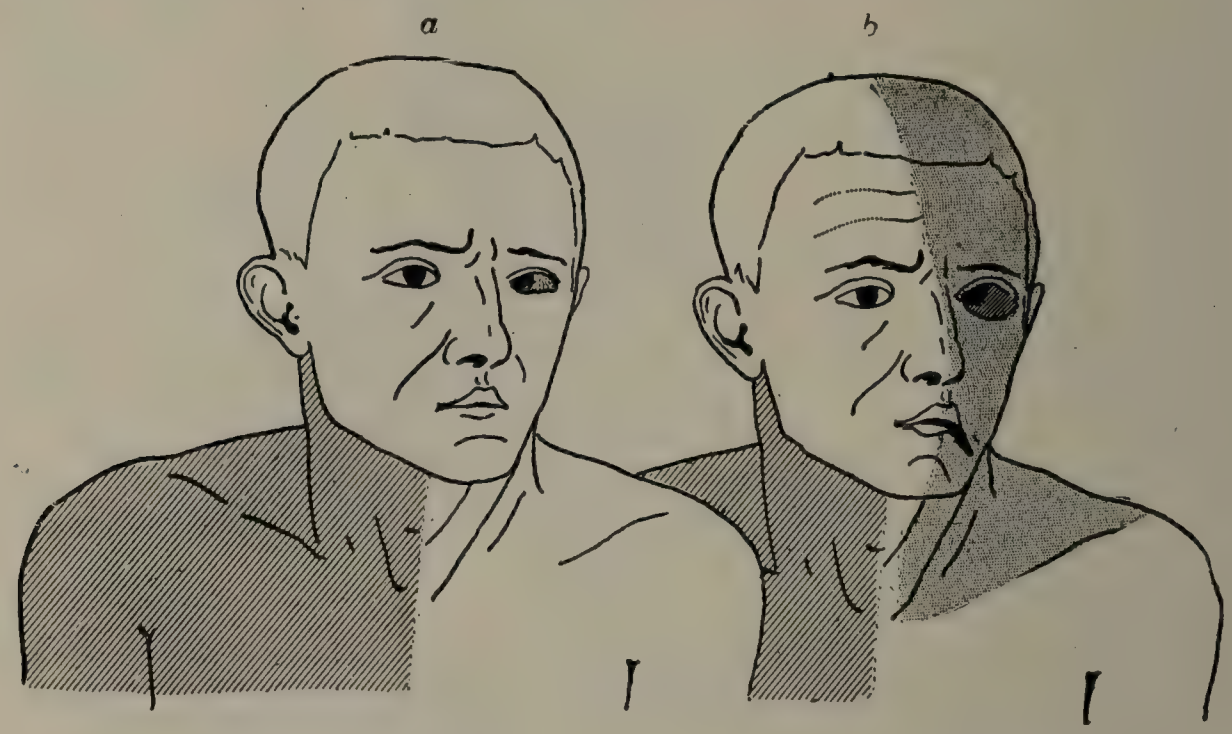


Fig. 318.—Anterior pontine syndrome. Inferior alternate paralysis — Millard-Gubler type. Contralateral hemiplegia of the trunk and of the extremities of the ponto-medullary type (without inferior facial palsy) without hemianesthesia, and without the syndromes of Foville, with alternate paralysis may be of the sixth nerve alone (*a*) or of the sixth and seventh (*a* and *b*). At this level the cortico-nucleo-facial fibers have left the pyramidal tract with the medullopontine aberrant fibers (see Plate I) and are in the tegmentum, hence the absence of the inferior contralateral facial palsy.

At the *right* there is hemiplegia of the extremities without paralysis of the inferior facial.

To the *left* there is convergent internal strabismus by paralysis of the external rectus; the predominance of the antagonists carries the eye to the left and up. There is no forced position of the head, the associated lateral movements of the eyes being possible. only the left eye is involved.

In (*b*) there is added to the preceding an atrophic facial paralysis with *RD* (*VII*), The facial folds are gone, there is lagophthalmus, drooping of the angle of the mouth and paralysis of the elevators. (Dejerine.)

Thus when a lesion in the medulla interferes with pain pathways, not only may the skin be insensitive to prick, but the readings of the pressure algometer may be raised on the analgesic side. In the same way the affected area of the body may be insensitive to all degrees of heat, and to all stimuli capable of evoking normally a sensation of cold. In the medulla, however, in distinction from lesions of the cord, the grosser forms of pain and discomfort may traverse other paths if the usual ones are closed, whereas in the cord all painful impulses are blocked by a unifocal lesion.

At the medulla and pontine levels, moreover, all three forms of sensibility may be affected together or any one may escape or be alone involved.

These impulses of pain, heat, and cold all run up in the neighborhood of the fifth nerve nucleus, and in cases of occlusion of the postero-inferior cerebellar artery and from other destroying lesions, the paths are usually implicated. This same accident may occasion a dissociation of the impulses underlying the appreciation of posture and passive movement from those concerned with spatial discrimination.

A summary of the findings which may occur from the lesions which cut off the sensory pathways between the nuclei of the posterior columns and the optic thalamus has been stated by Head and Holmes as follows:

1. The impulses for pain, heat, and cold continue to run up in separate secondary paths on the opposite side of the nervous system to that by which they entered. They receive accessions from the regrouped afferent impulses from the nerves of the head and upper part of the neck.

Although these paths are frequently affected together they are independent of one another, and any of the three qualities of sensation may be dissociated from the others by disease.

2. Lesions of the spinal cord tend to diminish simultaneously all forms of painful sensibility, but with disease of the brain-stem the gross forms of pain and discomfort may pass to consciousness, although the skin is analgesic. This applies not only to painful pressure, but to the discomfort produced by excessive heat.

3. The impulses concerned with postural recognition part company with those for spatial discrimination at the posterior column nuclei. Up to this point they have travelled together in the same column of the spinal cord, but as soon as they reach their first synaptic junction they separate. Above the point where they enter secondary paths the power of recognizing posture and passive movements can be effected independently of the discrimination of two points and the appreciation of size, shape and form in three dimensions.

4. It would seem as if those elements which underlie the power of localizing the spot touched or pricked become separated off from their associated tactile impulses before they have actually come to an end in the optic thalamus. The long connection of localization with the integrity of tactile sensibility is here broken for the first time.

All these changes are preparatory to the great regrouping which takes place in the optic thalamus.

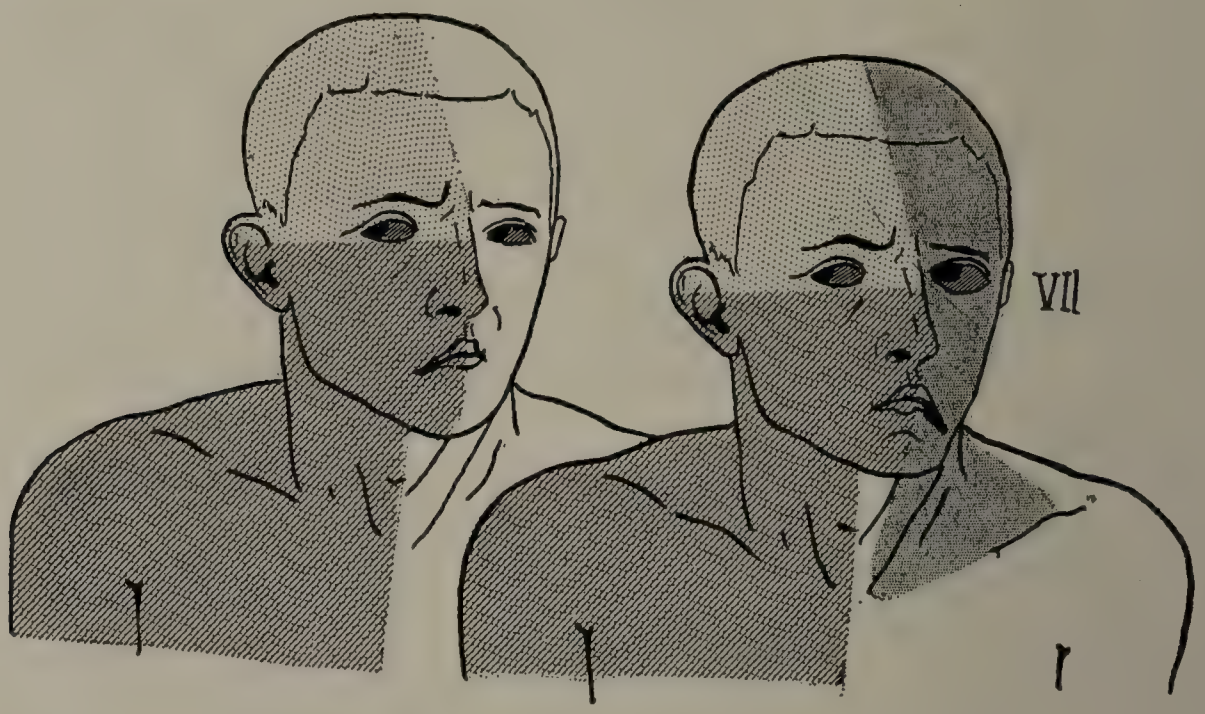


FIG. 319.—Pontine syndrome. Millard-Gubler-Foville syndrome. Hemiplegia of cerebral type with hemianesthesia of the extremities and of the head of the side opposite and alternate paralysis may be of the (*VI*) only (*a*) or of the *VI* and *VII*, (*b*) by thrombosis of the inferior portion of the basilar artery, in particular of the median pontine arteries of the left side. The lesion occupies the anterior part of the pons, destroys the pyramidal tract (*Py*) extends to the tegmentum, sections the median lemniscus fibers (*Rm*), the fibers of the reticular formation, the posterior longitudinal fasciculus (*Flp*), the root fibers of the *VI* pair. In (*b*) the more extensive lesion destroys in addition the root fibers of the facial and the nuclei of the *VI* and *VII* and the fibers of the lateral portion of the reticular formation.

On the *right* there is contralateral hemiplegia of the cerebral type—extremities and inferior facial, by lesion of the corticospinal pyramidal fibers and of the cortico-nucleo-facial (aberrant-medullo-pontine fibers). Hemianesthesia of the extremities and of the face in (*a*) involving particularly the tactile sensibility and postural sense, and in (*b*) all modes of sensibility, these being more marked in the head than in the extremities.

On the *left* side there is paralysis of the external rectus (*VI*) internal strabismus, to which there is added in (*b*) a paralysis of the facial (*VII*) inferior, superior, and of the platysma myoides, lagophthalmia, drooping of the angle of the mouth, flattening of the facial lines.

Furthermore, there is paralysis of lateral movements of the eyes toward the left by lesion of the left posterior longitudinal fasciculus (*Flp*). By reason of the predominance of the antagonists the patient looks toward the right. (Dejerine.)

Inferior Alternate Paralysis.—Anterior and Posterior Pontine Syndromes. Millard-Gubler, Foville Syndromes.

Reference to Plate I and Fig. 319, p. 624, shows the site of a lesion which produces the Millard-Gubler[1] type of inferior alternate paralysis. The lesion, a softening, hemorrhage, tabes, isolated tubercle, syringomyelia, bulbar palsy, gumma, polioencephalomyelitis, rarely recurrent palsy in migrainous vascular disturbance, in the anterior part of the pons low down at the emergence of the VI nerve and internal to the peripheral exit of the VII nerve, causes a partial paralysis of the limbs and trunk on the opposite side of the body to the lesion and an internal strabismus of the eye of the opposite side. There is (*a*) no facial palsy and no sensory loss—hemianesthesia—the median lemniscus



FIG. 320.—Hemorrhage of pons, causing posterior pontine syndrome. [Compare Fig. 322.]

(*Rm*) being spared in this lesion. If this lesion extends laterally (*b*) to include the peripheral VII there is a facial palsy on the side of the lesion. The sixth nerve palsy on the same side of the lesion causes an internal strabismus. The seventh nerve palsy is of the peripheral type (see *b*). There is reaction of degeneration, atrophy of the facial musculature, lagophthalmos from levator palsy, the angle of the mouth droops. There are no sensory disturbances.

A lesion lying internal to the peripheral emergence of the VI nerve can cause an ordinary hemiplegia indistinguishable from a cerebral palsy. It would have to be a very small lesion, however.

[1] Millard: Gaz. hebd. de méd. et chir., 1856, **3**, 816. Gubler: Mémoire sur l'hemiplegie alterne, Gaz. hebd. de méd et de chir., 1856–1858. Prevost: Thèse de Paris, 1868. See recent study by Gerber: Jour. Nerv. and Ment. Dis., 1927. Also consult Claude and Lévy-Valensi: Gilbert et Carnot's Traité de méd., 1922, vol. **32**. Tilney and Riley: Form and Function of the Nervous System, New York, 1923.

A still rarer delimitation by a lesion involving the VI and VII (between *a* and *b* peripherally in Fig. 318) at their convergence at this level will cause a facial palsy with an internal strabismus and no other symptoms. These are usually due to meningeal syphilitic lesions

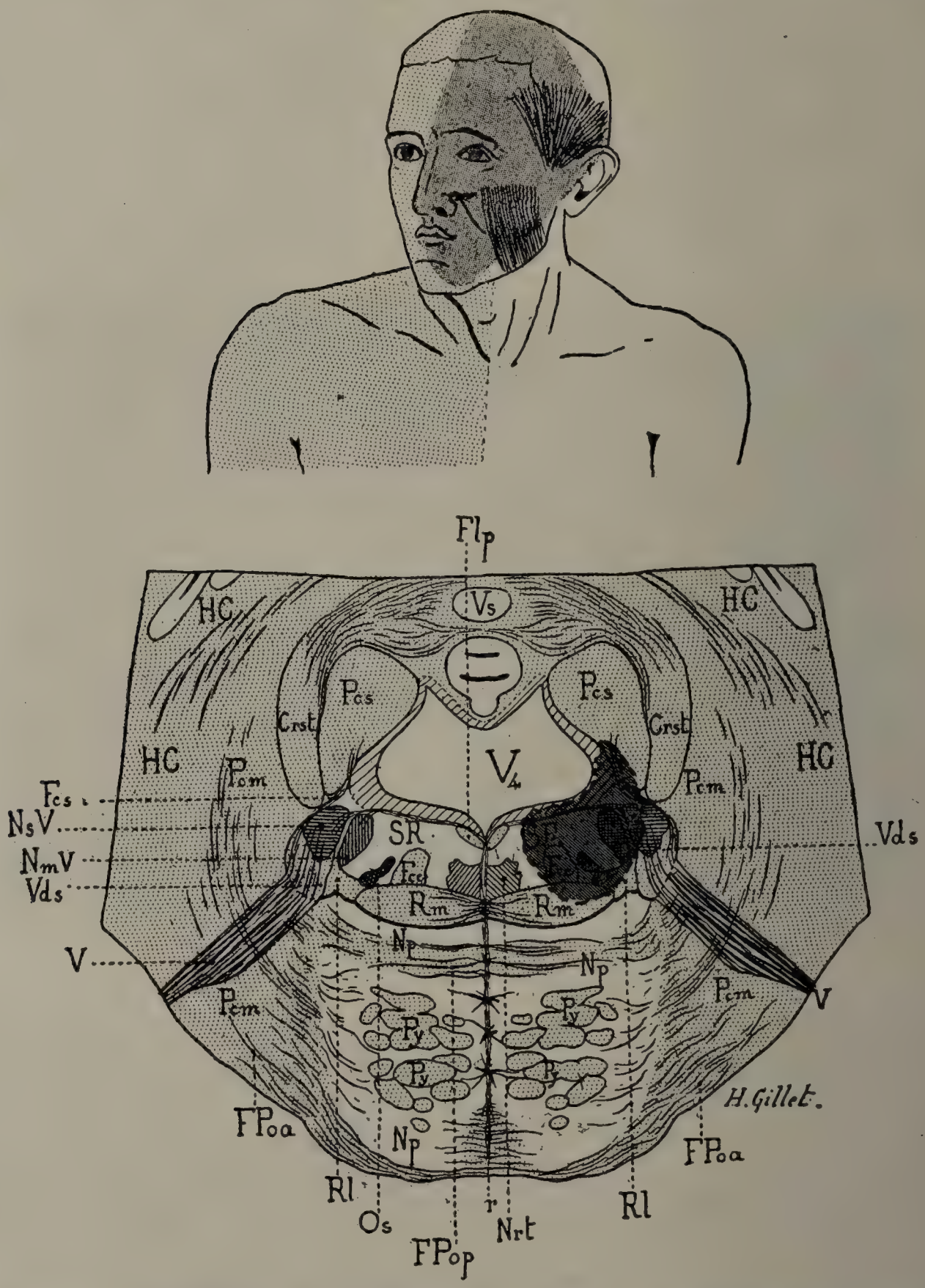


FIG. 321.—Posterolateral pontine syndrome. Lesion of the lateral part of the left pontine tegmentum, involving the nuclei of the trigeminus, the crossed secondary sensory paths of the tegmentum, partially involving the superior cerebellar peduncle (*Pcs*) and the median lemniscus (*Rm*), and not involving the anterior portion of the pons.

On the right there is hemianesthesia of the extremities of the syringomyelitic type, above all for pain and temperature sense.

On the left there is paralysis of the muscles of mastication (pterygoid, masseter, temporal) by lesion of the motor nucleus of the trigeminus. There is slight anesthesia in the trigeminus region (sensory nucleus *V*) and choreo-athetoid movements of the extremities from involvement of the superior cerebellar peduncle. (Dejerine.)

and several cases have come under observation. Two such patients have had two recurrences of this combination at a year's interval. There have been no migraine attacks in either. In one the Wassermann test was ++++; in another it was negative and a beginning multiple sclerosis was suspected.

Millard-Gubler-Foville Syndrome.—This syndrome was originally described by Foville.[1] The lesion which may be any of the types already cited for the Millard-Gubler syndrome, but is more apt to be vascular (thrombus of vertebrals, basilar involving the middle pontine vessels), occupies not only the anterior stage of the pons but lies along the central raphe in this region of the pons (Fig. 318). Thus the median lemniscus (fillet) (*Rm*) fibers are involved in addition to the pyramidal fibers, the fibers of the reticular formation, and even the posterior longitudinal fasciculus (*Flp*). Thus to the crossed hemiplegia there is added a crossed hemianesthesia and a paralysis of the associated eye muscles. The internal rectus of one side (III pair) and the external rectus of the opposite side (VI) cannot functionate synergistically. This oculogyric palsy may involve one or the other side according to the site of the lesion, the eyes being turned away from the site of the lesion (see Plate II). If the root fibers of the facial are involved (*b*), as they usually are, there is an inferior facial palsy of the same side. This type of paralysis is illustrated in Figs. *a* and *b* of Fig. 319. In (*b*) the facial palsy is figured. In (*a*) the lesion lies internal to the facial. The hemianesthesia involves deep sensibility of the body and face. Postural sense is also involved. The losses in sensibility are more marked in the face than in the extremities and there is no marked affective overresponse.

Posterior Pontine Syndromes.—Lesions at this same level (VI and VII nerve level—see Plate I) by lying in the tegmental or posterior part of the pons and behind the cortico-nuclear and cortico-spinal fibers produce a combination syndrome if the lesion lies internal to the corpus restiforme, a more extended syndrome when encroaching upon this structure and the VIII nerve.

In this more limited posterior pontine syndrome (usually softening or hemorrhage) there is a partial crossed hemianesthesia from minimal fillet (*Rm*) involvement, alternate paralysis of the VI and VII nerves, anesthesia of the trigeminus. The anesthesia is a syringomyelic dissociated type if only a part of the fillet is implicated. Tactile sensibility, sense of position, and stereognostic sense are intact (Fig. 321).

A more extensive lesion (tubercle, multiple sclerosis, gumma, softening (Fig. 322, p. 628) produces a much greater degree of crossed hemianesthesia by complete cutting off of fillet fibers (*Rm*). There is alternate VI and VII nerve palsy. There is conjugate palsy of the III and VI with turning of the eyes away from the side of the lesion, from

[1] Grasset (Rev. Neurol., 1900, **8**, 586) cites Foville (Bull. Soc. Anat., 1858, **33**, 393) as having given the first clear description of this syndrome. Consult Hassin: Pontine Hemorrhage and Foville's Syndrome, Jour. Nerv. and Ment. Dis., November, 1922. Gerber: Loc. cit. Claude and Lévy-Valensi: Loc. cit. Dejerine: Loc. cit.

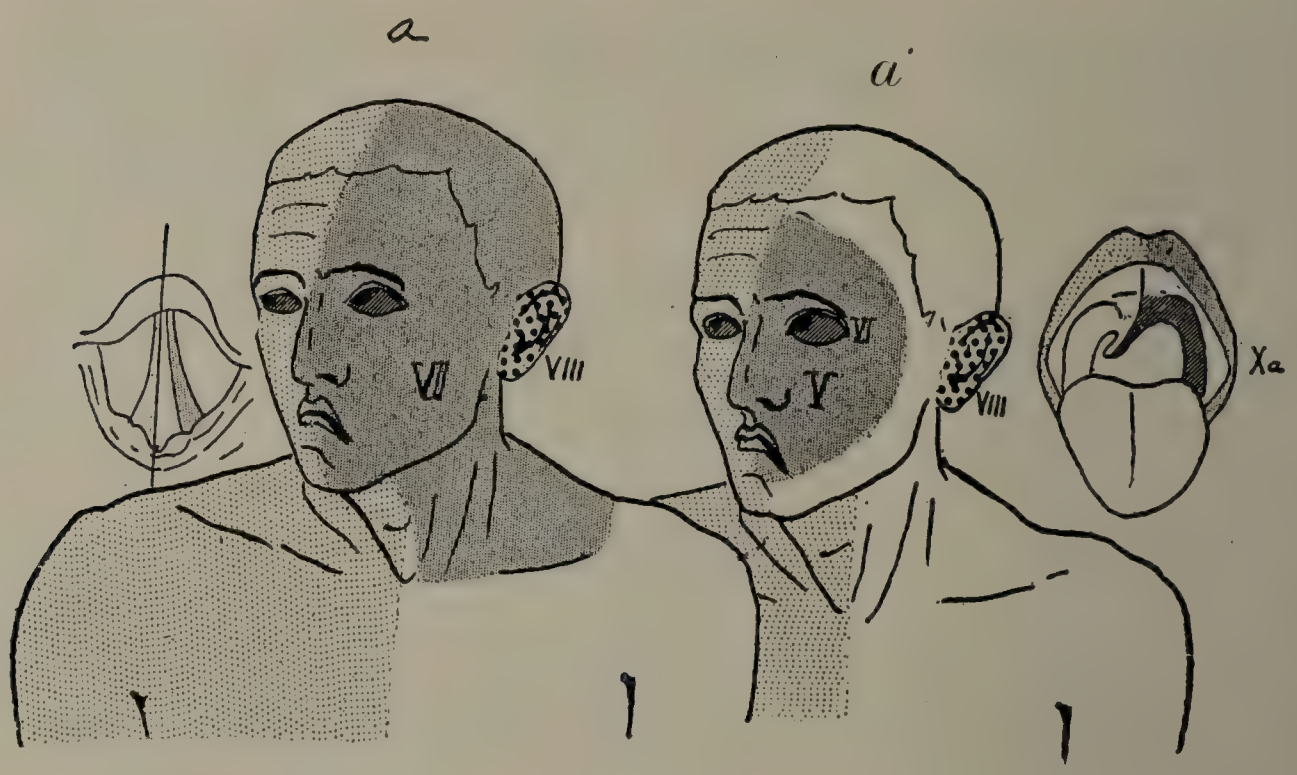


FIG. 322.—Posterior (caudal) pontine lesion. Contralateral anesthesia with alternate paralysis of the *VI*, *VII*, *VIII* pair and of the spinal vagus (*Xa*) from a tubercle in the inferior (caudal) portion of the left pontine tegmentum. The tubercle has pressed upon or destroyed the secondary crossed paths of the tegmentum (*SR*), the median lemniscus (*Rm*), the posterior longitudinal fasciculus (*Flp*), the nuclei and root fibers of the external rectus, of the facial, the vestibular and the cochlear branches of the eighth.

On the *right* there is hemianesthesia of the body and of the face for all modes of deep and superficial sensibility. Cerebellar hemiataxia and lateropulsion by lesion of the cerebellar pathways.

On the *left* there is atrophic facial paralysis. (*a*) superior facial, inferior of the platysma, lagophthalmia, drooping mouth angle, cochlear deafness, vestibular nystagmus, convergent strabismus, paralysis of the lateral associated movements of the eyes toward the left by lesion of the internuclear and vestibular left oculorotary fibers. By reason of the predominance of the antagonists the patient looks to the right. There is a velopalatine paralysis and a paralysis of the vocal cords (nuclei and root fibers of the spinal vagus). Hemianesthesia of the face by lesion of the descending root of the trigeminus (*a'*). (Dejerine.)

involvement of the posterior longitudinal fasciculus (*Flp*). Furthermore, there is a cerebellar hemiataxia from the cutting off of fibers going to the cerebellum (*Crst*). Involvement of the spinal vagus causes a palsy of the ipsolateral vocal cord, cutting off of the VIII causes deafness. Vestibular nystagmus also occurs from cutting off of vestibular fibers. Soft palate palsy is also present. Tumors of the pons give rise to a very varied syndromy.[1]

Postero-inferior Cerebellar Syndrome.[2]—The occlusion of the postero-inferior cerebellar artery is prone to produce a widespread pontine syndrome. It forms one of the apopletic bulbar palsies. The initial symptoms are apt to be acute dizziness, occasionally with transitory

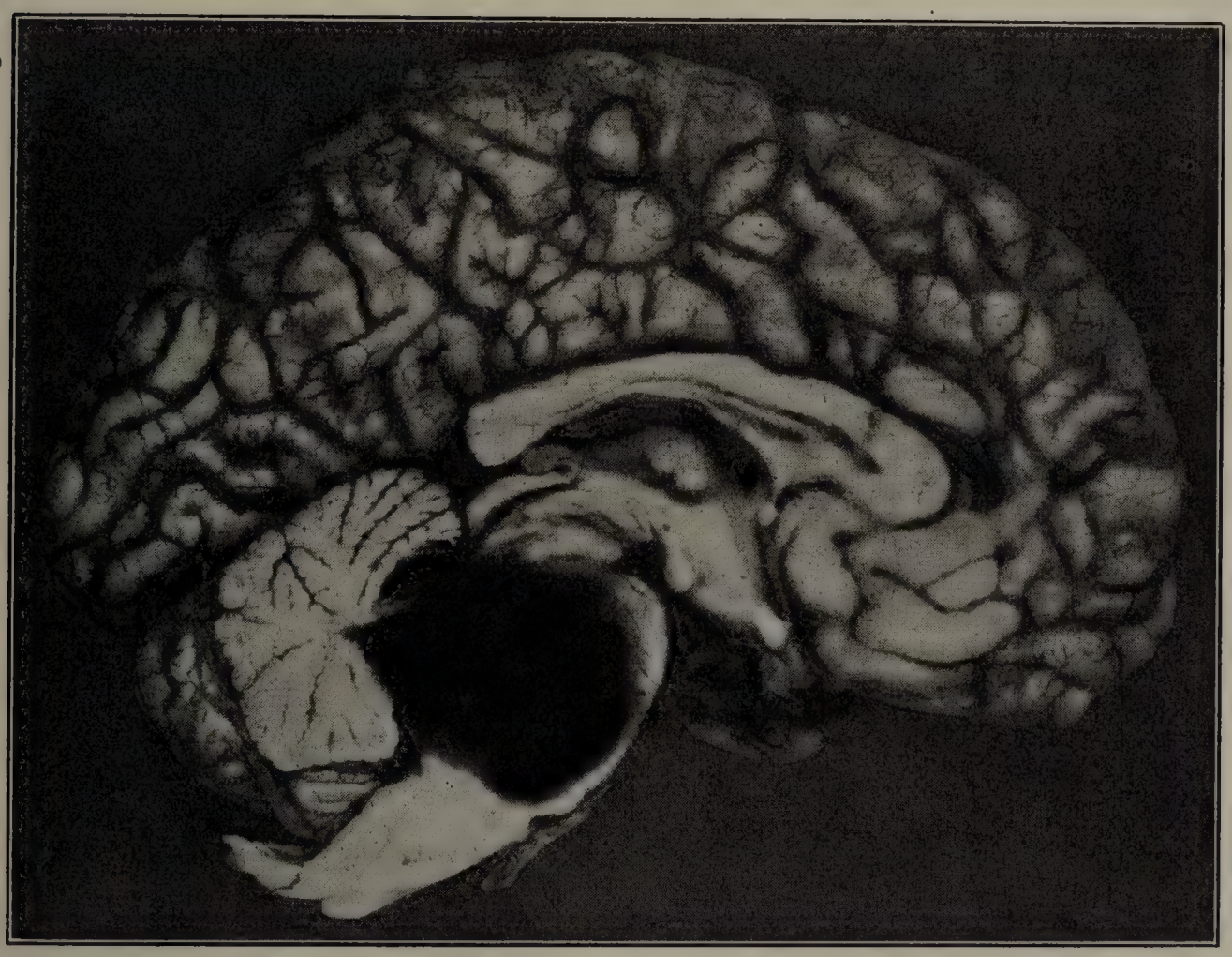


FIG. 323.—Hemorrhage of pons. [Compare Fig. 322.]

unconsciousness. There is slight, usually passing paresis of the opposite extremity with, it may be, transitory motor loss of fifth of the same side. Crossed hemianesthesia to pain and temperature and ipsolateral trigeminus sensory impairment of the first, second, or all three branch distributions, ipsolateral hemiataxia with lateropulsion to the side of the lesion. Bilateral nystagmus, worse on side looking toward lesion. Revolving vertigo, sometimes headache and vomiting. Avellis syndrome of palatal and laryngeal paresis on the ipsolateral side, causing dysphagia and a whispered speech, sometimes taste impairment in

[1] Varet: Thèse de Paris, 1905.

[2] Spiller: Jour. Nerv. and Ment. Dis., 1908, **35**, 365. Bury and Stopford: Med. Chron., December, 1913. Duhot: Paris méd., March 27, 1920. Claude and Lévy-Valensi: Loc. cit., 1922.

the anterior two-thirds of the tongue. Occasionally ipsolateral sixth and seventh nerve palsy from extension of the softening. Ipsolateral myosis, narrowing of the palpebral fissure, anhydrosis and enophthalmos. Occasionally ipsolateral loss of hearing.

The circulation of these portions of the cerebrospinal axis is subject to much variation. The postero-inferior cerebellar is usually given off from the vertebrals about 2 cm. from their basilar union. The anterior spinal is given off internally just below. This latter enters the raphe and is distributed along the raphe (see Fig. 313, *a* and *b*) to the antero-olivary bodies, posterior longitudinal fasciculus and twelfth nerve nucleus first on the floor of the fourth ventricle. The postero-inferior cerebellar artery is chiefly distributed to the lateral parts of the upper medulla and lower pons. Branches go to the corpus restiforme. (See Fig. 322, *Crst.*) There is so much variation in the arteries of this region that it is practically impossible to state always whether a clinical symptom-complex is due to vertebral or to inferior cerebellar artery disease.[1]



FIG. 324.—Weber's syndrome.



FIG. 325.—Weber's syndrome.

Peduncle Syndromes.—Lesions in the region of the upper level of the pons, lower border of quadrigemina, exit of III nerve (see Plate I), give rise to a very characteristic group of clinical pictures variously termed Gubler-Weber, Weber and Benedict syndromes.

These peduncular syndromes occur from lesions similar to those just discussed. Thromboses chiefly of the basilar, tumors, polioencephalitis, etc.

[1] Breuer and Marburg: Obersteiner's Arbeiten, 1902, **9**, 181. Blackburn: Jour. Comp. Neurol., 1907, **17**, 493. Duret: Arch. de physiol. norm. et path., 1873, p. 97 Wallenberg: Arch. f. Psychiat., **27**, 504.

Weber's Syndrome.—Superior alternate paralysis, Gubler-Weber.[1] These patients are taken acutely ill, are comatose or unconscious for the most part, occasionally as in Luton's case (earliest recorded) there is no loss of consciousness. If the patient recovers the residual paralyses show themselves. These will vary somewhat, depending upon the extent of the destruction in the peduncles.

When the lesion extends along the central raphe (as in Fig. 316), usually from arterial disease, an acute apoplectiform palsy, often with transitory or complete unconsciousness results. Many of these lesions, especially when extensive, result in sudden death. In the milder forms the various multiform after-pictures manifest themselves. The grave implication of the interspersed cortico-spinal fibers causes a contralateral hemiplegia with increased tendon reflexes, clonus and Babinski phenomena. Involvement of the cortico-facial fibers causes a contralateral lower facial palsy, paralysis of the tongue muscles, and of the motor fifth, deviation of chin to the sound side, protrusion of tongue to the paralyzed side. Hemianesthesia of the same side as the hemiplegia is also present. It involves the sense of position as well as all other types of sensibility on the entire half of the body. In middle-lying lesions the trigeminus may be spared. In more lateral-lying ones, posterolateral pontine syndromes (Fig. 321), the syndrome is less extensive.

In (Fig. 326, *a*) the lesion occupies the middle third of the right peduncle, cutting off only a portion of the cortico-spinal and cortico-nuclear pathways. There is a crossed hemiplegia of the entire side of the body, the extremities from the cutting off of the cortico-spinal, the inferior branches of the facial from the cortico-nuclear fibers. There are no atrophies and no reaction of degeneration, the paralyses being of the cerebral type. The tongue protrudes to the paralyzed side. In the early stages of a severe attack with coma or marked somnolence there may be no hypertonicity of the involved hemiplegic side, but later the increased knee-jerks, ankle-clonus and Babinski's phenomena appear. The lesion being confined below the locus niger gives rise to no sensory symptoms. In certain cases[2] (Gruenewald, Cestan) the lesion involves lemniscus fibers (Benedict syndrome types) and hemianesthesias appear.

On the same side of the lesion a III nerve palsy appears. There is ptosis and external strabismus. When the lesion extends, occupying two-thirds of the cerebral peduncles, additional symptoms appear (*b*). Thus there are added further cortico-nuclear involvements. Thus there is a conjugate deviation of the head and eyes, the head turned away from, the eyes looking toward the side of the lesion; there are motor

[1] Jelliffe: Superior Alternate Hemiplegia, Gubler-Weber Type, Int. Med. Jour., 1908, vol. **15**. Gubler: Gaz. hebd. de méd., 1856, **3**, 749, 789, 811; 1855, **5**, 721, 765, 801. Weber: Med. Chir. Trans., 1863. Gerber: Loc. cit. Dejerine: Loc. cit. Oppenheim: Loc. cit. Lewandowsky: Loc. cit. Claude and Lévy-Valensi: Loc. cit.

[2] Jelliffe: On Lesions of the Midbrain, with Special Reference to the Benedict Syndrome. Interstate Medical Journal, 1911, **18**, No. 8.

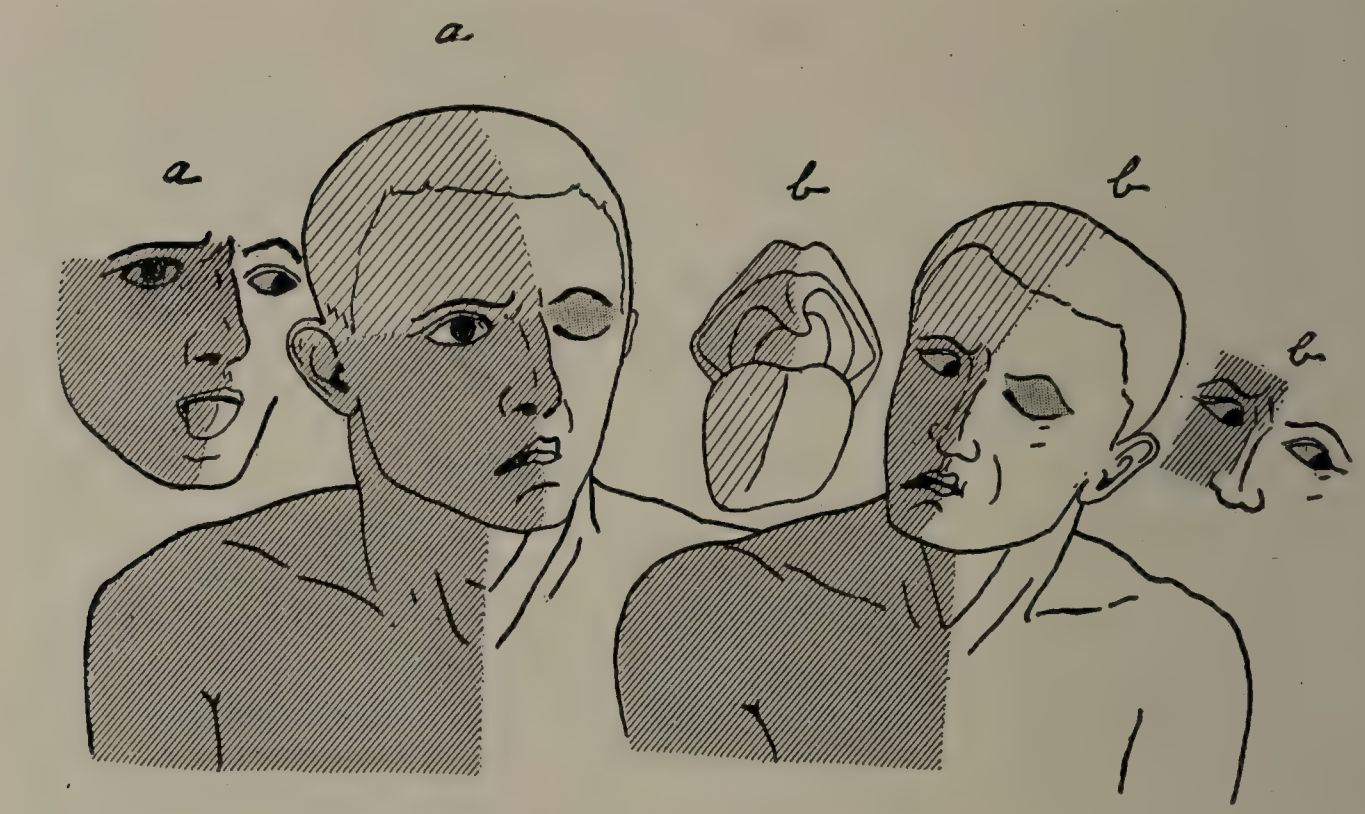


FIG. 326.—Anterior peduncular syndrome. Weber's syndrome. Superior alternate hemiplegia. Produced by cerebral peduncle and *III* root fiber lesion.

On the *right* side, contralateral hemiplegia of the trunk and extremities from lesion of the corticospinal pyramidal fibers. Facial hemiplegia inferior branches, hemiparesis of the tongue from lesion of the corticonuclear fibers of the hypoglossus. In (*a*) the lesion involves only a portion of the corticospinal and corticonuclear fibers. In (*b*) the lesion includes all of the pyramidal corticospinal and corticonuclear fibers, even those which in this region form the superficial and deep pes lemniscus fibers. (See Plate I.)

In addition to the symptoms common to (*a*) and (*b*) indicated in the illustrations, (*b*) also shows a conjugate deviation of the head and the eyes from a lesion of the cortical oculorotary fibers destined to go to the nuclei of the oculomotorius and abducens (*III–VI*); difficulties in mastication from lesions of the motor corticotrigeminus fibers; difficulties in swallowing, of phonation, and of articulation from lesions of the corticonuclear fibers of the spinal vagus.

As a result of the predominance of the antagonists of the sound side (left) in (*a*) and (*b*), there can be observed a deviation of the mouth (facial), and of the jaw (masticators) toward the left, a deviation of the tongue to the paralyzed side (action of sound genioglossus) and in (*b*) further a conjugate deviation of the head and of the eyes toward the left (action of the external branch of the spinal accessory and of the levorotary ocular fibers) and a deviation of the uvula toward the left (spinal vagus).

On the *left* side there is a direct paralysis of the third (*III*) nerve with ptosis, divergent strabismus, with or without mydriasis and pupils which do not react either to light or convergence, according to the grade of destruction of the root fibers of the oculomotorius (*III*). (Dejerine.)

fibers involved causing difficulties in chewing, and further, palsies of the soft palate and pharynx cause dysphagia. The uvula is deflected to the sound side (side of the lesion). There may be total mydriasis with loss of light and accommodation reflexes.

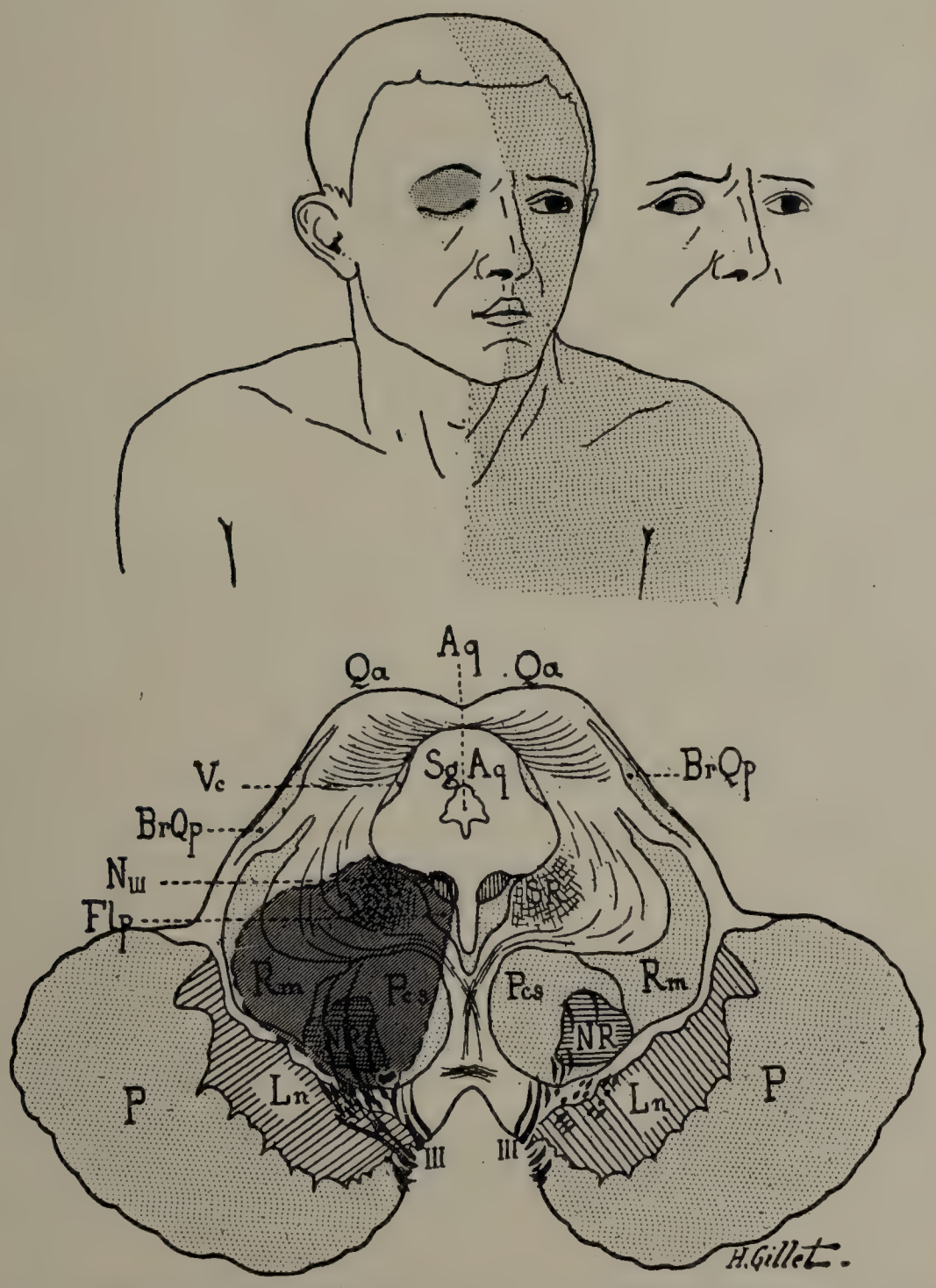


FIG. 327.—Posterior peduncular syndrome. Contralateral Benedict's syndrome. Hemianesthesia with choreo-athetoid movements and alternate paralysis of the third nerve (*III*) by reason of lesion of the right pontine tegmentum.

On the *left* side, contralateral hemianesthesia of the extremities and of the face from lesion of the secondary crossed sensory pathways (*Rm* and *SR*). Choreo-athetoid movements and at times tremors from lesion of the red nucleus and of the superior cerebellar peduncle (*Pcs*) below the decussation.

On the *right* side paralysis of the third nerve (*III*), with ptosis, external strabismus (non-resistance to external rectus), with or without mydriasis and pupils immobile to light and convergence, depending on the extent of the lesion of the root fibers of the oculomotorius (*III*). (Dejerine.)

Some of these patients make a fairly complete recovery, syphilitic arteritis with hemorrhage, but most show residuals and usually develop further attacks which lead to death.

Benedict's Syndrome.—Benedict first described this in 1872.[1] These patients usually show an apoplectiform onset. Benedict's first case was gradual in development (multiple tubercles). They may show the typical Weber hemiplegia and crossed third nerve palsy to which is added a hemianesthesia and a choreo-athetoid-like tremor.[2] When the lesion fails to reach the cortico-spinal or cortico-nuclear fibers in the peduncles there may be no hemiplegia.

Thus in the case illustrated (Fig. 327) there is a contralateral Benedict syndrome without any hemiplegia. There is a complete hemianesthesia from implication of the median lemniscus fibers (*Rm*). There is ptosis, external strabismus and pupillary immobility (complete or partial) from involvement of the III nerve. The choreo-athetoid movements result from cutting off of rubro-spinal (von Monakow) and rubro-cerebellar fibers.

These cases are due to hemorrhage, softening, tubercle, multiple sclerosis, syphilis, polioencephalitis, etc. The course and treatment will depend upon the causative lesion.

Corpora Quadrigemina Syndromes.—General lesions of the corpora quadrigemina bring about a syndrome often called Nothnagel's syndrome. Here the most characteristic pathological agents are tumors, either of the corpora quadrigemina themselves, or of the pineal body. When the roof of the midbrain is involved, there is usually headache and vomiting from increased intracerebral pressure. Optic neuritis often develops. Dizziness, staggering and rolling gait, with irregular forms of oculomotor palsy are present. The conjugate vertical movements of the eye are frequently involved, and not infrequently there is nystagmus. The pupils are usually widely dilated, or show anomalous reactions to light and accommodation. Hearing is modified if the posterior quadrigeminal bodies are involved. Tumors of the pineal body (see Pineal Syndrome) offer special features for consideration. These have been studied particularly by Marburg, Bailey and Jelliffe,[3] Horrax and others, and consist of: (1) General symptoms of intracerebral pressure; (2) oculomotor palsies with disturbed pupillary reactions and other signs of involvement of the corpora quadrigemina; (3) metabolic symptoms, due to the disturbed pineal itself, or more often to hydrops and pressure upon the *pituitary* stalk.[4] The metabolic symptoms are interesting, often consisting of adiposity, sexual precocity, with premature development of sexual characteristics, and occasionally cachexia. Lesions anterior to the midbrain involving the thalamus give a special symptomatology which is discussed under the head of Thalamic Syndrome (*q. v.*).

Colliculus Superior.—This is also known as the anterior quadrigeminal body. The general topographical anatomy has been discussed.

[1] Jelliffe: Loc. cit.; Benedict: Nervenpathologie, 1872.

[2] Jelliffe: On Some Obscure Tremors Due to Midbrain Lesions, Post-Graduate, 1914, p. 756.

[3] Tumors of the Pineal Body, Arch. Int. Med., 1911. Kidd: Rev. Neurol. and Psychiat., 1912, 1913.

[4] Bell: The Pituitary, 1919. Cushney: The Pituitary Body, Philadelphia, 1920. Also see literature quoted in section on vegetative neurology and endocrinophathies.

The finer anatomy of the superior colliculus shows it to be a highly complicated structure. It is arbitrarily separated from the lower structures of the midbrain. Only the most superficial features are here taken up. The serious student will find Winkler's *Manuel de Neurologie* (vols. 1–3) a dependable classic. For certain features of the implications of the optic pathways in this general region recent studies by Brouwer should be consulted.

A section at about the center of the midbrain shows the following structures: (1) An outer white layer, the stratum zonale, made up chiefly of fibers derived from the optic tract. There are also fibers from the superior quadrigeminal brachia and small horizontally lying nerve cells. (2) A gray layer, stratum cinereum, made up chiefly of ganglion cells whose axis-cylinders proceed inward. These are cells making up the synaptic junctures of the optic tract fibers and are concerned with establishing collateral associations with other nerve pathways, chiefly the oculomotor pathways to the nuclei of the eye muscles and also to those of the neck and body. (3) A layer of white fibers containing the greater mass of the optic tract fibers which are terminating about the ganglion cells of the third and the fourth layer. (4) A middle gray layer of cells or collateral associations. (5) The lemniscus layer made up probably of fibers of the median and lateral lemniscus. (6 and 7) Layers of ganglion cells with axones passing to the opposite side or passing ventrally to the aqueduct and central gray which decussate and form the tecto-bulbo-ponto-spinal tract going to the anterior columns for head, neck and trunk associations. Fibers from the spino-thalamic tracts and from the temporal and occipital cortex are also present.

This whole structure is part of the pathway for the mechanism of sight. It is in connection with: (*a*) Centripetal pathways: (1) From the optic tract through the anterior brachium of the superior colliculus. (2) From the spinal cord through the posterior columns, their nuclei, and median lemniscus. (3) With the spinal cord, lateral columns, tractus spinotectalis. (4) Occipital lobes through the internal sagittal layers.

(*b*) Centrifugal pathways: (1) To the occipital optic cortex by means of the anterior brachium. (2) To the medulla and spinal cord by means of the tecto-bulbo-spinal tracts. (3) To the nuclei of the oculomotor muscles, light reflexes, etc.

Thus the reflex collaterals in the superior colliculus are homologous to those in the inferior colliculus, the former serving to join up light stimuli with the rest of the body—with vegetative, sensorimotor and psychical levels, the latter subserving homologous functions for sound stimuli. These reflex collaterals do not subserve any sight functions in the narrower sense.

From these anatomical considerations it follows that a complicated symptomatology may result from colliculus lesions which will vary according to which tracts or groups of cells are involved. Minute lesions, as in poliomyelitis, encephalitis, cysticercus, small tubercles,

miliary aneurisms, may cause very few syndromes, isolated reflex disturbances, whereas gross lesions from pressure of tumors—pineal, tubercle, sarcoma, etc., may cause more widespread symptoms. One of the more roughly grouped of these is the so-called Nothnagel syndrome just described.

Lesions limited to the structures of the superior colliculus do not cause blindness nor hemianopsia. The lesion must extend as far as the geniculate bodies to reach those optic tract fibers which are continued farther back to the occipital cortex as sight fibers. Although fibers may go from the colliculus to the occipital lobes, just what relationship they have to seeing is still uncertain. The chief disturbances of pure colliculus superior disease are in the pupillary reflexes, eye movements, and balancing functions through eye movement.

The pathways taken by the pupillary fibers are not cleared up completely as yet.[1] They pass with the tractus in its middle portion, pass through the brachium anterius colliculi, also in the pulvinar surface, and end in part in the superficial gray layer of the superior colliculus. Here a synaptic junction takes place to connect up with the oculomotor. Lesions limited to these pathways may cause inequality of the pupils, possibly the Argyll-Robertson phenomenon. Such may follow trauma, alcoholism, or syphilitic meningeal infiltrative processes. The overwhelming majority of such lesions are syphilitic; poliomyelitis or infectious encephalitides occasion a smaller group of these disorders.[2]

A group of peculiar associated eye palsies are found in lesions affecting the colliculus itself or its connections with the eye-muscle nuclei. Thus a break in the connections between the abducens and the colliculus will result in a palsy of the rectus internus on looking to one or the other side or of only one side without any palsy of the abducens. Internus associated palsy without loss of convergence may occur (Fischer, Bielschowsky). The precise anatomical details are as yet lacking. Conjugate palsy has been frequently described as mostly due to tumors pressing upon the colliculus or involving it. Here the eyes may be directed to right or left but cannot be raised or lowered, or only one of these capacities is lost. In looking down the eyelids do not follow. Most of the described lesions are so gross that it is difficult to localize the precise mechanisms. Lewandowsky regards these palsies as due to a break in the pathways going from the cortex to the oculomotor nuclei.

The chief producing lesions are tumors (gumma, teratoma, sarcoma, abscess, cysticercus, etc.), multiple sclerosis, encephalitis, influenza, arteriosclerosis, etc.

More extensive eye-muscle palsies are referable to colliculus disease, especially if the lesion press farther caudad toward the nuclei themselves, or involve the posterior longitudinal bundle.

Ataxias are similarly present in disorder here from implication of

[1] See studies by Brouwer: Loc. cit. Also in his Herter Foundation Lectures, Williams & Wilkins, Baltimore, 1927.

[2] See Study by Riley: Neurological Bulletin, 1919, **2**, 181, on Conjugate Deviation of the Eye.

the eye-muscle functions of horizontal vision but they do not give a picture of pure colliculus disease, neither are the complex motor disturbances of choreic, paralysis agitans, or athetoid character such as have been described in von Monakow and other classics. These are due more particularly to cerebellar and vestibular pathway disturbances.

That the superior colliculus is associated with other motor reflexes is certain. Precise localizing symptomatology is evolving. The reflex collaterals with the vegetative permit the fear reactions through sight —and all of the considerations concerning the relation of the psyche mentioned in the paragraph on the inferior colliculus.

Colliculus Inferior.—Also known as the posterior quadrigeminal body. Bechterew (1885) was among the earlier investigators of the inferior colliculus to show its relations to the cochlear portions of the acoustic nerve and hence its importance in hearing. For the most part the results obtained by him in Flechsig's laboratory have been verified and amplified by the work of Held, Van Gehuchten, Lewandowsky, Winkler, Spitzer and Karplus. The inferior colliculus, in part with the median geniculate body, form the two chief secondary synapses of the auditory pathway. The neurons of this pathway are as follows:

First Neuron: Sensory receptor in Corti's organ, ganglion spirale; (*a*) ventral cochlear nucleus and (*b*) acoustic tubercle.

Second Neuron: (*a*) Ventral cochlearis nucleus—corpus trapezoides superior olives—ventral and dorsal lemniscus nuclei of opposite side; (*b*) acoustic tubercle and striæ acousticæ, superior olive of both sides.

Third Neuron: Superior olive, lemniscus nuclei (lateral lemniscus). colliculus inferior (posterior quadrigemina).

Fourth Neuron: Inferior colliculus, arm of colliculus, median geniculate body.

Fifth Neuron: Median geniculate—temporal-auditory cortical area.

Cajal assumes that the cells of the inferior colliculus are not direct but collateral synapses for shunting auditory stimuli (reflexes) to other mechanisms, ear movements, head movements, voice production, eye movements in part. Anatomy teaches that the pathways for sound are crossed and uncrossed as are other sensory pathways, hence a lesion of one colliculus causes no marked deafness, although interfering somewhat with hearing and more especially with many auditory reflexes. Experimental stimulation of the inferior colliculus has brought about dilatation of the pupil of the opposite side—raising of eyebrows, protrusion of eyeballs (autonomic stimulation from fear reactions from sound stimuli in normal physiology), turning of the head up toward the non-stimulated side, movement of ears (in animals) and a number of laryngeal and respiratory stimuli causing voice production (cries, grunts, etc.). Ferrier, who performed a number of such experiments, came to believe that a psychical center was revealed. Prus obtained somewhat similar results, which, with the information available from the studies in the vegetative nervous system, would tend to show that the auditory synapses in the inferior colliculus constitute a large factor in psychical reactions to sound stimuli as expressed through the autonomic (vagus) distribution. Cocainization of these bodies

caused clonic convulsions, raising of the head, movements of the eyes to the opposite side, dilatation of the pupils, exophthalmos, cardiac-cramp, and loud crying, *i. e.*, typical epileptiform phenomena. Prus interpreted the results as showing a motor center. Seen, however, from the viewpoint of vegetative neurology it would appear that disharmonic vagus activities come into activity (Lewandowsky) through cutting off of psychical (thalamic homologue) control, *i. e.*, thalamic overresponse occurred in the sense in which Head has described it for the thalamic lesions in which tactile sensibility was under discussion. These reflections would lead to a review of the subject of the psychogenic factors in certain epileptic attacks associated with infantile fear shock conditioned by auditory impressions. In this connection Oppenheim's statement that overresponse to sounds is seen in diplegias, might have a wider significance than he seems to give it. Furthermore, the problem of tone production, as contrasted with tone perception, acquires wider significance than that given to it by the original Helmholtz peripheral hypothesis, since the reflex activities of the tensor tympani are apparently involved in colliculus inferior disorder. Bechterew has further shown that the genital apparatus (erection, contraction of uterus, stimulation of milk secretion) is also reflexly affected through the inferior colliculus. As the autonomic pelvic functions are homologues of the vagus autonomics this seems logical, and contributes further light on the psychoanalytic hypotheses of repression of the libido and certain epileptic attacks (Maeder). From the reflex, somatic side, a study by Frey and Fuchs is of interest on the subject of reflex epilepsy and ear and nose disease, also the classical reports of Hughlings Jackson and Ormerod.

Clinical neurology still lags behind the anatomical and physiological correlations. Charcot has noted a tabetic with deafness in whom the inferior colliculus was involved. Flechsig has recorded hallucinations of hearing and Weinland has called attention to disturbances of hearing from lesions of the inferior colliculus, and possibly incorporating other geniculate fibers. The relationships of disturbed autonomic functions to collicular disease have not received sufficient attention from neurologists and otologists. The literature on deafness with dumbness has not been gone into. The tinnitus of neurasthenia, of the psychoneuroses, the great importance ascribed to sounds in many psychoses, hallucinations in schizophrenics, in manic-depressives, have as yet received no satisfactory analyses from either the neurological or the psychiatric disciplines.

RABIES.

Rabies is an acute, specific, infectious disease, which, after the symptoms have appeared, is almost, if not quite always, rapidly fatal. While the disease may occur in any mammal, it almost always results in man from the bite of the dog. The virus travels from the wound along the nerve trunks *via* the spinal cord to the medulla and brain much in the same way as does the tetanus toxin. It is contained in the saliva of the rabid dog. For these reasons wounds upon the exposed

portions of the body and those containing a large nerve supply are most dangerous.

The *incubation* period varies within wide limits, but in man on the average it is about forty days, though it may be prolonged for a number of months.

Symptoms.—The symptoms, appearing, as a rule, from two to six weeks after the wound, are at first pain and redness in the wound itself and a condition of depression, restlessness, headache, and insomnia—the prodromal stage. Two to four days later the stage of excitement begins with tonic spasms of the muscles of the pharynx and larynx, with involvement also of the respiratory muscles, producing difficulty of respiration. Increased flow of saliva and severe thirst is accompanied with an inability to swallow, because attempts at swallowing produce spasm of the muscles of the pharynx. It is because of this symptom that the patient fears to touch water, thus the common name of the disease—hydrophobia. The excitement increases rapidly for from one to three days, when the paralytic stage begins, with a rapidly developing weakness and paralysis involving the muscles of the face, tongue, and the eye muscles. Temperature rises and death occurs in a few days.

Pathology.—The most characteristic features of the pathology are: (1) the presence of the so-called Negri bodies (Neurocytes hydrophobiæ), which occur especially in Ammon's horn. The nature and meaning of these bodies is not altogether clear. Negri, as indicated by the name above, presumed them to be protozoa; (2) the so-called nodules rabiques of Babes, which are collections of small foci of mononuclear cells about the spinal cord nerve cells; (3) the lesion of Van Gehuchten and Nélis. These lesions are found in the peripheral ganglia of the cerebrospinal and sympathetic nervous systems, especially in the plexiform ganglia of the pneumogastric and in the Gasserian ganglia, and consist of a proliferation of the endothelial cells surrounding these ganglia, the nerve cells being pushed aside and even destroyed, the ganglia finally containing many round cells.

Treatment.—The immediate treatment of a wound inflicted by a suspected animal should be thorough cauterization by fuming nitric acid, if possible. In its absence the actual cautery may be used. Strong solutions of carbolic acid and formalin are poor substitutes, while nitrate of silver is of no value.

The most important treatment, however, is the Pasteur treatment. The principle of this treatment is the production of an active immunity by the use of an attenuated virus. For the details of the treatment the student is referred to special works. In any event, the general practitioner cannot apply it, but must take his patient to a laboratory especially equipped for the purpose.

The principle of the treatment is to produce immunity before the appearance of the symptoms. Inasmuch as the incubation period is on the average forty days, this is possible, as immunity is produced, by the injection of an attenuated virus, at the end of two weeks. This method of treatment has materially reduced the fatality of this disease.

ANALYSIS OF SYMPTOMS RESULTING FROM LESIONS AFFECTING THE PEDUNCULAR PATHWAYS.[1]

Level of lesion.	Topography of lesion.	Structures involved.	Etiology.	Symptoms.	Special names and remarks.
1. Cortical . . .	Motor area of cerebral cortex.	Pyramidal cells of Betz, irritative and destructive.	Thrombism, embolism, hemorrhage, tumor, tuberculous meningitis, encephalitis, meningo-encephalitis, traumatic meningeal hemorrhage.	Contralateral hemiplegia; convulsions (Jacksonian) common; aphasia frequent, intelligence often disturbed; may be monoplegia.	Cortical hemiplegia.
2. Subcortical . .	Centrum ovale or corona radiata.	Pyramidal cell axons while passing through medullary substance of brain.	Same as above	Contralateral hemiplegia; Jacksonian convulsions rare; monoplegia rare; intelligence seldom disturbed.	Subcortical hemiplegia.
3. Capsular . . .	Genu and posterior limb of internal capsule.	(1) Corticonuclear; (2) corticospinal contingents of pyramidal system.	Hemorrhage most common. Thrombus, embolism, tumors, cysts, and abscesses.	Contralateral hemiplegia; convulsions, aphasia, and mental disturbance absent.	Capsular hemiplegia.
4. Capsulothalamic .	Genu and posterior limb of capsule, with postero-inferior part of thalamus.	(1) Corticonuclear; (2) corticospinal contingents of pyramidal system with (3) thalamus.	Same as above.	Contralateral hemiplegia; hemianesthesia; hemiataxia; hemiathetosis and sometimes hemianopsia; pain in affected parts.	Capsulothalamic hemiplegia; thalamic syndrome of Dejerine.
5. Subthalamic . .	Mesial basal	(1) Pyramids; (2) subthalamic corticonuclear fibers.	As above	Contralateral hemiplegia; vertical paralysis of gaze (bilateral).	Subthalamic syndrome Foville
6. Peduncular (*a*) .	Basal.	(1) Pyramidal fibers; (2) pes lemnisci profundus and superficialis; (3) emergent III nerve fibers.	Same as above.	Contralateral hemiplegia involving face, tongue, arm, and leg; ipsilateral ophthalmoplegia.	Basopeduncular hemiplegia; Weber's syndrome; hemiplegia alternans superior (Fig. 320).
7. Peduncular (*b*) .	Tegmental.	(1) Superior cerebellar peduncle; (2) fillet; (3) red nucleus; (4) pes lemnisci; (5) emergent III nerve fibers.	Same as above.	Contralateral hemichoreoathetosis; hem tremor and hemianesthesia; ipsilateral external ophthalmoplegia.	Tegmentopeduncular hemianesthesia; Benedict's syndrome; hemiplegia alternans inferior (Fig 321).
8. Peduncular .	Mesial basal.	(1) Pyramids; (2) oculogyric fibers (3) cephalogyric fibers.	Inflammations, etc.	Contralate al hemiconvulsion; in convulsive type, eyes gaze at convulsed limbs	Peduncular convulsive syndrome of Foville
9. Peduncular . .	Same as above.	Same as above.	As above.	Contralateral hemiplegia; in paralytic type, eyes gaze at sound side.	Peduncular paralytic syndrome of Foville
10. Pontile . . .	Basal and tegmental.	(1) Pyramids; (2) post. long. fasc. (3) facial nucleus; (4) fibers of VI nerve.	As above.	Contralateral hemiplegia; ipsilateral internal strabismus; facial palsy; oculogyric paralysis.	Pontile syndrome of Foville (Fig. 168).
11. Pontile (*a*) cephalic . . .	Basomesial.	(1) Corticospinal fibers; (2) co ticonuclear to V, VII and XII nerves.	Hemorrhage embolism, thrombosis, tumor, or solitary tubercle, syphilis.	Contralateral hemipleg a; hemipropoplegia; hemiparesis; masticatory hemiparesis; lingualis.	Cephalic pontile basal hemiplegia.

[1] By coöperation with Dr. Frederick Tilney.

ANALYSIS OF SYMPTOMS RESULTING FROM LESIONS AFFECTING THE PEDUNCULAR PATHWAYS—Continued.

Level of lesion.	Topography of lesion.	Structures involved.	Etiology.	Symptoms.	Special names and remarks.
12. Pontile (*b*) cephalic	Mesial, basal, bilateral.	(1) Pyramids of one side complete; (2) pyramid of other side partial; (3) corticonuclear to VII and XII nerves on both sides.	Thrombosis of basilar artery.	Complete hemiplegia of one side; partial hemiplegia of other side; paralysis of both sides of face; paralysis of both sides of tongue with dysarthria.	Cephalic pontile mesial basal bilateral hemiplegia.
13. Pontile (*c*) cephalic	Basotegmental, disseminated.	(1) Fasciculus longitudinal posterior (internuclear oculogyric fibers); (2) pyramidal; (3) corticonuclear to VII and XII nerves.	Multiple foci, multiple sclerosis, syphilis.	Contralateral hemiplegia; contralateral paralysis of VII and XII nerves; contralateral fixed conjugate deviation in extreme lateral position; cannot move eyes out of this position; looks toward paralyzed limbs.	Cephalic pontile basotegmental;(disseminated) hemiplegia, with the syndrome of Foville (internuclear type).
14. Pontile (*d*) cephalic	Basotegmental	(1) Pyramids; (2) cortical oculogyric fibers; (3) cortical cephalogyric fibers; (4) corticonuclear fibers to VII and XII nerves.	Thrombosis.	Contralateral hemiplegia; ipsilateral conjugate deviation of eyes (patient looks to side of lesion); head inclined toward side of lesion; contralateral paralysis of tongue and face	Cephalic pontile basotegmental hemiplegia, with the syndrome of Foville (oculogyric and cephalogyric type).
15. Pontile (*a*) middle	Mesial basotegmental	(1) Pyramid; (2) corticonuclear to V, VII, and XII nerves; (3) fillet.	Thrombosis of middle portion of basilar artery.	Contralateral hemiplegia and contralateral paralysis of face, tongue, and masseters; contralateral hemianesthesia involving all sense qualities, but especially posture sense.	Middle pontile mesial basotegmental hemiplegia.
16. Pontile (*b*) middle	Lateral basotegmental.	(1) Motor and sensory V nucleus; (2) lateral portion of fillet; (3) superior cerebellar peduncle; (4) pyramidal in part; (5) corticonuclear fibers to V nerve.	Thrombosis, hemorrhage or tubercle (?), multiple sclerosis, syringomyelia, syphilis.	Ipsilateral paralysis of jaw muscles, ipsilateral hemianesthesia of face, contralateral anesthesia of head, neck, arm, and leg (less here); ipsilateral hemichorea-athetosis; contralateral hemiparesis, contralateral paresis of jaw muscles.	Middle pontile lateral basotegmental hemiplegia; alternating trigeminal hemianesthesia.
17. Pontile (*c*) middle	Lateral tegmental.	(1) Motor V nucleus, also sensory; (2) superior cerebellar peduncle; (3) spinothalamic tract; (4) fillet in part.	Same as above.	Ipsilateral paralysis of jaw muscles; ipsilateral hemianesthesia of face; ipsilateral hemichorea-athetosis; contralateral hemianesthesia (most for pain and temperature).	Middle pontile lateral tegmental; dissociated hemianesthesia.

ANALYSIS OF SYMPTOMS RESULTING FROM LESIONS AFFECTING THE PEDUNCULAR PATHWAYS—Continued.

Level of lesion.	Topography of lesion.	Structures involved.	Etiology.	Symptoms.	Special names and remarks.
18. Pontile (*a*) caudal	Transverse tegmental.	(1) Fillet; (2) F. L. P.; (3) N. VI; (4) N. VII; (5) vestibular and cochlear VIII; (6) corpus restiforme; (7) descending V.	Tubercle, multiple sclerosis, syringomyelia, syphilis.	Contralateral hemianesthesia; hemiataxia and lateropulsion; contralateral conjugate deviation; ipsilateral deafness and nystagmus; ipsilateral paralysis of palate and vocal cord (?); ipsilateral hemianesthesia of face, and ipsilateral paralysis of face.	Caudal pontile transverse tegmental hemianesthesia, with nystagmus and dysphonia (with syndrome of Foville).
19. Pontile (*b*) caudal	Basomesial	(1) Pyramids; (2) emergent VI nerve fibers, or (3) emergent VI and VII nerve fibers.	Hemorrhage, thrombosis, embolism, tubercle, or multiple sclerosis.	Contralateral hemiplegia; ipsilateral internal strabismus or ipsilateral internal strabismus and facial palsy.	Caudal pontile basomesial; inferior alternating hemiplegia of Millard-Gubler type
20. Pontile (*c*) caudal	Mesial basotegmental.	(1) Pyramids; (2) fillet; (3) F. L. P.; (4) emergent fibers of VI and VII nerves.	Thrombosis of basilar artery or left medial pontine branches.	Contralateral hemiplegia; contralateral hemianesthesia; contralateral ocular deviation (conjugate); ipsilateral internal strabismus and facial palsy.	Caudal pontine mesial basotegmental; inferior alternating hemiplegia of Millard-Gubler-Foville type
21. Pontile (*d*) caudal	Transverse tegmental incomplete.	(1) Fillet incomplete; (2) descending V; (3) emergent VII nerve fibers and VI nerve; (4) spinothalamic tract, (5) Deiters' nucleus.	Hemorrhage, etc.	Contralateral hemianesthesia for heat and cold; contralateral hemianesthesia incomplete; ipsilateral internal strabismus and facial palsy; contralateral conjugate deviation.	Caudal pontile transverse tegmental; incomplete hemianesthesia with the syndrome of Foville.
22. Pontile caudal .	Basal and tegmental.	(1) Pyramids; (2) fillet; (3) inferior cerebellar peduncle; (4) post. long. fasc.	As above.	Contralateral hemiplegia and hemianesthesia; ipsilateral tremor; hemiathetosis; hemiasynergia; hemiataxia; oculogyric paralysis.	Pontile syndrome of Raymond and Cestan
23. Bulbar	Basal and tegmental.	(1) Pyramids; (2) labyrinthine oculogyric fibers.	As above.	Contralateral hemiplegia, ipsilateral oculogyric paralysis.	Bulbar labyrinthine syndrome of Foville.
24. Bulbar	Basal and tegmental.	(1) Pyramids; (2) post. long. fasc.	As above.	Contralateral hemiplegia; ipsilateral oculogyric paralysis.	Bulbar internuclear syndrome of Foville.
25. Bulbar (*a*) olivary	Mesial basotegmental.	(1) Pyramids; (2) fillet; (3) nucleus XII.	Thrombosis of anterior spinal artery.	Contralateral hemiplegia; contralateral hemianesthesia (posture sense mainly); ipsilateral paralysis of tongue.	Olivary bulbar mesial basotegmental hemiplegic hemianesthesia; alternating hypoglossal hemiplegia, hemianesthesia.
26. Bulbar (*b*) olivary	Transverse basotegmental (incomplete).	(1) Pyramids; (2) fillet; (3) XII fibers; (4) cerebellar afferents; (5) oculopupillary fibers; (6) nucleus ambiguus; (7) descending V.	Thrombosis of vertebral artery.	Contralateral hemiplegia; contralateral hemianesthesia; ipsilateral hemiataxia and hemiasynergia; ipsilateral myosis, glossoplegia, laryngoplegia, cephalogyric paralysis, and hemianesthesia of face.	Olivary bulbar transverse basotegmental (incomplete hemiplegia; alternating hypoglossal hemiplegia, hemianesthesia, including syndrome of Avellis.

ANALYSIS OF SYMPTOMS RESULTING FROM LESIONS AFFECTING THE PEDUNCULAR PATHWAYS—Continued.

Level of lesion.	Topography of lesion.	Structures involved.	Etiology.	Symptoms.	Special names and remarks.
27. Bulbar (*c*) olivary	Mesial basotegmental bilateral.	(1) Pyramids; (2) fillets; (3) emergent XII fibers.	Thrombosis of anterior spinal artery.	Bilateral hemiplegia; bilateral hemianesthesia; ipsilateral glossoplegia.	Olivary bulbar mesial basotegmental bilateral hemiplegia; hemianesthesia; alternating hypoglossal bilateral hemiplegia, hemianesthesia.
28. Bulbar (*a*) postolivary . . .	Lateral tegmental.	(1) Cerebellar afferents; (2) spinothalamic tract; (3) descending V; (4) N. ambiguus; (5) sometimes oculopupillary nucleus; (6) hypoglossal nerve fibers.	Thrombosis of inferior and posterior cerebellar arteries.	Ipsilateral lateropulsion; hemiasynergia; hemiataxia; hemifacial anesthesia; palatoplegia, glossoplegia; laryngoplegia and myosis; contralateral anesthesia to pain and temperature.	Postolivary bulbar lateral tegmental hemiataxia, including the syndrome of Avellis.
29. Bulbar (*b*) postolivary . . .	Nucleus ambiguus and nucleus accessorius spinalis.	Nucleus of X and XI nerves.	Hemorrhage, tubercle or may be traumatic injury of nerves.	Ipsilateral palatoplegia; laryngoplegia and cephalogyric paralysis of same side.	Syndrome of Schmidt.
30. Bulbar (*c*) postolivary . . .	Nucleus ambiguus, nucleus spinal accessorius, nucleus hypoglossi.	Nuclei of X, XI, and XII nerves.	Same as above.	Ipsilateral palatoplegia; laryngoplegia and cephalogyric paralysis of same side, with ipsilateral paralysis and atrophy of tongue.	Syndrome of Jackson.
31. Bulbar (*d*) postolivary . . .	Nucleus ambiguus.	Nucleus of X nerve; fillet; spinothalamic tract.	Syringobulbia, tabes dorsalis, always central.	Ipsilateral paralysis of palate, vocal cord, and pharynx; contralateral hemianesthesia and loss of pain temperature sense (sensory troubles not essential).	Syndrome of Avellis.
32. Bulbar (*e*) postolivary . . .	Mesolateral tegmental.	Nucleus X, nucleus XII; or X and XII nerves.	Usually a peripheral lesion.	Ipsilateral paralysis of tongue and vocal cord.	Syndrome of Tapia.
33. Bulbar (*f*) postolivary . . .	Mesial basotegmental (irregular).	(1) Pyramids; (2) fillet; (3) inferior cerebellar peduncle; (4) reticular formation.	Multiple lesions in distribution of vertebral artery.	Contralateral hemiplegia; and hemianesthesia, ipsilateral hemiasynergia, and lateropulsion; myosis and enophthalmia.	Syndrome of Babinski-Nageotte.
34. Bulbar (*g*) postolivary . . .	Mesial basotegmental.	(1) Pyramids; (2) fillet; (3) inferior cerebellar peduncle; (4) reticular formation.	Thrombosis of vertebral artery.	Contralateral hemiplegia and hemianesthesia; ipsilateral hemiasynergia and lateropulsion, myosis, enophthalmia and velo laryngeal paralysis (Avellis type).	Syndrome of Cestan-Chenais.

TETANUS.

Etiology.—The cause of tetanus is the *Bacillus tetani*. This is an anaërobic bacillus, rather widely distributed, normally inhabiting the intestinal tracts of herbivora, particularly of horses and cows, and also of man. It is a spore-forming organism. The spores are extremely resistant to destructive agencies and are found in the soil, especially soil which has been contaminated by the feces of horses and of man. The symptoms are due to a toxin which travels up the axis-cylinders.

The *incubation* period varies within considerable limits, from three or four days up to as many as twenty days, the severity of the infection being fairly well indicated by the earliness of the onset of symptoms.

Symptoms.—The symptoms of the disease are essentially those of tonic spasmodic contractions of the voluntary musculature. The muscles involved at first feel stiff and are subsequently thrown into convulsions, particularly the face, giving rise to the characteristic *risus sardonicus*. Later the trunk muscles are involved, producing severe convulsions and bending of the body toward those most affected. The muscles of mastication are early involved, while the spasm of the muscles of respiration and of the larynx interfere with breathing and hasten exhaustion and the end. Facial paralysis is an occasional complication when the point of entry of the infection has been the face. Slight stimulation, such as noises, bring on convulsions.

Course and Diagnosis.—In the severe forms death generally eventuates in three or four days. Strychnine poisoning, tetany, hysterical and epileptic convulsions, hydrophobia, and meningitis are to be differentiated.

Treatment.—The prophylactic treatment is largely surgical, involving the proper treatment of wounds, particularly punctured, contused, and infected wounds. The bacillus appears to thrive especially in mixed culture, and so infected wounds are especially dangerous. The specific treatment is by the use of tetanus antitoxin, which should be administered after any suspicious wound, without waiting for symptoms. A prophylactic dose of about 1500 units may be given. As soon as symptoms appear, however, the larger dose, about 20,000 units should be administered. It is well, too, to inject some antitoxin into the large nerve trunks leading from the wound, and dry tetanus antitoxin may be dusted upon the wound itself. As the antitoxin is eliminated in about two weeks, if further effects are desired from it, additional injections will be necessary. After the toxin has combined with the motor nerve cells it cannot be displaced or neutralized by antitoxin. The antitoxin can only neutralize the free toxin. Good results have been reported by the intravenous injections of magnesium sulphate, in conjunction with the use of antitoxin.

The *prognosis* has been materially improved since the advent of antitoxin treatment.

CHAPTER VIII.

PARALYSIS AGITANS, LENTICULAR DEGENERATION, DYSTONIA MUSCULORUM, CHOREA, AND RELATED STRIATUM SYNDROMES.

THE complicated anatomical structures of the interbrain and the basal ganglia region will not be discussed here. The student is referred to standard anatomical treatises.[1]

Particular attention should be called, however to the basal ganglia because of the rapid extension in the analysis of certain neurological syndromes connected with lesions located in and about them. These ganglia are the: (1) Corpus striatum, made up of the caudate and lenticular nuclei; (2) the claustrum; (3) the amygdaloid nucleus.

The lenticular nucleus is roughly separable into an outermost area, the putamen and an inner area, the globus pallidus. The caudate nucleus is more or less anatomically and functionally related to the putamen. The globus pallidus is an old sensory motor system (paleostriatum), the caudate and putamen are comparatively new, phylogenetically speaking (neostriatum). A text-book presentation cannot go into all the extremely complicated situations connected with the analysis of this intricate region which is developing with great rapidity. Of the recent monographs, that of C. and O Vogt[2] stands out as a pioneer contribution. The literature to 1920 is given there. That to 1922 is given in Foerster's full discussion. More recent valuable clinical and anatomical studies are those of Souques, Pollak, Magnus, Jakob, Foix and Nicolesco, Lévy, Marburg, Hunt, Rademaker, v. Economo and Kappers, to mention but a few. The literature on epidemic encephalitis alone has 5000 titles (1927), a large number of which deal with anatomical and clinical problems of the interbrain and basal ganglionic regions. A rapid appraisal of these various studies develops the fact that a number of interrelated syndromes (dyskinesias) are recognizable due to irregular involvements of the general region of the striatum. To speak of definite *diseases* is misleading, if not quite erroneous. While certain trends, such as the senile types of the Parkinsonian syndrome, run so fairly definite a trend as to permit one

[1] Dejerine: Anatomie du Systeme Nerveux: Ziehen in Bardleben. Barker: The Nervous System. Winkler: Manuel de Neurologie. Tilney and Riley: Form and Functions of the Nervous System. Kappers: Vergleich. Anat.; loc. cit; Ontogenetic Development of Corpus Striatum in Birds. Konink: Ak. v. Wet. F., Amsterdam, 1923, **36**, 4. Magnus: Die Körperstellung, 1925. Jakob: Loc. cit. Foix and Nicolesco; Keschner: In Tice's Medicine, vol. **10**.

[2] Vogt, C. and O.: Zur Lehre der Erkrankungen des Striären Systems, Jour. f. Psychiat. u. Neurol., 1921, **25**, Ergänzungsheft 3; abstracted in English by Winkelmann: Arch. Neurol. u. Psychiat., 1923, **10**, 563. Lewy, Tonus und Bewegung, 1923.

to speak of paralysis agitans as a *disease;* this is only a concession to bad academic habits of nosology, which, while needed by the beginner, should be laid aside for better thinking in neurodynamics.

Thus among the striatum syndromes which have received more or less definite names are to be recognized paralysis agitans, dystonia musculorum deformans, Vogt's double athetosis, Westphal's sclerosis, Wilson's lenticular degeneration, tuberous sclerosis, Huntington's chorea. The analysis is far from being complete. Vogt and Bielschowsky's presentation offers useful summaries. Thus may be distinguished: (1) Status marmoratus, "Etat marbé," first described by the Vogts under the title *double athetosis* (certain examples of *Little's disease* may be here included). (2) The "Etat dysmyelenique," or progressive degeneration of the fibers passing between the pallidum, and the thalamus, and the hypothalamus. In one of Jelgersma's cases this showed a *paralysis agitans* syndromy. (3) Status fibrosus, "Etat fibreux," which shows as a progressive bilateral *chorea* without necessary psychical symptoms. (4) An initially acute and then stationary fibrosis as a partial expression of a cerebral hemiatrophy (Bielschowsky) with non-progressive athetosis and minimal paresis of the bodily musculature. (5) Total necrosis of the striatum confined chiefly to the putamen, at times reaching other parts of the globus pallidus. Here Wilson's *lenticular degeneration* types are chiefly to be included, and some cases of *dystonia musculorum.* (6) Acute infectious striatum syndromes during or following epidemic encephalitis, poliomyelitis, influenzal encephalitis, etc. Parkinsonian types, paramyoclonus, myoclonus, athetotic, choreic, tic, bulbar palsy, involuntary laughing and crying, hiccoughing, syndromes. (7) Presenile degenerations involving chiefly the striatum and pallidum combined with neuroglia proliferations in the striatum. (8) Status disintegratus with Parkinsonian symptoms in which the striatum is involved either as (*a*) general massive atrophy, chiefly of caudatus, cells and fibers, (*b*) small lacunar softenings or hemorrhages, (*c*) rarefaction and absorption related to Marie's lacunar softenings. The lenticular degeneration of carbon monoxide poisoning may be included.[1]

As Wilson, as well as others, has clearly emphasized there is a tendency to put too much upon the striatum, and hasty generalization has erected syndromes of no lasting value. The student is expressly cautioned that a text-book, other than a dogmatic quiz compend, can only hope to record the more or less well-agreed-upon types. He

[1] For literature, see among the many references of recent times the following: Thomalla: Ztschr. f. d. ges. Neurol. u. Psychiat., 1918, vol. **41**. Vogt: Sitzungsbericht d. Heidelberg Akad. d. Wiss., 1919. Bielschowsky: Monatschr. f. Psychiat. u. Neurol., 1918, **24**, 20. Foerster: Ztschr. f. d. ges. Neurol. u. Psychiat., 1921, **73**, 1. Hunt, J. R.: Trans. Am. Neurol. Assn., 1920. Spiller: Jour. Nerv. and Ment. Dis., 1916, **43**, 23; Trans. Am. Neurol. Assn., 1920. Lewy, Tonus und Bewegung, 1923. Boestroem: Die amyostatische Symptom-complex, 1923. Magnus: Körperstellung. Jakob: Die Extrapyramidale Erkrankungen, 1923. Souques: Rev. neurol., 1921. Wilson: Croonian Lectures, Lancet, July 4, 1925. Spiegel: Der Tonus der Skelettmuskulatur, Berlin, 1927. Spatz: Physiologie und Pathologie der Stammganglien, Berlin, 1927. Lotmar: Die Stammganglien, Berlin, 1926.

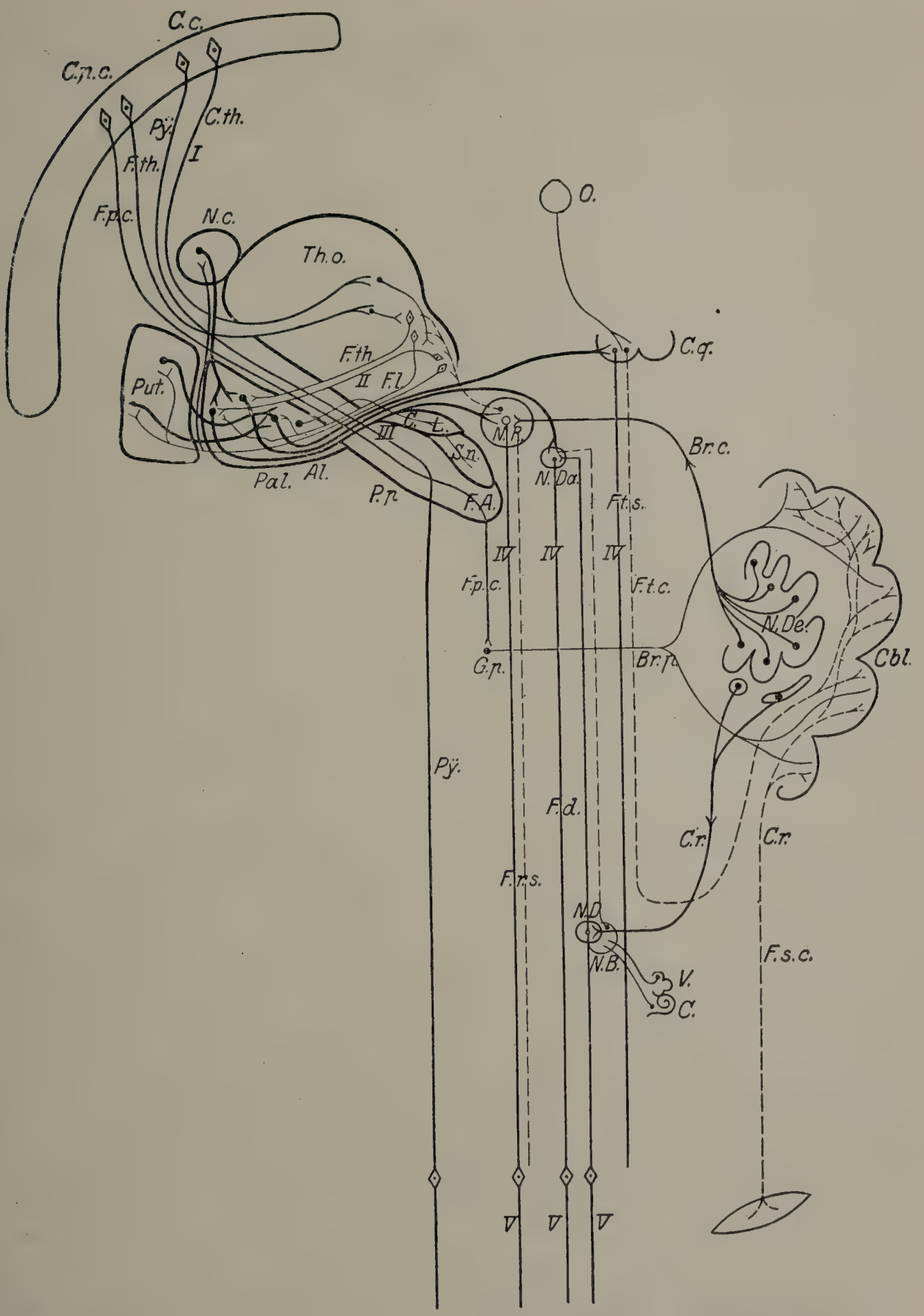


FIG. 328.—Vogt's scheme of lenticular connections. Modified by Foerster. *Cc.* cortex centralis; *C.p.c.*, cortex precentralis; *C.th.*, centro-thalamic tract; *Py.*, pyramidal tract; *F.th.*, fronto-thalamic tract; *F.p.c.*, fronto-ponto-cerebellar tract; *N.c.*, nucleus caudatus; *Put.*, Putnam; *Pal.*, pallidum; *Th.o.*, optic thalamus; *F.th.*, fasciculus thalamicus Foreli; *F.l.*, fasciculus lenticularis Foreli; *A.l.*, ansa lenticularis; *C.L.*, corpus Luysii; *S.n.*, substantia nigra; *P.p.*, pes pedunculi; *F.A.*, fasciculus Arnoldi (fronto-ponto-cerebellar tract); *N.R.*, nucleus Ruber; *N.Da.*, nucleus Darkschewitschi; *C.q.*, corpus quadrigemina. *O.* Oculus; *F.r.s.*, fasciculus rubrospinalis; *F.D.*, fasciculus Darkschewitschi; *F.t.s.*, fasciculus tecto-spinalis; *F.s.c.*, fasciculus spino-cerebellaris; *C.r.*, corpus restiforme; *Cbl.*, cerebellum; *N.De.*, nucleus dentatus; *Br.c.*, brachium conjunctivum; *N.D.*, nucleus Deiters; *N.B.*, nucleus Bechterew; *V.*, vestibularis; *C.*, cochlearis; *G.p.*, Griseum pontis; *Br.p.*, brachium cerebelli ad pontem.

is further here reminded that there are innumerable kaleidoscopic variables and intermediary forms to be found in this large and important group of so-called striatal syndrome.

PARALYSIS AGITANS SYNDROMES.

History.—The clinicians of the times immediately preceding Parkinson grouped the paralysis agitans cases of the present day in several different categories. Galen had noted the characteristic tremor, and the class of tremors of this kind; the "tremblement palpitant" of Preysinger was part of the earlier *palmos* of Galen. Francis de la Böe was shrewd enough to notice the difference, afterward forgotten, between the tremor produced by attempts at motion and the tremors present while the limbs were at rest, and his term *tremor coactus*, for the tremors of paralysis agitans, was utilized up to Parkinson's time. Juncker had also described a paralytic-like tremor, *tremores paralytoidei*, which included some of these patients.

Not only was the tremor appreciated, but the clinicians of the eighteenth century (Gaubius, 1751) had called attention to the propulsion of these patients, and Sauvage grouped them in his choreas, as Scelotrybe precipitée (Danse de St. Guy precipitée, L.).

Parkinson, in his famous thesis on the "Shaking Palsy," London, 1817, made a synthesis of several of these conditions, and erected a new clinical form to which he gave the name shaking palsy (paralysis agitans), and gave the following short and striking description: "Involuntary tremulous motion, with lessened muscular power, with a propensity to bend the trunk forward, and to pass from a walking to a running pace, the senses and the intellect being unimpaired."[1]

All of the case histories cited by Parkinson were type cases of our present-day presenile and senile paralysis agitans syndrome, but the group as then understood still contained certain of the chronic choreas, and certain cases of multiple sclerosis, possibly certain thalamic cases, etc., which late clinicians have agreed to separate. The chronic choreas were definitely excluded by the work of the Germain Sée, 1851, and the researches of B. Cohn,[2] Ordenstein,[3] and Charcot finally separated the multiple sclerosis syndrome.

Within recent years a large medley of closely related syndromes has been described in all of which certain resemblances to the more classical types of Parkinson's syndrome may be recognized. Many have been named; thus the pseudosclerosis of Westphal-Strümpell, double athetosis, certain forms of Huntington's chorea, Wilson's lenticular degeneration, Cecil Vogt's syndrome of the corpus striatum, Bechterew's hemitonia apoplectica, Hunt's syndrome of the globus pallidus, Oppenheim's dystonia musculorum, the Parkinsonian syndrome of epidemic encephalitis, Spiller's family forms of pseudo-

[1] Complete reprint of Parkinson's paper, Am. Arch. Neurol. and Psychiat., June, 1922.
[2] Wien. med. Wchnschr., No. 180.
[3] Thèse de Paris, 1868.

sclerosis, etc.[1] Some of these trends have been sufficiently isolated to permit short descriptions in this work, but the interested student is referred to the bibliographic notes here appended for further investigation into most profitable fields for research.

Charcot's studies, the monographs of Wollenberg, Heimann, Manschot (Winkler) (1904), Zingerle (1910), Mendel (1911), Wilson (1913), Spiller (1916), Hunt (1917), Souques (1920), Lewy (1922) contain the chief steps made, showing the steadily advancing trend to regard the Parkinsonian type not in the light of a functional disturbance, as Charcot taught, but as a closely related group of somatic syndromes, due to the involvement, either circumscribedly or more diffusely of certain cerebellar, thalamic and lenticular pathways, and of certain synapses in the motor and vegetative cells of the globus pallidus and related interbrain and cerebral ganglionic structures.

Presenile and Senile Types. Parkinson's Disease.—We shall here discuss *only* the classical type, *i. e.*, the presenile and senile arteriosclerotic type of Parkinson, again reminding the student that *names* stand for *general categories*, rather than for specific things. The splitting up of this large group is still going on and the details must be consulted in special monographs.[2] The Parkinsonian types of epidemic encephalitis are dealt with in the section upon Epidemic Encephalitis, and other types of Parkinsonism, syphilis, tumor and various toxemias are discussed in their various chapters. Also see Examination methods.

Etiology.—The changes of age seem to be the most striking etiological factors. The majority of the patients are between fifty and seventy. Patients of nineteen, fifteen, twelve, ten, and even three years of age are recorded as having *paralysis agitans*, the vast majority of these Parkinsonian syndromes having resulted from multiple sclerosis encephalitis, syphilis or poliomyelitis. Hereditary factors may play a role, probably through vascular disease. Berger, Gowers, Borgherini, Clerici and Medea and others have reported cases occurring in two generations or in more than one member of the same generation, and Erb reports that in 15 per cent. of his cases the parents or grandparents suffered from the same disease.

Emotional disturbances undoubtedly play a large role in causing the arteriosclerotic foundation. The direct relation of the striatum to emotional activities should be borne in mind.[3]

Trauma may be a sufficiently exciting cause to bring the symptoms of a slumbering paralysis agitans to the surface, or those of a mild case rapidly to a severe stage. In the great war shell shock brought certain paralysis agitans and multiple sclerosis-like tremors to the fore. Many were the results of somatic lesions.

[1] See Hunt, J. R.: **Brain, 1917.** Spiller: Jour. Nerv. and Ment. Dis., 1916, p. 23. Wilson: Brain, **35**, 36. Vogt: Monatschr. f. Psychiat. u. Neurol., 1917, vol. **18**. Westphal: Arch. f. Psychiat., 1913, vol. **51**, and preceding literature.

[2] Wilson: Loc. cit. Hunt: Loc. cit. Tilney: Loc. cit. Lewy, Tonus und Bewegung: Paralysis Agitans in Lewandowsky, Handbuch, 1921. Jakob: Die extrapyramidalen Symptomenkomplex, 1924.

[3] See Kappers: Vogt, in the Heidelberg paper.

Physical stress as advancing an arteriosclerosis may be a factor. Toxic factors play some role. Their coincidental occurrence is frequently reported; the same may be said of infections.

Arteriosclerosis is the chief factor in bringing about the presenile syndrome. The clinical features of the syndrome are matters of localization in the implication of certain pathways by the sclerosing process.

Symptoms.—In the presenile and senile types these develop for the most part very slowly although fulminating and atypical developments are known.

The vast majority of the patients show, on close analysis, prodromal symptoms which are chiefly sensory, in contrast to the sensorimotor symptoms of the more advanced stages of the picture. The more characteristic of these prodromata are fugitive, irregular pains of a sharp, lancinating character, frequently found in the extremities first to be affected by the motor disturbances, and usually ceasing as these latter advance. Paresthesiæ are also frequent, causing sensations of tickling, cold spots, hot spots, gastric distress—almost crisis-like attacks, with diarrhea and colicky disturbances in the large intestine.

General malaise with headache, sweating, mild vertigo, palpitation, sialorrhea, anxiety, pressure of blood in the head, easy excitability, are general symptoms accompanying many senile and presenile conditions, but are so frequently found as forerunners of the motor symptoms, and persist with such marked increase of severity throughout the disorder that their appearance is to be regarded as more than coincidental.

The symptoms of the more classical syndrome may be grouped as follows: (1) The main group of sensorimotor disturbances, varying in intensity and location in different individuals. (2) A number of sensory, vasomotor, trophic and secretory disturbances already indicated as often in part occurring as prodromes. (3) Psychical symptoms which are somewhat variable.

The sensorimotor disturbances are predominantly increase of muscular tonus, with rigidity, and resulting contractures, and motor disturbances with tremor, compulsory gait, forced attitudes, forced movements, and loss of mimetic expression.

An increase in the muscular tonus is a most fundamental feature in the concept paralysis agitans. Accompanying it there follows the rigidity, the mask-like countenance, and the contractures. The increase in the muscle tonus usually is a very early sign, although positive traces of rigidity may not appear until later. It is practically always found, whereas there are some patients who have little or no tremor[1] and yet the name paralysis agitans is properly used. Associated with the hypertonus and the rigidity there is a slowness of movement, and a steadily increasing stiffness, and also retardation of the motor and ideational impulses. Practically all cases of paralysis agitans show akinesis as a primary sign. It is rarely as pronounced as in the encephalitic Parkinsonian types.

[1] Fürstner, et al.; see Zingerle.

The muscular rigidity varies widely in its situation at the beginning. As a rule the symptoms first become manifest on one side of the body, and the symptoms are usually more severe on one side, it may be, for years. In the well-developed syndrome the rigidity involves the muscles of the neck and trunk particularly, the patient assumes the bent-over attitude, such as one naturally assumes when shivering from the cold, and the face is mask-like (corrugators) and staring, the eye muscles also sharing in the rigidity, with Stelwag's sign.

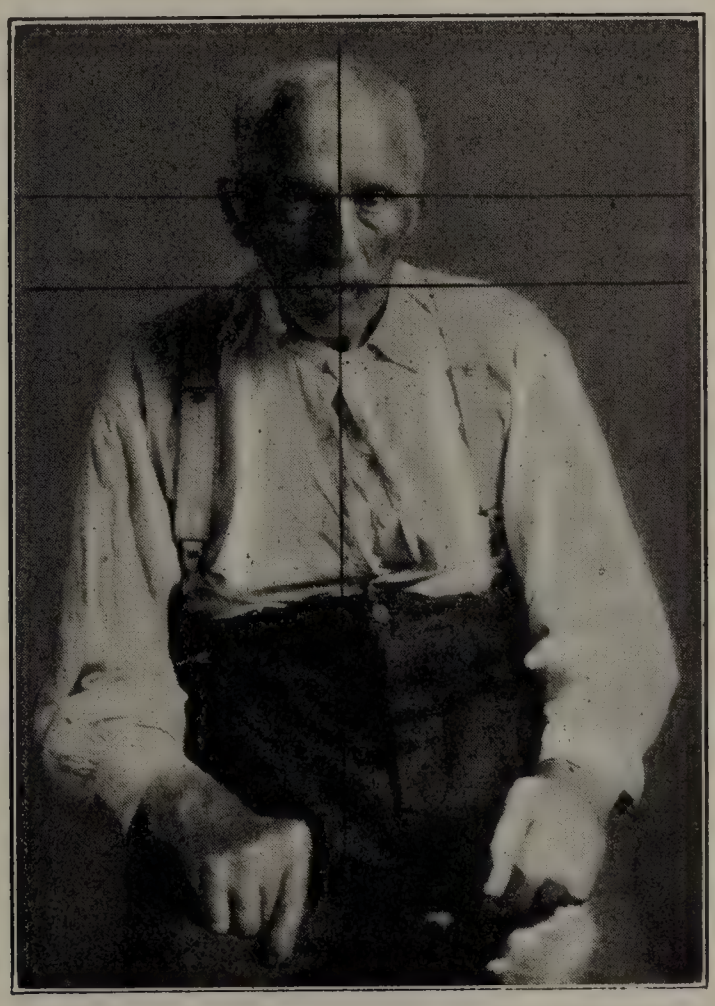


FIG. 329.—Attitude of paralysis agitans patient. (Tilney.)

Whereas the muscles of the neck and back are most affected, almost any group of muscles may be involved. The arms and legs are almost always implicated, and so also are the muscles of the face. Occasionally there is ptosis, or the patients open their eyes with difficulty after closing them. One of us has seen this as an initial symptom. The patients read with difficulty because of the stiffness of the ocular movements. Ocular palsies may result—pseudo ophthalmoplegias. Slow pupillary reactions are occasionally found. The pharyngeal and laryngeal muscles being involved, as others in the body, results in slow, difficult speech, becoming fainter and fainter as the years go by, until finally the patient, in addition to being unable to move, to dress himself, eat without help, finally is unable to talk or to swallow.

The hypertonus and rigidity, however, is not assoc ated with the usual increased reflex signs of pyramidal tract involvement, the reflexes are either normal, or only slightly exaggerated, no clonus, Babinski, Oppenheim, etc., and the contractures may be easily over-

come by passive movements, in marked contrast to the contractures of psychomotor cortical origin. The muscular power is also not so involved, the patients show muscular weakness, but not paralysis. There is a striking contrast between the strength of active movements and that of resistance movements. The former are weak, the latter rarely less than normal. Walshe[1] has shown that intramuscular injections of novocaine abolishes the rigidity, leaving voluntary power unaffected. Tremor is unaffected.

A few cases of extensor rigidity are recorded, but the involvement is predominantly of the flexor muscles.

With the rigidity there is a feeling of tension which the patients dislike usually much more than the almost universal tremor. This causes them to feel as though they were bound. Their motor impulse seems interfered with. This may even involve their urination and defecation, and their deglutition.

Attitude.—These patients show a definite defensive attitude largely conditioned by the interference in the associated movements and the postural and tonus alterations. Here the fundamental righting reflexes (stell reflex of Magnus and de Kleijn) are involved, showing even the absence of hypertonia or rigidity. Adiadocokinesia is a frequent early sign (Zingerle[2]), the speech disturbances and micrographia being attributed to it by some observers.

Tremor.—In the majority of the cases this objective sign seems to be the first, although, in reality, vasomotor signs precede as a rule. It was the symptom first noted by Galen. It is characterized by its uniformity and steadily increasing severity, both in point of advance of the disease, and also during the movements themselves. It is a tremor that Franciscus Sylvius first noted was present while the limb was at rest. It ceases during movement, especially if the movement is rapid, and in the beginning of the disease. In the later stages it becomes continuous, and some patients with paralysis agitans show some intention tremor. Again, a certain number of patients show little or no tremor.

The tremor is characterized by the uniformity of its excursions, which are at first small, slow, and rhythmical. They average about three to five in a second, according to the muscles involved. Tremor in the muscles of the thumb, which is often an early sign, gives rise to the well-known "pill-rolling" movements. Similarly, one has the movement of "beating the drum," and other attitude types when the larger muscles of the arms are implicated. The muscles involved in the early tremors vary considerably; usually the upper extremity is involved before the lower, the hand particularly; but with the progress of the disease the tremor tends to become widespread, almost universal; most diverse localizations are on record, anything is to be expected. There is no great preponderance of one hand over the other, although there is a marked tendency to disproportion in the severity,

[1] Brain, 1924, **47**, 159.

[2] Jour. Psychiat. and Neurol., 1922, **27**, 152.

and a hemiplegic type of onset and persistence is frequent, if not characteristic. Monolimbic types are encountered. Micrographia is frequent.[1]

At first the tremor is absent during sleep, but in the advanced stages it frequently persists and constitutes one of the factors in sleeplessness which finally exhausts the patient. de Jongh has demonstrated that it is an *action* tremor. It is not affected by muscle injections of novocaine and hence is not, as Jackson claimed, "rigidity spread out thin." It is not a proprioceptive reflex reaction. (Walshe.)

Motion tends to diminish the tremor, as also does attention; emotional disturbances and cold increase it markedly. Grasping the tremulous member, touching it, or changing its position results in a temporary cessation of the tremor. The restlessness of these patients is largely dependent upon the constant and continual shifting of the body, in order to obtain comfort, *i. e.*, before they become too rigid—akathisia (Haskovec).

Statistical studies show that tremor may be absent in as high as 20 per cent. of the cases—a much greater proportion than show absence of rigidity, which also may be absent or scarcely noticeable. These are variations, in precise pathway-blocking, the lesions showing slight variability in localization.

Disturbances of Equilibrium.—Propulsion, which apparently was first noted by Gaubius (1751), is one of the cardinal symptoms in Parkinson's original definition. The patient on walking tends to fall forward, and in his effort to keep his equilibrium goes faster and faster, until he either falls or stops himself by grasping a support. Lateropulsion and retropulsion are also present. These are due either to the stiffness with the slowness of muscular movement, or to a central disturbance of equilibrium, which latter is perhaps the preferable explanation, since exquisite examples of gait disturbances are known without any marked stiffness or rigidity. In a few cases the loss of equilibrium in one direction alters to that of another during the course of the disease.

Secretory, Vasomotor, Trophic Symptoms of the Vegetative Level.—These make up the second category of symptoms almost universally found to a greater or less degree in paralysis agitans. As noted, many are prodromal symptoms. What relation these symptoms, all of which have some definite relation to the vegetative nervous integrations, probably at thalamic, striatum and hypothalamic levels, bear to the almost universally present arteriosclerosis is not yet clearly analyzed.

The most important of the secretory changes are increased perspiration—sometimes unilateral, increased salivation—one of the most distressing of the symptoms—and polyuria, with occasionally diarrhea. Changes in pancreatic and liver function may also be observed.[2]

Among the vasomotor changes are rushes of blood to the head,

[1] *Micrographia:* See Pick (1903), Bernhard, Bing, Foerster (loc. cit.), Wilson (Croonian Lectures, Lancet, July 4, 1925. *Akathisia:* See Wilson.

[2] Lewy: Zeit. f. d. exp. Med., 1922, **26**, 87–95.

reddening of the face, cyanosis, tachycardia, acroparesthesia, with hot and cold spots, alteration of temperature—sometimes unilaterally disposed, and dermographia, which is almost constant.

Trophic changes in the skin, such as atrophy, thickening, edema, are among the rarer findings.

Psychic Disturbances.—Depression, anxiety, ideas of self-destruction, scornfulness, savage raillery, sarcastic pessimism, euphoric compensation and sublimation, resignation to the will of God, etc., these are but a few of the innumerable attitudes which these patients show at one or another time during their long period of almost unbearable suffering.

Parkinson said the "senses and the intellects are not impaired," and intelligence tests bear this out. In some patients one naturally finds a senile deterioration, and for most, in the later stages, the mental signs of an arteriosclerotic deteriorating process are present. Acute exhaustion, delirious states, often close the sad chapter; but these are not a part of the paralysis agitans.

Sensory Symptoms.—Tactile, thermal or pain disturbances are not definite. They are not often striking features of the disorder. The early pains are usually fugitive, and apart from the dull and most oppressive sensation due to the tension and stiffness, pain is not prominent. Irregular anesthesiæ, hyperesthesiæ, paresthesiæ are frequently found, but are so inconstant that one can say that definite sensory disturbances rarely belong to the paralysis agitans picture. When present in striking fashion they are probably due to lenticular or thalamic involvement of deep sensibility pathways.

The *reflexes* are not markedly disturbed. Considerable variation exists, but there is no constant picture as yet known which is pathognomonic of the condition. Reflex activities due to pyramidal tract involvement are occasionally found. Increased knee-jerks, clonus, and Babinski phenomena are at times found, but they are not constant, and represent occasional rather than essential features.

Loss of the Achilles reflex is a not infrequent symptom the significance of which is as yet not definitely placed.

The abdominal, cremasteric, epigastric, and anal reflexes are not involved.

Other clinical findings are inconstant. The blood practically shows nothing, some anemia at times, but nothing striking; the cerebrospinal fluid is practically negative. The urine, apart from a frequent polyuria, shows no quantitative or qualitative anomalies, beyond the excessive phosphate elimination, which is indicative of the exhaustion.

Course and Progress.—Rudimentary forms are not unknown. Many senile patients show conditions closely approaching the milder grades of paralysis agitans, and intermediary stages, with muscular stiffness, slowness of movement, retardation of motor impulses, etc., are not infrequent. Certain stationary cases, non-progressing for twenty-five years, are also known, and infrequently patients make partial recoveries. These are possibly syphilitic cases showing the syndrome.

But the usual course of the more frequent arteriosclerotic cases is a long, slow, and gradual progression, lasting over many years with annoyance, inconvenience, discomfort, distress, and agony until life becomes a burden. Remissions and exacerbations belong to almost every case.

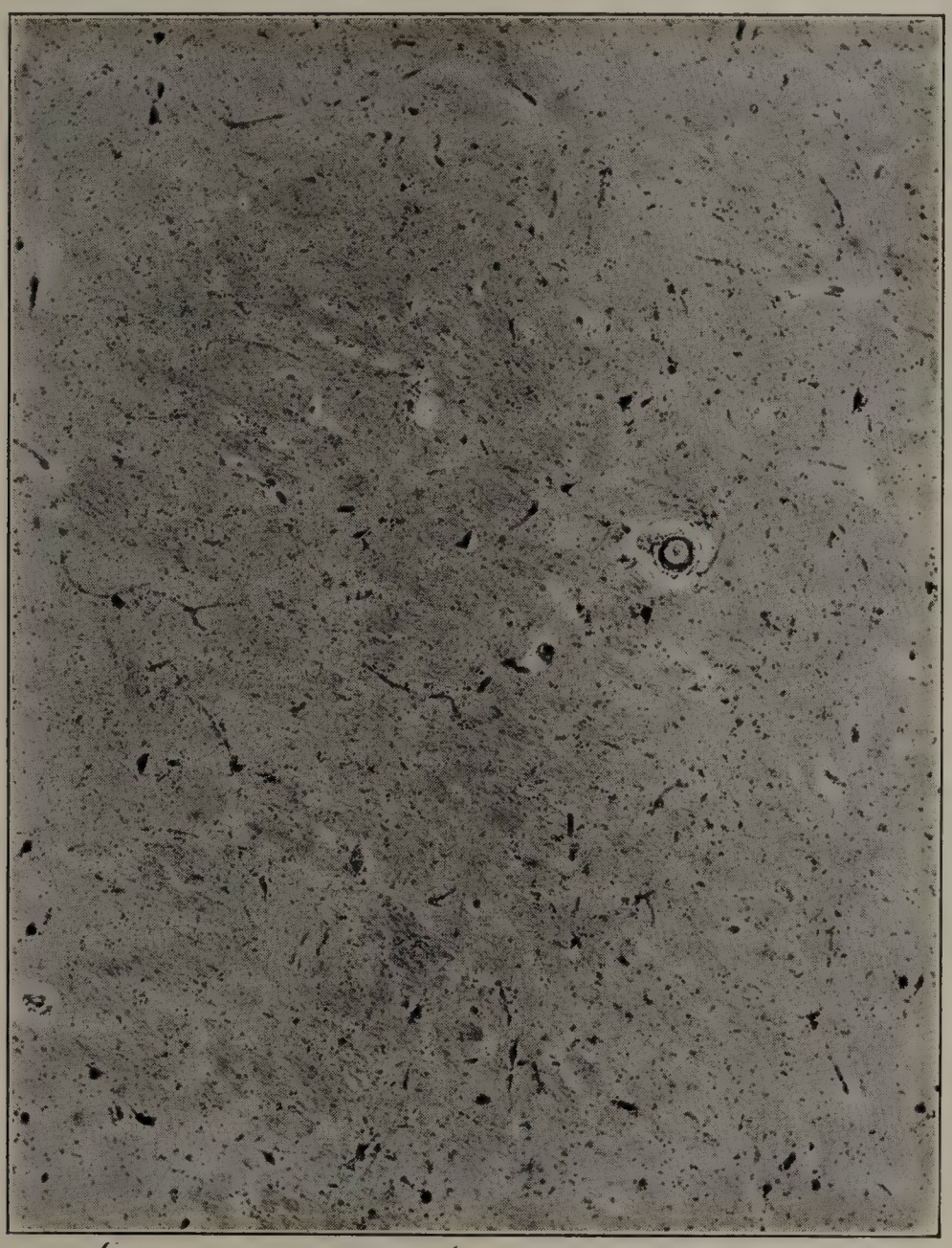


FIG. 330.—Primary atrophy of the pallidal system. Progressive atrophy of the globus pallidus. Juvenile paralysis agitans. Section through the globus pallidus, showing atrophy and diminution of the large motor cells of the globus pallidus, with increase of glia nuclei. (Ramsay Hunt.)

Patients with little hypertonus and muscular rigidity seem to progress less rapidly, and many cases beginning in younger individuals do fairly well. Emotional disturbances act badly, almost invariably causing marked progress of the disease.

The majority of the patients get worse gradually. The increasing weakness and stiffness limits them more and more in their work, until that becomes impossible. Then the walking, to which they are unconsciously attracted, becomes more and more difficult. They are then confined to their chairs for a few years, or to bed, and finally come to be helpless invalids, who must be fed, turned over, attended to like

living rigid statues, which in the presence of relatively intact intelligence, though robbed of the power of expression, reading, writing, even pantomime, constitutes one of the most ghastly afflictions in the entire realm of nervous disorders.

Decubitus, pneumonia, exhaustion delirium, and starvation, are the usual precursors to the end of a disorder whose prognosis is bad. Recoveries in this stage there are none, although stationary cases are occasionally seen.

Differential Diagnosis.—The diagnosis of the classical Parkinsonian type is rarely difficult. The attitude, gait, facial expression, and tremor are so characteristic as to stamp the patient at once. In the initial period, before the stiffness, tremor, pulsions, etc., have developed, the diagnosis may be difficult, but after its classical development it cannot be mistaken for anything else.

Hysteria sometimes comes into review, but here the character of the hypertonus is quite different, the tremor is rarely classical, and can be more readily influenced by diversion and distraction. The exaggeration of a paralysis agitans symptom-picture is characteristic of the hysterical type.

Syphilis, tumor, hemorrhage in the basal ganglia at times produce the syndrome.

Multiple sclerosis of the cerebellar type frequently shows the classical paralysis agitans picture, plus the evidence of pyramidal tract involvement, nystagmus, bulbar speech, etc., of this disease. It is usually present in younger individuals. Some of the so-called juvenile Parkinson cases are cerebellar types of multiple sclerosis.

The chief development of recent years have been toward the separation of other types of the syndrome as an expression of an involvement of the globus pallidus (Hunt), Cecile Vogt's syndrome, characterized by a spastic diplegia with pseudobular palsy and athetosis, with necrotic lesions in the caudate nucleus and putamen; Wilson's lenticular degeneration and various admixtures of these syndromes. Postencephalitic Parkinsonian syndromes were extremely common following the 1917–1922 epidemic of encephalitis. Their discussion opens up a large chapter bearing on the distribution, localization and dynamic interrelationships of the grouping of the sensory, motor and vegetative pathways in the basal ganglionic synaptic junctions. This is too detailed and complicated to be entered into here. (See Encephalitis.)

Pathology and Pathogeny.—A dogmatic presentation of the causes for paralysis agitans is not yet available. The trend of opinion is that it represents a senile or presenile degeneration of certain cerebellar, thalamic, and lenticular mechanisms. Whether they are confined to the basal ganglia regions is not proved—neither are these mechanisms—but the evidence points in that direction. The rigidity, attitude, slowness of motor impulses, tremor, has its analogies in disorders of the fronto-cerebellar, cerebello-rubo-spinal and thalamic systems. Hunt[1] would localize this motor part of the syndrome to

[1] Hunt, J. R.: Loc. cit.

disease of motor cells in the globus pallidus, a view closely related to that of Winkler eleborated in Manschot's thesis (1904). The disturbances of equilibrium are suggestive of the cerebellar type. Thus the automatic proprioceptive tonus impulses passing over impaired cerebellar pathways are not counterbalanced by the influence of the cortico-medullary pyramidal systems. Because of the lesions in the globus pallidus with the degenerations in the ansa lenticularis, there are not enough centripetal cortex impulses to act. The rigidity, forced attitude of the head and trunk possibly may be so interpreted. The mechanisms of the righting reflexes (Magnus, de Kleijn, Rademaker) are involved.

The vasomotor, secretory and trophic symptoms represent central vegetative disorders, which are referred with greatest probability to interbrain nuclei, the hypothalamus, corpus Luysii, substantia nigra, etc. Thus Dresel and Lewy[1] have shown that both the glycogenic and hepatic functions are interfered with in paralysis agitans possibly from lesions of the nucleus paraventricularis hypothalamici, thus interfering with the pancreatic pathway and integrations. (See Vegetative Neurology.)

The important reviews of Zingerle, Jelgersma,[2] Hunt,[3] the Vogts,[4] Foerster, Souques, Magnus, Kleist, Pollak, Boestroem and others (loc. cit.) brings these features to the fore. Few complete series of microscopical sections through the cerebellum and midbrain region have as yet been studied. Loss of motor cells in the globus pallidus and distinct atrophies in the ansa lenticularis, in thalamic and lenticulr regions are present in those cases studied by the serial section method. Tretiakoff (1919) described changes in the locus niger and Winther (1921) has found lesions in the internal nucleus of the thalamus.

The muscular hypotheses,[5] which are many, are totally inadequate, as is also the parathyroid hypersecretion hypothesis advanced by Lundborg. There are a number of pathological processes operative. Arteriosclerosis is the chief factor thus far observed in the type here considered.

Therapy.—Notwithstanding the gloomy outlook, much can be done to relieve the patients. They must be guarded against cold, and as far as possible, from emotional disturbances and mental and physical strain.

They should live in warm, dry, sunny rooms if possible, be much in the open air, eat what they like and avoid hypochondriacal making treatment charts of all kinds and descriptions. Such, we are convinced, are devices of commercially, rather than humanistically minded

[1] Dresel and Lewy: Ztschr. f. d. ges. exp. Med., 1922, **26**, 95. Lewy in Lewandowsky's Handbuch, and Tonus und Bewegung. Also see Hess and Goldstein: Ueber viscerale Innervationsstörungen bei Parkinsonismus, Wien. klin. Wchnschr., 1926, **39**, 1181; Wechscler and Brock: Righting Reflexes in Paralysis Agitans, loc. cit.

[2] Verhandl. d. Gesellsch. deutsch. Naturfschr., 1908, **80**, 383.

[3] Hunt, J. R.: Brain, 1917, complete bibliography.

[4] Vogt, C. and O: Loc. cit. Kleist, P. A.: Stammganglien u. Mittelhirn, Deutsch. med. Wchnschr., 1925, No. 51.

[5] Camp: Jour. Am. Med. Assn., 1910.

physicians. Alcohol and coffee may be used as common sense dictates. Tobacco is not necessarily taboo. The regimen should under no circumstances be so strictly adhered to as to cause the patients, already suffering from irritating conditions, to become further annoyed thereby. Diet has relatively little power to modify the trouble, and fussy dietaries are for the most part superstitious nonsense.

The greatest relief from rigidity comes from the regular use of passive movements. The Zander apparatus can be utilized. Working with carpentry or garden tools is often very helpful. Heat is grateful and helpful and passive motion combined with warm (not hot) baths is particularly gratifying. A few patients react badly to baths. Charcot used to advise his patients to ride in the shaky railroad cars of his period. Automobile riding is much enjoyed by many patients.

Such attempts at occupation therapy must be carefully dosed. Fatigue must be avoided. Five to ten minutes is sufficient in the early stages. Such therapy is solely of value from a psychical standpoint.

Drug therapy is at times of doubtful service in controlling the tremor. The remedies are those with marked action on overaffective autonomic stimuli—notably the alkaloids of the belladonna group, of which hyoscine, duboisine, scopolamine and atropine are the most available. In view of the chemical uncertainties concerning the alkaloids of this group one should obtain good products and try the different derivatives. The dosage must be tested with each case. Bulbocapneine and related alkaloids[1] are of no permanent service.

The analgesics, particularly in combination with salicylates, are useful in relieving the muscular soreness and pain of tension—phenacetin, aspirin, acetanilid combinations, etc.

For sleep, the best form of hypnotic is not yet known. Bromides are at times available, at other times the alcohol hypnotics—trional, sulphonal, again urea substitutes, as veronal—are useful. One should avoid morphine as much as possible, particularly bearing in mind that the emotional hyperactivity may have little real feeling behind it. It is often mostly mimicry which is uncontrollable because of the motor defect.

Some of the postencephalitic Parkinsonian syndromes recover spontaneously. (See Encephalitis.) Salvarsan has helped many of these (arsenic).

In the beginning of many a presenile or senile Parkinsonian syndrome an enlightened psychotherapy may be of signal service, especially in regulating faulty emotional reactions. As de Jongh in the analysis of the tremor has shown an *action* component, these patients, being *hindered*, are often *inwardly* (unconsciously) consumed with anger. This tends to augment the vascular alterations (affect reaction) and increase the somatic disintegration. Efforts which will show the patient a better philosophy of self control (relaxation, Jacobson) are often surprisingly effective in quieting the tremor, giving rest and sleep.

[1] Fraenkel: Arznermittelsynthese, 6th ed., 1927. de Jongh: Loc. cit.

HEPATOLENTICULAR DEGENERATION: WILSON'S DISEASE: WESTPHAL–STRÜMPELL PSEUDOSCLEROSIS. DYSTONIA MUSCULORUM.

In a fully documented monograph Hall,[1] of Copenhagen, has offered a synthesis of two syndromes, Wilson's lenticular degeneration and the Westphal-Strümpell types of pseudosclerosis under the title Hepato-lenticular Degeneration. Although there are considerable differences in these two types, both clinically as well as anatomically, Hall believes these differences to be solely quantitative rather than qualitative. Just as multiple sclerosis is well known to present a great variety of clinical pictures due to the different localizations of the plaques, so the types here brought together by Hall may show a variability and yet be essentially one form of disorder.

Hall is also inclined to bring within the group the torsion spasms of Schwalbe- (1908) Ziehen (1910) to which Oppenheim (1911) gave the designation dystonia musculorum deformans, which seems justified especially when Thomalla in one of Flatau and Sterling's cases of torsion spasm, and Wimmer and Neel[2] also report the liver changes thought to be pathognomonic of Wilson's disease.

This synthesis has received considerable confirmation, although, as already stated, it is thus far premature to establish too closely delimited disease types for disorders in a region so complex as that of the nervous system at the striatum level.

It seems fairly certain that the Westphal-Strümpell pseudoscleroses were too illy defined and covered too many diffuse and diverse conditions. The tendency at the present time is to disregard this term entirely. The so-called pathognomonic sign of a greenish cornea pigmentation may be found in the entire group and hence loses its specificity.

In previous editions of this text-book the lenticular degeneration of Wilson and the torsion spasm of Ziehen-Oppenheim were given separate descriptions, but the synthesis of Hall, Mahaim and others is preferred.

History.—Frerichs, in 1858, described the earliest (?) case of hepatolenticular degeneration. He discussed it as a disease of the liver and missed the striatal pathology. The case was that of a boy, aged ten years, who developed dysarthria, dysphagia, tremors and weakness of the limbs and marked motor disturbance, slowness, without paralyses. Gowers, in 1888, described a *tetanoid chorea* which covered most of the classical signs of hepatolenticular degeneration, but the elaboration of one of the outstanding types was due to Kinnier Wilson, of London, who in 1912 brought together (1908–1912) a complete monographic survey of 12 cases and established this syndrome on a firm descriptive basis. Fleischer (1912) described independently analogous

[1] La Degenerescence Hepato-lenticulaire, Paris, Masson, 1921; English translation, 1922.

[2] Hospitalstidende, July, 1921.

cases, emphasizing the liver pathology rather than that of the striatum. It is of historical interest that the dystonia concept was being elaborated in Germany, as Schwalbe (1908) first described 3 cases from the Charité service of Ziehen, who in 1910, called the syndrome torsion spasm. Shortly therafter Oppenheim proposed the name dystonia musculorum deformans.

The complete historical summary is available in Hall's monograph, where it may be seen that the author is inclined to maintain two types, *Wilson's Disease* and *Dystonia Musculorum*, with a series of intermediary and intermingling forms, sometimes described as one, sometimes as another, with pseudosclerosis as an indefinite third. Hall submits 68 of all these types to an analysis. Here one cannot enter into all of the details.

Wilson's Disease, Lenticular Degeneration Type.[1]—This is usually a fatal form, although Curschmann has described benign cases.

It is familial, in the sense that more than one member of a family may be affected. Direct hereditary factors (recessive) are usual. It may occur sporadically. It has been observed chiefly in young people, either in an acute or a chronic form. It is progressive, its duration ranging from six months or a year to as long as four or five years in Wilson's cases; in others longer.

Symptoms.—The clinical symptoms consist of involuntary movements, nearly always a bilateral tremor of both upper and lower extremities, the head and trunk also being sometimes involved. The tremor is usually rhythmical but occasionally irregular, and increasing with volitional movement, *i. e.*, of a paralysis agitans type, but may be absent (v. Economo) or be coarse and athetotic (Thomalla). There is often pronounced rigidity of the limbs and of the face, the latter being usually set in a spastic smile, while in the later stages contractures of the limbs develop; there is dysphagia and dysarthria, the latter eventually degenerating into complete anarthria; there is also spasmodic laughing and emotionalism. Articulation may be difficult, the mouth is frequently held open and salivation is pronounced at stages. As a result of the extraordinary degree of stiffness of the musculature there is considerable difficulty in maintaining equilibrium. Little or no true paresis or paralysis occurs, however, inasmuch as most ordinary movements can be executed, although it may be slowly and feebly. In spite of the great degree of motor weakness and helplessness, in a pure case, the abdominal reflexes are present and a double flexor response is obtained. Corneal pigmentation may be present as a 2-mm. wide greenish-brown haze at the periphery (Fleischer, 1903; Spielmeyer, 1920). It is accompanied by a hypertrophic cirrhosis of the liver, often latent (Barnes, 1924).

The chief clinical features rapidly summarized then are: (1) Mus-

[1] Progressive Lenticular Degeneration: A Familial Nervous Disease Associated with Cirrhosis of the Liver, Brain, 1913. Lewandowsky's Handbuch. Tilney: Neurol. Bull., 1918. Hall: Hepato-lenticular Degeneration, 1925; French edition, 1921. Wilson: Croonian Lecture: Disorders of Motility, Lancet, July 4, 1925, *et seq.*

cle hypertonicity; (2) more or less regular rhythmic tremor, increased by effort; (3) dysarthyria and dysphagia; (4) hypertrophic cirrhosis of the liver; (5) absence of pyramidal tract involvement; (6) mental changes usually develop.

Pathology. — The chief neuropathological feature of the syndrome is a bilateral degeneration of the putamen and globus pallidus, in particular the former. Of the 30 cases examined between 1912 and 1925, Walshe states 16 presented softening and cavitation of the lenticular nuclei. In 14 these were shrunken and microscopically showed cellular alterations. Cortical changes were present in 9, and there were lesions also of the red nucleus, in the dentate nucleus, and superior cerebellar peduncles. Wilson and Bielschowsky, on the one hand, hold that the cellular changes of pseudosclerosis (Alzheimer) are different from those of lenticular degeneration, whereas Spielmeyer, from 5 cases, states they are identical, even if varying in degree. A constant essential, and in all probability primary feature of the pathology of the syndrome is hypertrophy of the liver, not syphilitic or alcoholic; it is multilobular or mixed in types, always pronounced, but presenting a varying pathological picture of necrosis, fatty degeneration and regeneration.



FIG. 331.—Progressive lenticular degeneration. (Tilney.)

As yet correlations between the liver and the striatum changes are speculative. It is certain that a double situation is possible. That liver disease may induce central nervous changes localized in the striatum is to be experimentally proven. Some investigations seem to show it. On the other hand central nervous disease may cause isolated and characteristic organ disorders. This is notoriously true for certain psychogenic organ disturbances and the recent trend is to show that somatic disease is dependent upon nervous system disorder. Dresel and Lewy's observations on disturbed hepatic function in

paralysis agitans (loc. cit.), as well as a series of related observations, offer light upon this hypothesis.[1] The synapses of the higher vegetative (motor) pathways are located in part in the corpora striata, thalamus and hypothalmic regions. The analysis of these pathways is still in its infancy. (See discussion of vegetative pathways in section on Visceral Neurology.) This syndrome as well as that of paralysis agitans and related interbrain and basal ganglia syndromes offers excellent material for the analysis of these higher vegetative pathways and synapses.[2]

Dystonia Musculorum Deformans Syndromes.[3]—Under this term Oppenheim described a peculiar syndrome first called attention to by Ziehen and Schwalbe, as a tonic torsion neurosis. Flatau and Sterling termed it a progressive torsion spasm. It is preëminently a disorder of children, most of the observed patients having been between eight and fourteen years, and almost all of the Jewish race. Apparently there are no sex differences. Oppenheim's cases did not lead him to any light on the disorder as regards heredity. Three of Ziehen's cases were brothers and sisters. Exciting or other causative factors are not known. Some anomalous cases have followed epidemic encephalitis. Transitional types (Rosenthal), allied to double athetosis, Westphal-Strümpell pseudosclerosis, Wilson's lenticular degeneration, occur.

The illness comes on apparently gradually and subtly in one arm or in both arms; occasionally first in the legs or in the spine, but in progression. The patient twists the spine in a peculiar fashion, tilting the pelvis, and bringing about a marked torsion of the entire vertebral axis, with lordosis, scoliosis, and tilting of the pelvis, the arms and legs moving in a peculiar manner. The mode of progression at times resembles that of astasia abasia. The whole musculature, when in action, is extremely stiff and hypertonic; when at rest, hypotonia is apparent. In the general attitude of the patient in walking one is inclined to regard the whole matter as one of extreme suggestibility; a psychogenic-hysteriform affair. The movements of the hip are very typical. It is tilted or thrust forward or backward in an awkward manner. Oppenheim has likened it to a dromedary in some of the positions assumed. Walking seems to fatigue the patients greatly. They perspire, get red, and show signs of fatigue, getting out of breath, and one of our (J.) patients grunted involuntarily. One of Oppen-

[1] See Schneider's Study of the Liver in Thomalla's Case of Dystonia with Hypertrophic Cirrhosis, Ztschr. f. d. ges. Neurol. u. Psychiat., 1920, **53**, 289. Thomalla: Ibid. Also Boenheim, F.: Ztschr. f. d. ges. Neurol. u. Psychiat., 1920, **60**, 10. Hess and Goldstein: Wien. klin. Wchnschr., 1926, **39**, 1181. Stevenen and Ferrara: Riforma Med., 1924, **49**, 3, 54. Ferraro, A.: Arch. d. pat. e clin. med., 1924, vol. **3**.

[2] See Malone: Studies on the Vegetative Synapses of the Diencephalon. Kraus: Klinische Syszioologie, vol. **2**, pp. 1922, 1926. Brugsch and Lewy: Biologie der Person, 1926.

[3] Oppenheim: Neurol. Centralbl., 1911, No. 19. Ziehen: Neurol. Centralbl., 1911. Schwalbe reported on by Maas eight years later: Neurol. Centralbl., 1918. Wechsler-Brock: Arch. Neurol. and Psychiat., 1922, vol. **8**. Frankel and Abrahamson: Jour. Nerv. and Ment. Dis., 1912. Flatau and Sterling: Ztschr. f. d. ges. Neurol. u. Psychiat., vol. **7**. Rosenthal: Arch. f. Psychiat., 1922, **66**, 445; 1923, **68**, 1. Mendel: Monatschr. f. Psychiat. u. Neurol., 1919, vol. **11**; monograph with literature to 1919.

heim's patients could walk backward better than he could walk forward. On sitting down or lying down the movements cease (Beling's case), or are much reduced in frequency and in clownishness. Ziehen's cases were at times continually in motion, and had to be kept in a special bed, against which they frequently bruised themselves by their impulsive movements. The peculiar activities come into play as soon as there is an attempt to make any voluntary movement. Writing becomes difficult or impossible. There is no paralysis. Oppenheim speaks of a dystonia, Ziehen of a hypertonia. The movements are not athetoid nor choreic. They are wide, irregular, and yet partake of the nature of both, and at times resemble those of Huntington's chorea.

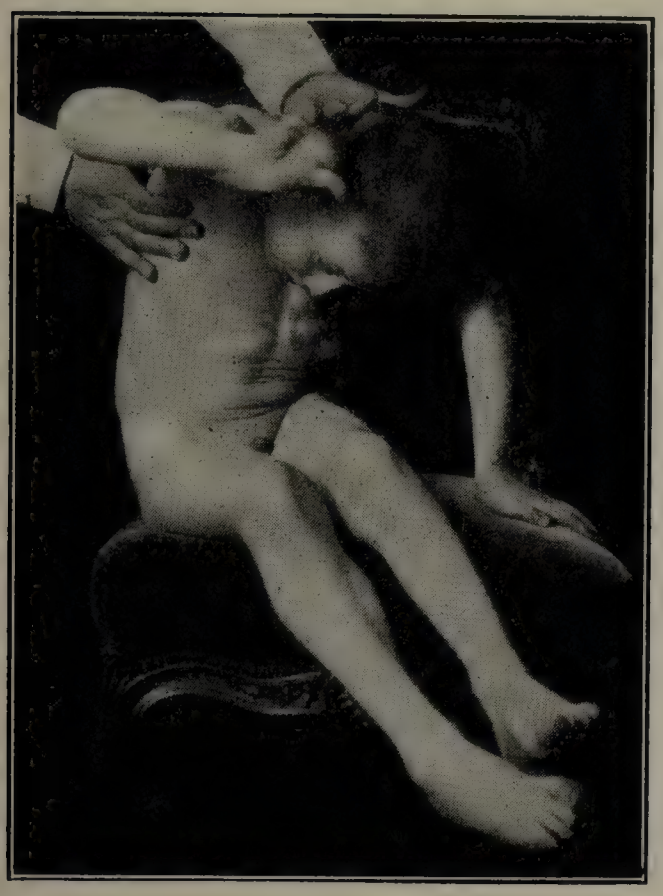


FIG. 332



FIG. 333

FIGS. 332 and 333.—Attitudes in dystonia musculorum cases. (Flatau.)

Tonic and clonic extension of the muscles, particularly of the biceps and rotators of the thigh, were marked in Oppenheim's cases. Thus there is an alternation in tonicity of the muscle. Abrahamson has also reported three cases in one family, each at different stages of the syndrome.

The knee-jerks are apt to be much diminished, coming out in Oppenheim's cases only by Jendrassik reinforcement.

The relationships are difficult to state. Hysteria should be excluded. Double infantile athetosis shows a similar picture. These cases have been studied especially by Lewandowsky.[1] Cecile Vogt and Oppen-

[1] Deutsche Zeitsch. f. Nervenheilk., xxxix, 1908.

heim have reported on infantile pseudobulbar palsy, which also is to be considered in this connection.[1] Transitional and related dystonias are frequent in epidemic encephalitis. The patients apparently hold their own for some time, and but little is known of the development of this type save that no recoveries are reported. Maas reported eight to ten years later upon one of Ziehen's cases, on whom an orthopedic operation had been performed. The movements were still in evidence but were much ameliorated in severity.

Thomalla[2] has reported an autopsy on one of these cases in which the pathological alterations were similar to those of Wilson's lenticular degeneration. This patient also had the liver changes characteristic of the Wilson cases (Schneider, loc. cit.). From this it may be seen that transition forms characterize the syndromy (C. and O. Vogt).

No known method of treatment seems useful. Suggestion is of no value, nor are bromides. The movements cease during sleep.

THE CHOREAS.

The somatic choreas are due to definite but usually recoverable brain conditions, chiefly located in and about the cerebral and cerebellar cortex and the static motor pathways. Meningitides, encephalitides, new growths, severe concussions, hemorrhages, toxemias, and marked fatigues, etc., are among the causative factors. They are a vast conglomeration of conditions, certain trends of which have been separated out under a variety of types.

The detailed history of this sorting process would lead too far. The beginning of the present-day groupings are to be found in the classic of Germain Sée (1850). The chief trends center about the study of the movements which are present. These are of three main forms: (*a*) spontaneous movements, (*b*) coördination disturbances and (*c*) disturbances of purposeful movements. In most choreas the three forms are present but in varying proportions.

The chief nosological entities which have been created are:

1. *Chorea Minor or Sydenham's Chorea.*—The most widespread and frequent of the trends, usually found in children or young adults, resulting from various infections and usually recoverable. They are erroneously spoken of as functional.

2. *Chorea Chronica.*—A stationary form of the former, or when occurring in old age, chorea senilis.

3. *Chorea Huntingtonii.*—A chronic progressive type with certain definite hereditary factors and one showing a vast variety of other choreic anomalies in the "non-Huntington chorea" members.

4. *Chorea degenerans of Brissaud,* occurring as a result of presenile breakdown in unstable neuropathic individuals.

[1] Jour. f. Neurol., 1911, vol. **18**. Rosenthal: Torsionsdystonia and Athetosis, Double, Arch. f. Psychiat., 1923, **68**, 1.

[2] Thomalla: Ztschr. f. d. ges. Neurol. u. Psychiat., 1917, **41**, 311.

5. *Chorea electrica of Dubini,* an acute, usually fatal disturbance, often occurring with epileptiform attacks, paralysis and death. They are not improbably forms of polioencephalomyelitis, of striatal localization and have been observed in the lethargic encephalitis epidemic.[1]

6. *Chorea electrica of Bergeron and Henoch,* occurring in young persons, seven to fourteen years, with rhythmic lightning-like movements of the neck, shoulders, and upper arms. It has allies in certain epileptic-like choreas.

7. *Chorea epileptica,* continuous with the preceding or related to cortical epilepsies. (See Epilepsy.)

8. *Choreas of general paresis,* in which spontaneous choreiform movements occur. (See Paresis.)

9. *Choreas of many Psychoses.*—Motility psychoses of Wernicke, chiefly schizophrenic individuals in whom Kleist has endeavored to show an involvement of the cerebello-rubro-cortical tracts.

10. *Choreas of Congenital or Infantile Cerebral Palsies.*—(See Hemiplegia, Thalamic Syndrome.)

11. *Chorea Postapoplectica. Posthemiplegic Choreas.*—(See Hemiplegia).

12. *Chorea Striata and Thalamica.*—(See Thalamic Syndrome, and chiefly in Epidemic Encephalitis.)

13. *Choreas due to Disorder Involving the Superior Cerebellar Peduncles in Friedreich's Syndrome and Other Cerebellar Pathway Involvements.*—(See Cerebellar Syndromes.)

14. *Choreas of Cerebellar Origins.*—(See Cerebellar Syndromes.)

15. *Chorea tabica* in tabes with choreic crises with or without pain.

16. *Psychogenic Choreas.*—(See Psychoneuroses, Hysteria, Compulsion Neurosis.)

Chorea Minor (*Sydenham's Chorea, St. Vitus' Dance*) is the most frequent of these disturbances of spontaneous, purposeful and coördinating movements, which, occurring in children, usually recovers.

Infection and maldevelopment are the most frequent etiological factors. The most widespread infecting agents are various streptococci (tonsillitis), infectious diseases, measles, whooping-cough, etc., hence the frequent complications of infectious arthritides, endocarditis, with a mild meningitis which is usually present.

Girls are apparently less resistant following these infections, and hence show a higher percentage of choreic attacks.

Symptoms.—These show considerable variation, ranging from slight motor unrest and irritability to marked motor disturbances, with corresponding modifications of conduct and emotional response. The latter at times are so severe as to constitute a psychosis (choreic mental disease, *q. v.*). The motor symptoms are best grouped as pyramidal or extrapyramidal tract and cerebellar disturbances, either or both occurring in most patients. They are the results of definite irritations, occasionally of defect (diaschisis) lesions.[2]

[1] Boveri: British Med. Jour., April 24, 1920.
[2] Förster: Volkmann klin. Vort., 382.

The *spontaneous* movements are quick and show comparatively wide excursions. In the distal extremities cramps of single muscles or groups of muscles occur, with complete rest following the movements. There is great irregularity in the affected groups; there will be one or two movements, which are followed by opposite muscle action.

In mild cases these movements are limited to the face and to single muscles. In the more severe ones the entire body musculature is involved.

The arms are flung about, the legs are wobbled and pulled, walking is impossible, the larynx, lips, and eyes are in activity. The patients gasp, snort, and groan. In the mild cases the movements may cease during sleep; in the more severe ones the movements are continuous.

Any sensory stimulus may increase these spontaneous movements. They do not behave like willed movements. Hence extrapyramidal systems are also involved.

Pyramidal Tract Disturbances.—Certain choreic, jerky movements are observed apart from the more usual ataxias and incoördinate movements. The latter diminish with rest, quiet, and relaxation; the former do not seem to diminish as muscle activity is withdrawn.

A group of minor signs appear on close examination. One of these is the Babinski hand sign (dysmetria; see Fig. 340). When the choreic patient slowly raises the arms in front of the body, palm down, one side, that most affected, has a tendency to sag. Or if the hands are hanging by the side, the more affected hand shows a position half-way between pronation and supination, whereas, since the normal muscle tonus is greater in the pronators, the more healthy side is held more pronated. This is in accord with the general tendency for one-half of the body to be more affected than the other. The affected side is hypotonic; the shoulder droops more. There is apt to be exaggerated flexion or extension of the arms. In the lower extremities hyperflexions of the leg on the thigh occur. Since hypotonicity is characteristic of underdeveloped psychomotor integration, younger children do not bring these contrasts into relief.

If the patient lies flat upon the back and attempts to sit up, the arms being folded, the leg that is most affected is flexed on the thigh and raises from the bed, the healthy side remains flat. (Babinski thigh sign—Hoover). Similarly, if the patient lying on the back raises the legs simultaneously, the sound side rises higher than the other, or if one leg is raised and then the other, the sound side is raised higher and the choreic side also falls more readily.

The Hoover procedure by measuring the pressure of the leg on the heel during the movements just mentioned shows diminished pressure on the weak side. (See synergistic tests in chapter on Examinations.)

Förster showed that in choreics, particularly in the more unilateral cases, the closing of the fist of the sound side called forth an associated closure of the fist of the affected side, but not *vice versa*. Similar associated abductor or adductor movements usually occur in the lower limbs.

The Babinski, Oppenheim, Gordon, and Chaddock great-toe extension sign is occasionally present. Schilder denies this finding.[1]

Strümpell's contraction of the tibialis anticus occurs when the patient, lying on the back, attempts to flex the leg on the thigh against passive resistance applied to the thighs. The foot assumes the equinovarus position.

The tendon reflexes, patellar and Achilles, are apt to be variable and often delayed.

These signs are all suggestive of mild types of hemiplegia, and have been collected by Lhermitte under the title of the "Little Signs of Hemiplegia."[2] Some one or all may be present in even mild cases of chorea, especially when the disturbance is at its height and particularly in the severe infectious types. Their disappearance often takes place with the stage of recovery. Mild cases may show only the most passing signs, or very mildly developed ones.

Cerebellar Signs.—These are chiefly adiadokokinesia and asynergia. The former is frequently found, especially on the more hypotonic side. It is sometimes complicated by the choreic movements.[3]

Asynergia is the usual choreic type of movement. There is a jerky, irregular movement of the muscles which fail to perform well-adapted movement. Thus the patients drop things, lurch, stumble or fall, spill their food, or speak in a jerky, at times mumbling, manner. They are incapacitated from writing, playing on the piano, or for making any finely adaptive movements. The finger-nose and finger-finger tests show this asynergia by the overshooting of the mark, pseudo-ataxia. Attempting to grasp an object, the choreic opens the affected hand over-widely; the pencil test also shows similar overshooting.

Lymphocytes may be present in the cerebrospinal fluid. (See Meningeal Inflammation.)

Etiology and Pathogenesis.—A too narrow view of the causation of chorea has prevailed. While many patients undoubtedly develop a chorea following streptococcic or other infection, this whole group should be looked at from the developmental point of view as well. This renders fatigue factors comprehensible.

The so-called hereditary factor is probably dependent upon an inferior or slowly developing psychomotor-cerebellar integration. In certain patients the rapidly developing body calls for a higher grade of motor adaptation than the developing motor integration can subserve. This is seen normally in the cerebellar static apparatus in what is so widely called the awkward age, or in the "puppy" stage, where the analogues to mild choreic affections are obvious. Chorea may thus develop in adolescents from the mildest of infections or even from excess of motor activity. Thus chorea may be a fatigue symptom.

Ancestral syphilis is responsible for a small percentage of choreas, especially as affecting the complete development of the nervous

[1] See Parker and Stengel.

[2] Jelliffe; Little signs of Hemiplegia, Post-Graduate Medical Journal, 1912.

[3] Grenet et Loubet: Rev. Neur., December 15, 1912, p. 632.

system, thus allying such choreas with related spinal, cerebellar, and cortical ageneses (Friedreich, etc.). Hence the Wassermann test should be applied in chorea diagnosis. Milian has found from 60 to 70 per cent. of fifteen choreics examined to show positive Wassermann reaction.[1] Other investigators have found much smaller percentages. This may account for the good results from salvarsan in the treatment of some choreas usually of syphilitic origin.

Acute rheumatic infection loses much of its so-called specificity. Tonsillitis is assuredly not the universal malefactor. A large number of other infections, measles, scarlet fever, whooping-cough, typhoid fever, infected teeth (*Streptococcus viridans*), tonsils, intestines are important, and of later years the choreas of epidemic encephalitis have been frequent.

Diagnosis.—The chief disorders to be excluded are hysteria, frequently from imitation, and the more persistent and milder of the compulsive tics. Occasionally the choreic movements of myoclonus, multiple sclerosis, and explosion shock concussions are to be separated. The organic signs enumerated will be found in the majority of choreics, if carefully looked for. Lumbar puncture may be resorted to in difficult cases, although those with definite leukocytosis are apt to show the organic signs. In certain obscure subinfections, teeth, tonsils, intestines, *Streptococcus viridans* may be recovered from the blood. Streptococcus-fixation tests are also of value in the diagnosis.

The diagnosis of hysteria will depend upon the finding of definite conversion mechanisms, while that of the tics will on analysis show the typical substitution mechanisms, especially the infantile reproaches, in the unconscious. The movements in both may be similar to those called choreic, but in both hysteria and in tics hypotonus is less liable to show. There is a greater likelihood that so-called hysterical movements will turn out to be choreas than the reverse, especially in younger children. In older children or in young adults the opposite may be more seriously entertained.

Treatment.—The best treatment is rest in bed, with partial isolation, no playing, local treatment of infected throat, tonsils, teeth, full diet, with increased fatty ingredients (milk and eggs).

The rest in bed should be practically absolute for six weeks, and if anemia is present, and it usually is, iron and arsenic are useful.

One may start with milder arsenical preparations in less severe attacks, and in those where sudden disproportionate growth seems to play a larger role, Fowler's solution ♏ v–x or the acidi arsenosi may be used, either alone or in pleasing vehicles.

In severe and protracted cases the intravenous use of doses of 0.05 to 0.2 gm. of salvarsan, according to age, once a week for four weeks, is advantageous. Nirvanol, 0.3 gm. per day has been of service.[2] (Parker has obtained good results in septic cases by the cautious

[1] Milian: Soc. Méd. d'Hôp., November 29, 1912.
[2] Gottstein, W.: Klin. Wchnschr., October, 1927.

use of trypaflavin intravenously. Necrosis in paravenous injection should be looked out for.)

Most of the mild cases recover on prolonged rest in bed, without medication, if on a full diet with milk and eggs in abundance.

Huntington's Chorea.—This is a disorder of the nervous system, named after George Huntington (born 1850), an American physician, who gave the first essential, comprehensive and distinctive description of the disease. Huntington's grandfather and father had observed the disorder in one of its American foci, Easthampton, Long Island, and chiefly through their studies of several generations of afflicted families the essentially hereditary nature of the malady became apparent.

Huntington's chorea has no relationships to Sydenham's chorea, that essentially infantile disturbance of cerebro-cerebellar tract coördinations following so frequently upon infectious disease or exhaustion. Huntington's chorea is essentially hereditary and chronic, occurring, as a rule, in adults from thirty to fifty years of age.

The condition did not escape earlier observers. Thilenius gave a report of a case apparently as early as 1816. Rufs, another in 1834. Waters made his striking comment in 1841.[1] Dr. Charles G. Gorman, of Luzerne, Pa., wrote an inaugural thesis, in 1848, on this affection, which has been lost. Dr. Irving W. Lyon, while house physician at Bellevue Hospital, wrote a paper on "Chronic Hereditary Chorea," which was published in the *American Medical Times* in 1863. Huntington's paper appeared in the *Medical and Surgical Reporter,* Philadelphia, in 1872. Since this time a rich bibliography has accumulated, which in the Huntington number of *Neurographs,* edited by Dr. William Browning, in 1908, mounted to 200 titles.

Etiology.—So far as is known the disorder is hereditary. From eugenic studies of Davenport, Muncie, and Jelliffe,[2] the chief determinants appear to behave as Mendelian dominants in which the motor and mental factors are separately inheritable. Heilbronner has said that the disorder appears at later intervals in succeeding generations, but evidence from extensive eugenic studies, which includes the study of 1000 cases of Huntington's chorea, limited to a few families, shows that it appears at earlier years in succeeding generations. The data here indicated that the disease behaves as a complex in which age, motor disturbances, and mental defect behave more or less independently one of another. When all three factors combine, the result is Huntington's chorea. No other etiology is known. Certain clinical pictures indistinguishable from the symptoms alone from hereditary Huntington chorea have been observed in the encephalitides and in certain neurosyphilis cases.[3]

[1] Dunglison's Practice of Medicine, vol. **2**, 312.

[2] American Neurological Association, 1913. Muncie and Davenport: Am. Jour. Insan., 1916. Lewandowsky Monographs, Rüdin, 1921. Entres, Berlin, 1925.

[3] Urechia, Rusdea, Urechia and Elekes: Rev. neurol., May, 1922, 1923, p. 473; September, 1924, p. 269. Vogt, C. and O.: Monatschr. f. Psychiat. u. Neurol., 1920. Lhermitte, Wimmer, *et al.*

Symptoms.—As it is not possible in a short résumé to discuss the separate factors, the older lines of description which regard the disorder as a unit will be followed. Thus one speaks of an insidious onset, usually coming on between the years of thirty and forty. The earlier signs are either slight changes in character, irritability, moroseness, eccentricities, or the choreic movements become prominent in the picture. The facial, neck, and upper extremity muscles are usually involved first. There are involuntary, jerky movements, usually of muscle groups, not of muscle fibers. The excursions brought about are massive, *i. e.*, excessive, loose, and hypotonic. The hand is thrown to one side, the whole arm sweeping outward; the neck is jerked backward, the head bowed forward in a quick, loose-jointed, jerky sort of way. The motor unrest spreads over the entire body.

The patient, after several years—for the motor disturbance advances slowly—becomes jerky, and although for a long time voluntary movement is able to check the excessive motion, finally control is lost and the patient is confined to a chair or his bed, making his peculiar broad, jerky movements. During sleep the motions cease.

Nearly all of the voluntary muscles may be involved. The eye movements seem to resist to the last. The speech becomes explosive, or grunt-like, very incoherent at times by reason of the involuntary movements of tongue, lips, diaphragm, and chest. Even swallowing is involved in the later stages. Writing soon becomes impossible by reason of the jerkiness of the hands and arms. Walking becomes successively more and more unsteady until the patient becomes bed-ridden.

There are few disturbances of sensibility and these are observed only late in the deteriorated phases. The knee-jerks are active or uninvolved; there are no atrophies, nor paralyses, nor hypertrophies.

The mental changes may develop apart from the motor ones, and in choreic families mental choreics are to be recognized who perhaps may never show motor signs or those who show choreic movements very late. This is the basis of Diefendorf's constitutionally defective group. These patients even in childhood may be excessively nervous, irritable, and excitable. There are often difficult children to manage. Their eccentricities become more and more marked with advancing years. Some show marked grades of feeble-mindedness and occasionally are born choreic and defective.

In the great majority of the cases the insidious and slow development of great instability and irascibility shows itself coincident with or following the choreiform movements. Angry outbursts and destructive impulses occur, often followed or preceded by periods of marked moroseness and despondency. This depression or gloom may be a forerunner of suicide. Diefendorf remarks that this despondency is not due entirely to the realization of having the disease. With some patients, however, the suicide is to be traced to the knowledge of the taint. Suspiciousness, paranoid ideas and jealousies are not infrequent mental signs. Emotional deterioration follows. The patient

loses all interest in his work, his appearance, his home, etc. Some become tramps. Intemperance and free erotic activities may show themselves with this gradual deterioration. Indifference shades off into absolute incapacity and deterioration becomes profound, always, however, showing itself in the affective sphere more prominently than in the intellectual capacities, although these latter are not free from gross defect; the patients being forgetful, poor in ideas, disorderly in thought and weak in judgment.

Diefendorf speaks of a group in which the mental symptoms develop somewhat similarly to those seen in the hebephrenic types of dementia precox. These patients complain of insomnia and general malaise. They often then develop ideas of reference, anxiety, suspiciousness and ideas of infidelity. In some of these impulsive activities show themselves. Homicide has taken place. Kraepelin cites an illustration of a choreic father who killed his three small children by hanging, as he feared he could not support them, then quietly took a walk and was quite unconcerned about the affair at a judicial hearing. The eating is often impulsive and ravenous. In some of these cases, as with the inferior group, the mental symptoms may develop long before the choreic symptoms, and the diagnosis of a katatonic schizophrenia may be made as the motor symptoms become manifest. Here the diagnostic difficulties are very definite.

It would appear from Diefendorf and the studies cited that the mental and motor traits are more or less independent one of the other. In inheritance they seem to show as such. Some patients have shown choreic movements for twenty to thirty years without mental signs.

Course.—This is subject to great variation. Often the patients suddenly develop great motor unrest; the mental signs augment rapidly, and the patient dies in exhaustion five, ten or fifteen years after the onset of the symptoms. Usually the disorders, *i. e.*, motor and mental, are progressive; rarely they may diminish after reaching a severe grade. Many die of intercurrent disease.

Pathology.—No unitary interpretation seems yet possible. The motor and the mental symptoms are best considered separately.

A number of autopsies have shown a variety of findings. At times there are chronic meningeal thickenings, again generalized brain atrophy. This reduction of the cortex may show to a marked degree in the loss of cells, particularly of the third layer. There is a compensatory increase in neuroglia. In some patients arteriosclerosis has been present, in others not. The older patients showed the arteriosclerotic changes. These cortical cellular changes are apparently more frequent in the frontal areas. They are correlated with the mental defects.

The pathology behind the motor manifestations is less obscure. Theoretical considerations as well as pathological findings point to an implication of the striato-thalamic, strio-rubral and strio-cerebellar pathways as chiefly responsible for the perverted movements. Kleist, Zingerle, Jelgersma, Hunt, Lewy and Winkler adduce observations from various sides which tend to show that these mechanisms are

involved.[1] Numerous autopsies show changes in the lenticular region which may be taken to support these contentions. Thus the motor signs have a pathology closely related to that seen in paralysis agitans and other striatum syndromes. The putamen region shows distinct losses.[2] Terplan reports dentate nucleus changes.

Therapy.—There is no efficient therapy. Many patients need hospital care. Prophylaxis is important, Mendelian dominance arguing for certain factors at least, that these patients should not procreate. The percentage of chance of escape for Huntington choreics is about one in four at the best. Some branches escape, and in accordance with the law of dominants, a branch once free, is always free. Only a few exceptions to this are found in the Davenport-Muncie series.

[1] Roussy and Lhermitte: Arch. d. méd., 1915.

[2] See Lewy: Ztschr. f. d. ges Neurol. u. Psychiat. (loc. cit.). Hunt, J. R.: Trans. Am. Neurol. Assn., 1920; the literature quoted in the section on Striatal Syndromes, particularly that of the Vogt, Wilson, Hunt and Foerster. Terplan: Virchow's Arch. f. path. Anat., 1924, **252**, 146. Dunlap: Pathological Changes in Huntington's Chorea, Arch. Neur. and Psych., 1927, **18**, 867.

CHAPTER IX.

CEREBELLAR SYNDROMES.

The cerebellum occupies the posterior cerebral fossa; is separated from the occipital lobes of the cerebrum by the tentorium, and rests upon the pons and medulla, forming part of the upper boundary of the fourth ventricle. It is connected with the rest of the nervous system by the anterior medullary velum, the superior, middle, and inferior cerebellar peduncles and posteriorly by the posterior medullary velum. The tela choroidea forms the posterior continuation of this last structure, and serves as a roof to the posterior part of the fourth ventricle.

Being intimately connected with structures in the midbrain, the red nucleus and the optic thalamus, with bulbar and pontine centers and with the cord; containing important nuclei,[1] the dentate nucleus, Deiters' nucleus, nucleus globosus, nucleus emboliformis, tectal nuclei, etc., with a richly developed cortical ganglionic system, and with a multiplicity of incoming and outgoing pathways, the possibilities of symptomatology are very numerous.[2]

The cerebellum is a central switchboard, composed of complex fiber and synaptic connections, the chief function of which as seen at present is the coördination of the reflexes of the proprioceptive system (Sherrington), for the purpose of properly synergizing bodily displacement activities. This synergism may involve visceral movements. An older, paleocerebellum, is to be distinguished from a neocerebellum. (Winkler.)

It thus represents the entire body in a proprioceptive inner regulatory sense in which knowledge of the muscle group localizations has commenced to accumulate and be definite. Visceral or vegetative localization factors are still speculative although probable. They are denied emphatically by Jelgersma, as well as all sensory function save that of muscular sense. The extremely intricate and much discussed problem of cerebellar localization, particularly of different muscle groups cannot be entered into here very exhaustively. The student is referred to the studies of Bolk, Rijnberk,[3] Lewandowsky, Herrick, Thomas, Brouwer,[4] Ingvar, Jelgersma, Tilney and Fulton for more details.

[1] See Tilney: Brain, 1927, **50**, 275, for these nuclei and Demole for connections, Deutsch. Arch. Suisse de Neur., 1927, **20**, 271.

[2] Thomas: The Cerebellum, Nervous and Mental Disease Monograph Series; Pathologie du Cervelet, Nouv. traité de méd. Roger, Widal, Teissier, Masson, 1925. Bolk: Das Cerebellum der Säugethiere, 1906.

[3] v. Rijnberk: Les dernieres recherches, etc., Arch. Neerl., 1925, **10**, 155, et seq.

[4] Brouwer: Psychiatrische und Neurologische Bladen, 1915, p. 104; Arch. f. Psychiat. 1913, **51**, 539. Jelgersma: Die Funktion des Kleinhirns, Monatsschr. f. Psychiat. u. Neurol., 1918, **33**, 137. Tilney: Neurological Bulletin, 1920. Tilney and Riley: Loc. cit. Bruns: The Cerebellum. Thomas: Loc. cit; l'Encéphale, 1920, **15**, 114. Marburg: Handb. d. Neurol. des Ohres. Mills and Weisenburg, Jour. Am. Med. Asso., 1923, **1**, 175. Duchenne de Barenne: See Study by Winkler: Neopaleocerebellum, v. Monakow's Festschrift. Schw. Arch., 1923, vol. **13**. Symposium on Cerebellum Brain, 1927, **50**, 275. Goldstein: Das Kleinhirn, 1927, and valuable summary in Fulton's important work on Muscular Contraction and Reflex Control, Chapter XX, 1926. Complete bibliography.

We add here a tabular review of Bolk's localization hypothesis which, in the main, has seemed to be supported by most modern investigations, although there are certain investigators who challenge Bolk's hypotheses (A. Troell and C. Hesser).[1] It may be stated that there exist in the cerebellar hemispheres centers for both upper and lower extremities. These centers are decomposable into secondary centers representing the chief articulations and the mode of action of the muscles; abduction, adduction, flexion, extension, etc., are all represented. In the vermis centers exist for the muscles of the head, eyes, tongue, jaws, face, larynx, pharynx and trunk. Passive as well as active motion are involved in the disorders of these centers and the asthenias and attitudes of the muscle groups offer important diagnostic signs.

Anterior lobe		Lobulus 1, Lobulus 2, Lobulus 3, Lobulus 4	Lingula; lobulus centralis alæ; lobulus lunatus anterior. (Head muscles: eye, chewing, mimetic, tongue, larynx, pharynx.)
Posterior lobe	Lobulus medianus posterior (vermis inferior)	Lobulus C^2	Lobulus simplex; neck muscles.
			Lobulus C^2 (impaired extremity center).
		Lobulus C^1, pyramis; Lobulus b, uvula; Lobulus a, nodulus	Breathing and perineal muscles (?).
	Lobulus lateralis posterior	Lobulus ansiformis	Crus primum (Lob. semilunaris; extremity muscles; paired forefeet.)
			Crus secundum (lob. cruceatus; extremity muscles; paired hindfeet.)
		Lob. paramedianus;	tonsilla; extremity muscles; paired hindfeet
		Paraflocculus (flocculus; formatio vermicularis gluteal muscles)	Pars tonsillaris; tail muscles.
			Pars floccularis.

Deficiency of cerebellar function is replaced by intervention of the cerebrum, and for this reason, in cases of progressive lesions (especially in the olivo-ponto-cerebellar region), the cerebellar symptoms make their appearance only if the cerebrum, too, is involved. These circumstances explain the grave functional results from even very minute lesions of the central nuclei (nucleus dentatus and of the tegmentum) or of their afferent or efferent paths, as in these regions are stored or conducted in very limited spaces all the excitations coming from the cortex. Destructions in the inferior superior cerebellar peduncles are rarely limited to these regions, hence their gravity. Lesions of the cerebellum do not suppress motions entirely, at least in certain territories; those of the cerebrum are definite.

From the cerebellar cortex, which may thus be looked upon as

[1] Troell and Hesser: Acta chir. Scand., 1921, vol. **54.** Meyers: Jour. Nerv. and Ment. Dis., 1919. Bechterew: Die Funktionen der Nervencentra, 1909, vol. **2.**

chiefly, if not exclusively, sensory, these impulses pass to the various nuclei of the cerebellum, and are there redistributed. It would appear that these intrinsic cerebellar nuclei are mainly motor.[1]

In summarizing the evolutional significance of the cerebellum, Tilney and Riley[2] conclude: (1) That the cerebellum has been added to the neuraxis as a suprasegmental structure in the interest of coördination. (2) That in its primitive form it correlates sensory impulses received from all the chief receptors of the body and utilizes the impulses thus associated most advantageously to accomplish the purposes of coördination. (3) That in the lower vertebrates this coördination requires the harmonious action of the axial musculature, and depends upon the bilaterally synergic control of the central arched portion of the cerebellum, corresponding in position to the vermis, although certain lateral structures, such as the paraflocculus and flocculus, were added for the more or less independent movements of the tail. (4) That when the upper and lower extremities made their appearance and became capable of independent movements, they needed something more than bilaterally synergic control. Out of this necessity grew the expansive lateral portions of the cerebellum, the cerebellar hemispheres, which provide unilateral synergic control for the arms and legs, exercising regulation over such independent and individual movements as these extremities have the capacity to develop. (5) That, as a natural consequence, the greater the range of independent movement in the extremities, the greater has become the cerebellar area required for its control. (6) That the greatest range of independent movement is seen in the skilled performances which are the special attributes of man, who, as a result, has the most highly developed cerebellar hemispheres. (7) That the central or vermal portion of the cerebellum not only still retains its primitive control of the bilaterally synergic movements of the eyes, face, tongue, jaws, larynx, neck and trunk, but also maintains a similar relation to movements of the extremities which require bilaterally synergic control.

Thomas[3] has proposed the term *stagia* for what is here called *synergia*.

The main connections of the cerebellum, afferent as well as efferent, are by means of the *inferior*, *middle* and *superior* peduncles.

The Inferior Cerebellar Peduncle (*Restiform Body*).—This structure consists of a massive tract of fibers connecting the spinal cord and medulla with the cerebellum. It contains spinal and bulbar components. The chief constituents of this peduncle are afferent fibers and consist of:[4]

1. The dorso-spino, or *direct cerebellar tract*, of Foville and Flechsig, the fibers of which arise in Clarke's column, pass upward in the cord. They form synapses in the cortex of the anterior part of the superior

[1] Horsley: Function of the Cerebellum, Brain, 1906, **29**, 446.

[2] Tilney and Riley: Form and Function of the Nervous System, New York, 1923.

[3] Thomas: Cerebellar Symptomatology and Localization, l'Encéphale, 1920, **15**, 114; Pathologie du Cervelet, loc. cit., 1925.

[4] Tilney and Riley: Loc. cit. Bechterew: Loc. cit. Thomas: Loc. cit.

vermis on the same side. Some (Monakow?) fibers reach the opposite side of the vermis by way of the superior commissure. This tract conveys impulses from the proprioceptors of the limbs and trunk.

2. The *arcuate fibers* arise in the nucleus gracilis and nucleus cuneatus of the opposite and the same side and enter into the formation of the inferior cerebellar peduncle. These axones end in the cortex of the superior vermis of the same and opposite sides. Some of these fibers (Thomas) enter into the formation of the median lemniscus.

3. The *olivo-cerebellar tract,* arising in the opposite inferior olive and to a limited extent in the olive of the same side, form the lateral portion of the restiform body, end in the cortex of the hemisphere and vermis, as well as in the fiber capsule enveloping the nucleus dentatus. Most of the fibers in this connection are afferent. It is possible, however, that some axones are efferent and conduct impulses from the cerebellum to the olive.

4. *Fibers from the nucleus lateralis of the medulla,* which pass to the cortex of the cerebellar vermis.

5. *Fibers from the arciform nucleus,* which pass to the cerebellar cortex of the opposite side.

6. The *nucleo-cerebellar tract,* comprising fibers from cells in the reception nuclei of the trigeminal vestibular, facial, glossopharyngeal and vagus nerves. The tract occupies the mesial portion of the peduncle and ends in the nucleus tecti of the same and opposite sides.

7. *Other fibers pass in the reverse direction* from the roof nucleus to the dorso-lateral vestibular nucleus (nucleus of Deiters) and thence by the Deitero-spinal tract to the medulla and spinal cord.

8. *Additional vestibular and possibly other sensory fibers* pass without interruption by way of the restiform body to the roof nuclei and constitute the direct sensory cerebellar tract. The majority of these fibers enter into the formation of a discrete fasciculus mesial to the inferior cerebellar peduncle, the juxta-restiform body.

The Middle Cerebellar Peduncle.[1]—This connection, an afferent one, is also known as the *brachium pontis* (Tilney and Riley). It is a continuation of the pons into the medullary portion of the cerebellum, and transmits impulses arising in the frontal, parietal, occipital and temporal areas of the cerebral cortex. It does not establish a direct means of communication, but, by relay in the pontile nuclei, completes the pallio-ponto-cerebellar connection. The chief constituents of the middle cerebellar peduncle are the ponto-cerebellar fibers forming the continuations of the tracts connecting the frontal, temporal, occipital and parietal areas of the cerebral cortex with the pontine nuclei.

These fibers take origin in cells of the frontal, temporal, occipital and parietal lobes respectively. They descend through the internal

[1] Bechterew, vol. **2**, 961. Bing: Die Bedeutung des spino-cerebellaren Systeme, 1907. Rynberk: Folia Neurobiologica, 1907. Tilney and Riley: Loc. cit. Schaffer: Der Kleinhirnanteil der Pyramidenbahn, Ztschr. f. Neurol. u. Psychiat., vol: **47**, 435. Bruns, R.: Zur Kenntniss der Bildungsfehler des Kleinhirns, Arch. suiss. d. neurol. et psychiat., vols. **1**, **2**, **3**. Thomas: Loc. cit., 1925. Minkowski and Rademaker: Deutsch. Ztschr. f. Nervenh., 1926, **94**, 144.

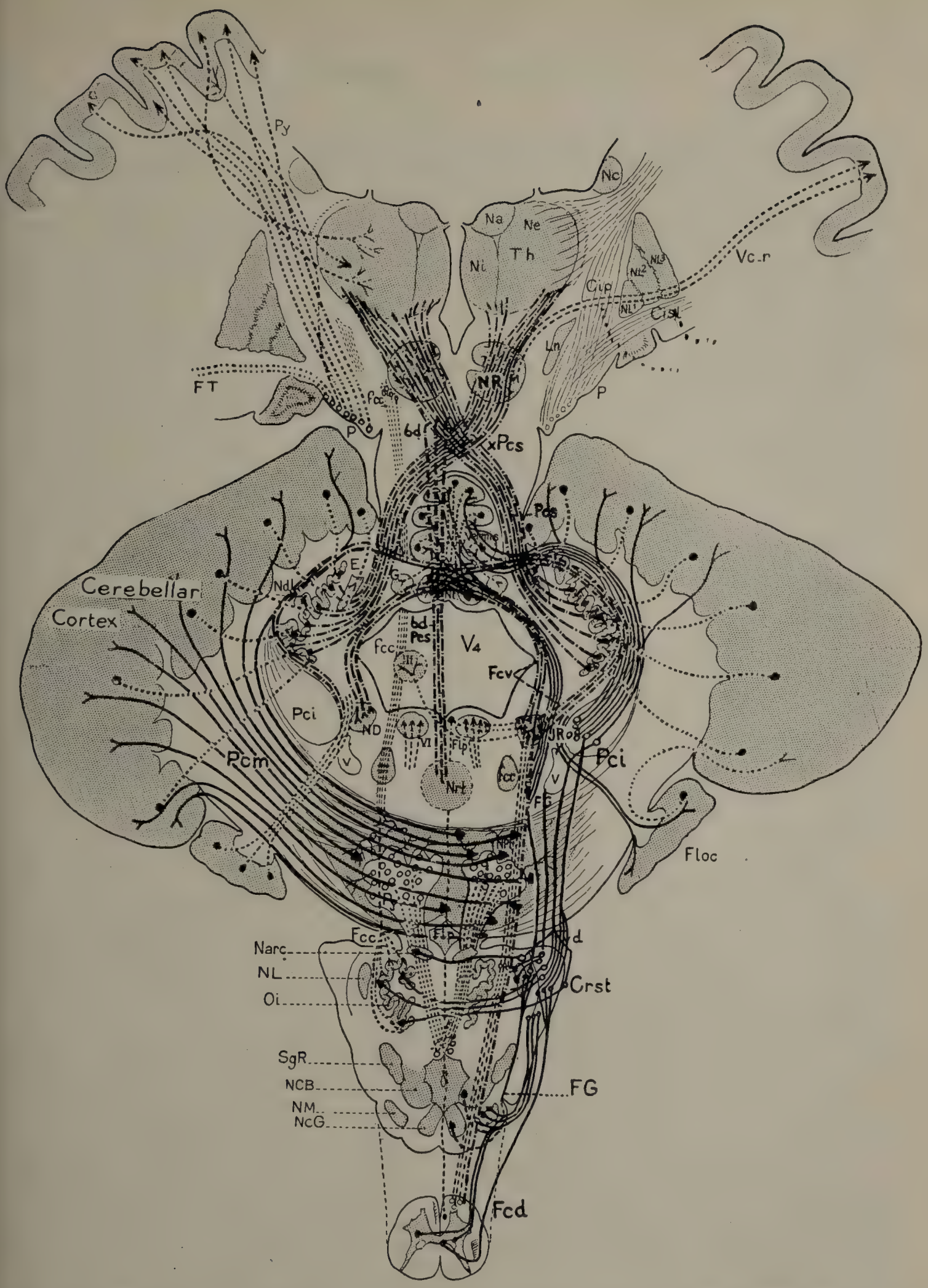


FIG. 334.—Scheme of Cerebellar Connections. Modified from Thomas, Pathologie du Cervelet. In Roger, Widal, and Teissier Nouveau Traité de Med. Fasc. 19. Masson, Paris, 1925. Projection fibers, dotted line: Afferent fibers, solid line: Efferent fibers, broken – . – line.

Projection fibers. Architecture of Cerebellum. Direct and indirect cerebellar connections. Cip. Caps. Int. post: Cisl. Caps. int. retrolent. E. Embolus. Fcc. Central fasciculus of the calotte. Floc. Flocculus. Flp. Post. long. fascic. FT. Türcks Bundle. Fvd. Descend. Cerebell. fasc. G. Glubulus. Ln. Locus niger. Na, Ne, Ni.: Ant., ext., and int. nuclei of thalamus. Narc. Arciform nucleus. NC.^{n}. Caudate^{n}. NCB^{n} Burdach^{n}. NCO^{1}. Goll. N. ND. Deiters N. Ndl. Dentate—olivo-cerebellar: Nl. N. later. col. NL,^{1} NL,^{2} NL,^{3} Lenticularis: N. Ns. N. v. Monakow: NPE. Pons Nuc. N. R. Red nuc. Nrt. Nuc. retic. tegm. pontis. Nt. Roof nucl. Oi. Infer. Olive. P. Cerebr. Ped. Py. Pyramid. Pci. Inf. cerebell. ped. SgR. Subst. Roland. Th. Thalamus. V.^{4} Fourth Vent. Vcr. Cerebrorubral tract. III. Oculomotor. V. Descend. Fifth. VI. Ext. Oculom.

Afferent Fibers. Crst. Corp. restiforme. Fcc. Direct cerebellar tract. FG. Gowers. Pci. Inf. cerebell. ped. Pcm. Middle cerebellar ped.

Efferent Fibers. Fcv. Cerebell. vest. tr. J. R. Corp. juxtaforme. Pcs. Super. cerebell. ped. Bd. Pcs. Desc. branch sup. cerebell. ped. Pcs. Decuss. sup. cerebell. ped.

capsule and the cerebral peduncle to end in the cells of the pontile nuclei. From these cells arise the ponto-cerebellar fibers, which decussate in the pons and convey impulses to the hemispheres, to the vermis and possibly to the dentate nucleus of the cerebellum. Cerebello-pontile fibers have been described, but their existence is by no means well established.

The Superior Cerebellar Peduncle.—Tilney and Riley[1] state that this peduncle forms the pathway by means of which the cerebellum exerts its influences over the muscles and distributes impulses to other destinations. Together with its fellow of the opposite side, it undergoes a complete decussation before reaching its next relay station, *the red nucleus* of the midbrain. Its chief constituents are the *cerebello-rubral tract* and the *cerebello-thalamic tract.* It also contains a *cerebello-tegmental tract.* The principal elements of the latter fasciculus are fibers which arise in cells of the dentate nucleus, emerge from its hilus, receive augmentations from the roof nucleus and become consolidated as a rounded bundle. After passing cephalad in the lateral wall of the fourth ventricle, the superior cerebellar peduncle sinks ventrally and disappears beneath the inferior colliculus. Many of its fibers continue their course through the tegmentum of the midbrain into the subthalamic region of the diencephalon. Before reaching the level of the red nucleus the peduncle undergoes decussation, thereby establishing a contralateral connection between the dentate and rubral nuclei. After decussation the peduncular fibers form a capsule which surrounds the red nucleus. Many of the fibers entering into this capsule penetrate and form synapses in the nucleus ruber. This establishes a crossed dentato-rubral connection. The cells of the red nucleus give rise to descending axones which, immediately upon leaving their nucleus of origin, undergo complete crossing to the opposite side in the *ventral tegmental decussation of Forel.* The fibers then become collected to form a descending fasciculus, the *rubro-spinal tract of Monakow.* By means of this double decussation, first in the superior cerebellar peduncle and then in the ventral crossing of Forel, the cerebellum exercises an ipsilateral control over the muscles of the body.

Some peduncular fibers which participate in forming the capsule of the red nucleus either pass directly forward to the subthalamic region or are first relayed in the nucleus and then enter the subthalamus. The significance of this dentato-subthalamic connection is not clear. It furnishes the possibility of a cerebello-subthalamo-cortical pathway for the communication to the cerebral cortex of impulses concerned in equilibratory sensibility.

The cerebellum therefore forms an important sensorimotor station in a complicated series of reflexes which work for the most part automatically. The cerebellar cortex contains the chief primary reception synapses for relaying the impulses coming from the cord and brain-stem traversing the paths just enumerated, convey receptor stimuli

[1] Tilney and Riley: Loc. cit. Fulton: Loc. cit. Demole: Loc. cit. Thomas: Loc. cit.

chiefly concerning the states of tension of the muscular apparatus of the trunk, the extremities, and the head, and the states of pressure in the joints and tendons. The reflexes combine to regulate the constantly altering positions of the entire body in space (synergia), and also possibly of the viscera. The cerebellum therefore, in this sense, acts as a regulating, coördinating organ for the estimation of the body in space. To the spinal, midbrain, cerebral reflex arcs there are also added fronto-pyramido-ponto-bulbo-cerebellar arcs which contain involuntary as well as voluntary regulatory coördinating impulses, acting to orient the body in practically all of its spatial relations. Cyon adds that therefore the cerebellum is intimately concerned with the mathematical sense in its objective space relations.[1] In its symbolic component the cerebellum and art (symmetry) must be intimately related. No studies have been carried out along this line.

Vegetative functions are not definitely located, but are probable. Willis early assumed the cerebellum to be the center of organic life, as Gall localized the genesic sense there, but the researches of recent years have been silent on the question of the cerebellum and metabolism. Only very recently has the subject of central control of metabolism been reopened to investigation. (See *Vegetative Neurology* and studies by Dresel, Lewy and others.) Shinosaki, working in Lewy's laboratory, has shown that parts of the sugar-regulating pathways are present in the vermis. Bolk has also said that as the caudal fibers of the dorso-spino-cerebellar tracts end in the uvula, here vegetative synapses are to be expected. The subject is yet too unsettled for textbook presentation.[2]

Symptoms.—The general symptoms of disturbances of the proprioceptive pathways and the cerebral projicient systems as they converge in the cerebellum, offer a multitudinous complex, the details of which, and their anatomico-pathological correlations, are still being rapidly added to. Among these, however, there are certain outstanding trends which are capable of partial differentiation as syndromes at the present time, and to which attention will be directed.[3]

Cerebellar Ataxia (*Asynergia*).—This complex of disturbances is one of the classical signs of cerebellar disorder. It consists in a disturbance of the coördination of the larger muscular group actions, principally of the trunk and lower extremities, although the head, eyes, tongue and upper limbs also show it. Thus, standing, walking and the making of fine coördinated movements are interfered with in the absence of signs of distinct paralysis. (Mills and Weisenburg.)

In walking, the wobbling, side-stepping gait, so well described by early French authors (Duchenne of Boulogne) as the drunken gait, is characteristic. In severe grades of trunkal asynergia standing, and

[1] Cyon: Das Ohrlabyrinth, 1908. Kooy: Inferior Olives in Vertebrates, 1916.

[2] Shinosaki: Ztschr. f. Neurol. u. Psychiat., 1926, **106**, 483. Papillan et Cruceanu. Vegetative Functions, Jour. Phys. et path. gener., 1926, **24**, 47.

[3] See symposium on the Cerebellum papers by Weisenburg and discussion by Walshe Brain, 1927, **50**, 357, et seq. Also Bard. Rev. Neur., 1925, **1**, No. 5, and Rossi, Studi Neurologica Tanzi, 1926.

even sitting, becomes impossible. In milder grades one sees these types of disturbance in many choreas, in paralysis agitans, in general paresis, multiple sclerosis, etc.

A partial study of the asynergic cerebellar gait has shown several trends of disturbance:[1] first, staggering (lateropulsions) toward the affected side, at times forward or backward, according to location of lesion in vermis (or dentate nucleus). The patient feels as though shoved to one side and in the attempt at restitution overcorrects (asynergia of Babinski) and thus sways; second, the entire orientation in space is influenced and the patient's movements, as a whole, swerve in the direction of the affected side (menagery movements, as seen in whirling white mice; tumbling movements, as seen in the tumbler pigeon).



FIG. 335 FIG. 336

FIGS. 335 and 336.—The peculiar station and gait of cerebellar ataxia. (Thomas.)

Conscious attempts at correction (fronto-cerebellar paths) produce the larger zig-zags and the slowness in the general course of the progression. Forward and backward movements have their special localizing signs to be spoken of. From the anatomical standpoint these ataxias may result from involvement of the spino-cerebellar paths (Flechsig, Gowers —vestibular systems), as in the Friedreich and Marie ataxia hereditary groups; from involvements in the cerebellum itself (tumors, cysts,

[1] Stewart and Holmes: Brain, 1904. Hoff and Schilder: Exaggeration of Imitative Movements, Deutsch. f. Ztschr. Nervenh., 1926, **93**, 161.

ageneses, scleroses); in affections involving the superior cerebellar peduncles—from bulbar and pontine involvements of these paths, and also from implication of the cerebello-rubral, cerebello-thalamic and fronto-cerebellar paths. Atypical cerebellar ataxias are thus seen in some frontal tumors, with classical intoxication gait, but on close scrutiny these frontal ataxias show resemblances to so-called hysterical ataxias with predominant retropulsion features (Schilder[1]).

Dysmetria.—This is an asynergia of muscle groups not primarily involved in equilibrium maintenance. The term has been used to describe chiefly the faulty measuring movement of the upper extremity, bilateral or unilateral.[2] The individual muscular activities are illy coordinated, both as to space and time (asynergic), the desired act only being arrived at after several efforts, with over- and under-correction in point of the measure of the movement and as to its timing. This dysmetric asynergia may be brought out by grasping a glass, when the brusqueness of the attack and the over-extension of the fingers, dominant and check elements in the synergic unit (Tilney), is manifest. It is also shown by the brusque pronation and supination of the hand, by the finger-nose test, finger-finger test, finger-ear test and by writing. Visual aid, or its lack, has little or no influence on the dysmetria as tested by the finger-nose test and finger-finger test, and the dysmetria is a constant one, non-increasing as in an intention tremor (with varying gradations, for certainly the intention tremors of multiple sclerosis are often due to interference with cerebellar mechanisms). Guillain has reported an interesting war picture of multiple sclerosis-like tremor from cerebellar involvement.[3]



FIG. 337.—Cerebellar gait attitude. (Thomas.)

The Bárány finger pointing tests almost invariably show important modifications in cerebellar disease and are always to be utilized. His work on cerebellar localization is of great importance. (See Examination chapter.)

[1] Gerstmann: Wien. klin. Wchnschr., 1927. Also consult Rademaker: Statik und Motilitätstörungen kleinhirnlosen Thieren, Verh. d. 16th Congr. Deut. Nerv. Aertze, Deut. Ztschr. f. Nervenheilk., 1926, who shows that following the removal of the cerebellum the postural reflexes are similar to the righting reflexes (Magnus).

[2] Soc. de neurol., November 9, 1899; Rev. mens. d. méd. int. et de thérap., May, 1909. See Jelgersma's discussion, Monatsschr. f. Psychiat. u. Neurol., 1918. Tilney and Riley: Loc. cit., p. 471. Simonelli: Arch. d. physiol. norm. et path., 1926, **24**, 461.

[3] Réunion du vi Armée, May 17, 1915.

Adiadokokinesis.[1]—Originally described by Babinski[2] as characteristic of cerebellar disease, this symptom, consisting of a disability in

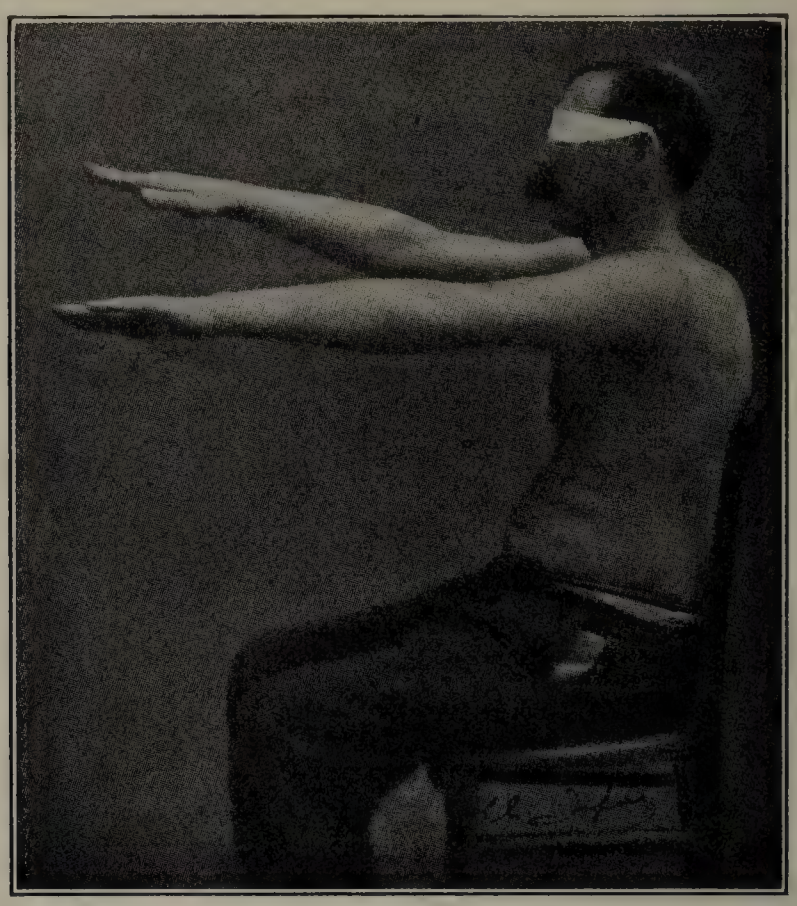


FIG. 338.—Test for dysmetria in cerebellar disturbances.



FIG. 339.—Showing cerebellar dysmetria.

the performance of rapid movements involving the alternating actions of agonist and antagonist muscles (see Examination), is not invariably

[1] Macfie Campbell and Crouzon: Rev. neurol., 1902, p. 1186.
[2] Soc. de neurol., November 6, 1902.

found nor always clearly indicative of cerebellar disorder; yet it is so frequently found as to merit special attention. It is in part a variant of asynergia brought out by a special test. Here the synergic group unit action, dominant and check (Tilney), is at fault. In the absence of paralysis it usually is indicative of cerebellar path involvement. It is frequently absent in extracerebellar tumors in which there are other well-marked cerebellar signs, as in frontal tumors.

Vertigo.[1]—As the chief organ of orientation in space, severe disturbance of certain of the cerebellar reflex paths causes vertigo, which is apt to be a prominent and a fairly constant sign. The vertigo is of a rotatory character. The patient may not only feel himself revolving in space, but objects may go around from right to left or from left to right; more rarely the vertigo has an up or down character. Each of these two characters is to be closely inquired into. Here the chief lesions are connected with the vestibular paths, as the labyrinth is the chief cephalic ganglion in the whole proprioceptive system, of which the cerebellum constitutes the coördinating switchboard. Thus labyrinthine disease itself, as well as disease of the extracerebellar or intracerebellar vestibular paths may give rise to the symptom. By means of the specific tests devised by Bárány (see chapter on Examination) a separation of labyrinthine diseases of extracerebellar origin is usually possible.

Further, enough experience has accumulated (Stewart and Holmes, *loc. cit.*) to show that, in general, objects rotate from the diseased to the well side for intracerebellar as well as extracerebellar affections, whereas the subjective sense of rotation is usually from the diseased to the well side in intracerebellar involvement, and the reverse in extracerebellar involvement of the paths. This generalization, a little too broad, is in need of further study, and of more accurate localization.

Nystagmus.—A fourth sign, rarely absent in cerebellar path disturbance, is nystagmus. It is also closely related to the vestibular reflex system, and may result from extracerebellar involvement as well as from intracerebellar implication of the paths.

True vestibular nystagmus is almost invariably accompanied by vestibular vertigo and ataxia. Vestibular nystagmus itself is usually modified by the position of the head, hence every person with nystagmus must be examined with the head in three planes; a patient with vestibular nystagmus tends to rotate within the plane of the nystagmus, and in the direction opposite to that of the quick nystagmus movement.

A patient with vestibular nystagmus then, who bends his head forward at 90 degrees will rotate in a direction directly opposite if he bend his head backward 90 degrees. The laws of intracerebellar nystagmus, apart from actual vestibular disease, remain to be investigated (conjugate deviations, skew deviations, and other eye displacements are

[1] Moulonguet: Les Vertiges Labyrinthiques, Paris, 1927. Lévy-Valensi et Halphen, Les Vertiges Maloine, 1926.

to be interpreted in the light of forced movements having their analogies to nystagmus, and are considered in the chapter on Midbrain Disease. See illustrations of conjugate palsies; also in chapter on the Eye Paths. See plate of oculorotary and cephalorotary mechanisms (Plate X).

Closely associated with disorder of the vestibular system are pain in the muscles of the neck, nausea, vomiting, amblyopias, and loss of consciousness.

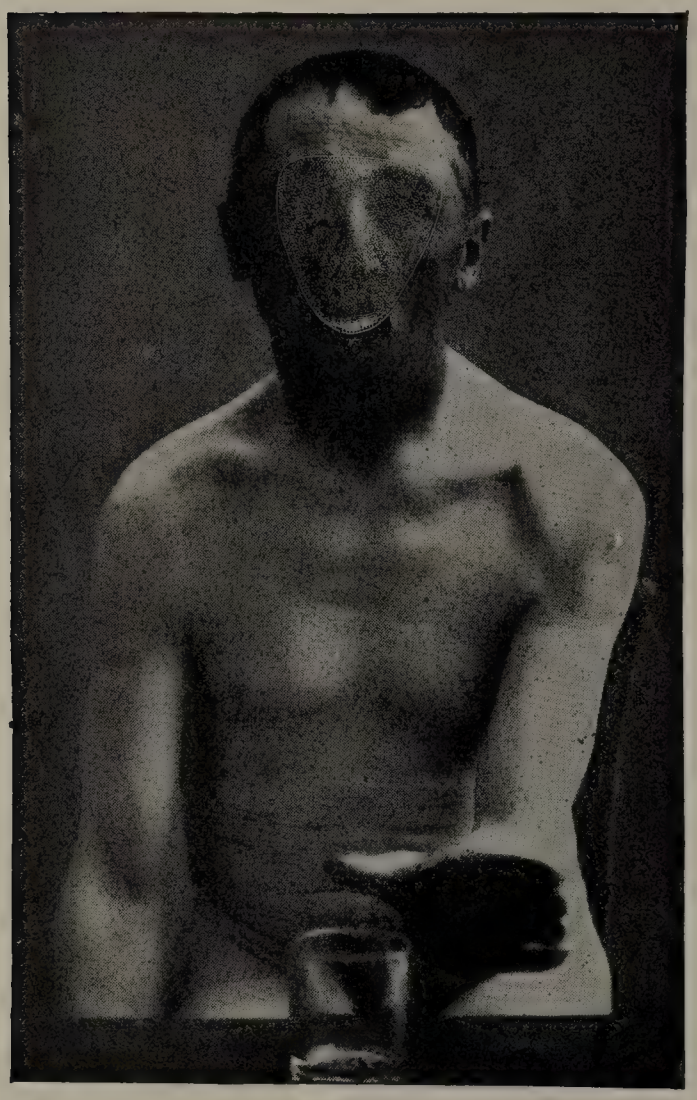


FIG. 340.—Dysmetria of Babinski developed on attempting to take hold of a glass. The fingers are held very far open. (Thomas.)



FIG. 341.—Asynergia of Babinski. (Schaller.)

Cerebellar Hypotonus.—Palpation of the muscles, testing of resistance movements, and looseness in the performance of passive movements, reveal a type of muscular hypotonus in cerebellar affections which is usually homolateral. This hypotonia, or atonia, is usually accompanied by normal or even exaggerated tendon reflexes in contrast to that of peripheral neuritis or tabes. One feature of this hypotonus noted by Stewart and Holmes is striking. If a resistance to a definite movement be suddenly relaxed, in the normal flexing of the arm, for instance, there is a sudden flexor-jerk, followed by an extensor recoil. In a cerebellar hypotonic reaction the flexor-jerk is excessive, and is rarely followed by a recoil.

Asthenia.—A paresis or asthenia, usually homolateral, is closely related to cerebellar hypotonus. Its presence in cerebellar disorder has usually been interpreted as due to a lesion of the pyramidal tracts by contiguity; unquestionably, however, it is a true cerebellar symptom. It is chiefly present when the cerebello-vestibulo-spinal and rubro-spinal tracts are affected.

Cerebellar Fits.—Originally described by Jackson[1] as tetanus-like convulsive seizures, with characteristic holding of the body in extended rigid position. (Catalepsy of Babinski.)

Forced Movements.—These are present in the neck muscles, muscles of the eyes, and appear as irritative or as defect symptoms, due to disease of the hemispheres or of the middle cerebellar peduncle. (See Diseases of Midbrain.)

Speech Disturbances.—Dysarthrias usually indicate the same type of asynergia as found in other muscles of the body, adiadokokinesis. They are usually present with defects of the cerebellum, and may indicate general defect of the entire apparatus, disease of the bulbo-cerebellar tracts or pressure upon the bulbar nuclei from contiguous new growths.

Past Pointing.—Bárány has shown that past pointing and spontaneous deviations are frequent symptoms found in cerebellar disease.[2]

Magnus Reflexes.—The studies of Goldstein and Riese, Hoff and Schilder, Rothfeld, Zingerle, Kroll and others have shown that cerebellar disease is of considerable importance for the Magnus reflexes. These latter are naturally involved in all of the proprioceptive, labyrinthine and cortical adaptations, but their description by Magnus will tend to reorient neurological phraseology regarding the generalization of synergia. The investigation of the Magnus reflexes is of primary importance in all disturbances, particularly of automatic associated movements. The subject is too complicated for further text-book presentation.[3]

Cerebellar Tremor.—This varies considerably and is mingled with the asynergic movements. In the few small muscles of the hand it shows as a fine oscillating tremor. In writing this tremor is often very characteristic.[4]

Chief Syndromes.—In discussing disorders of the cerebellum it is convenient to take up first affections of the peduncles, although very rare, then of the cerebellum itself, and finally diseases of extracerebellar location, where pressure-producing growths occupying the posterior fossa implicate the cerebellar mechanisms, and those of its contiguous structures, the pons, medulla, and fourth ventricle.

Inferior Cerebellar Peduncle.—Corpus Restiforme.—Isolated disease of this structure is rare. Pressure due to bulbar and pontine disease is

[1] British Med. Jour., November 4, 1897; Reprint, Brain, 1906, p. 425.

[2] Friesner: Loc. cit. Löwenstein: Ztschr. f. d. ges. Neurol. u. Psychiat., 1918, **24**, 534. Wodak: Der Bárányesche Zeigeversuch, Berlin, 1927.

[3] See Magnus (Körperstellung, loc. cit.; Kroll: Ztschr. f. d. ges. Neurol. u. Psychiat., 1927, **110**, 729) for recent literature on cerebellar disease and Magnus reflexes.

[4] Schuster: Ztschr. f. d. ges. Neurol. u. Psychiat., 1924, **91**, 531.

not uncommon. The most characteristic symptoms are modifications of the eye movements, convergent and divergent strabismus, even skew deviation, forced positions, turning of body toward the site of lesion, and vertigo, with tendency to fall in the direction of the side of the lesion. This is closely related to the Babinski-Nageotte syndrome.[1] The chief mechanism involved is the cerebello-vestibulo-spinal tract.[2] (See Midbrain Lesions.)

Lesions in the Middle Cerebellar Peduncle.—These cause rolling movements of the body on its vertical axis, skew deviation of the eyes, Magendie-Hertwig syndrome, consisting in one eye being higher than its mate. The patients behave as though they had bilateral vestibular disease, causing the rolling motions; the eye symptoms depend on lesions of the fasciculus antero-marginalis, fibers to the posterior longitudinal bundle, and interference with the fibers to the abducens nucleus. There are few uncomplicated cases on record.[3]

Pontine lesions often give rise to cerebellar symptoms from implication of the middle peduncle. They are not infrequent.[4] Thiers has described an interesting series of this type of case under the title of cerebellar hemorrhage, variety cordonale. Three types of syndromes have been isolated: (1) A cerebello-thalamic type with sensations of heat, cold, formication and hemianopsia; (2) cerebello-ocular type with third nerve palsy of side opposite to the cerebellar hemisyndromy. The cerebral peduncles, third nerve nucleus and the red nucleus is implicated.[5] (3) Cerebello-pyramidal type with dysarthria. Here there is a hemicerebellar syndrome with pyramidal tract signs. (See Midbrain Syndromes.)

Lesions in the Superior Cerebellar Peduncles.—Isolated lesions of these peduncles are rare. Porot has described an isolated hemorrhage. The symptoms are usually choreic, or paralysis agitans-like tremors on the same side of the lesion—possibly due to implication of the cerebello-rubro-spinal bundle in the tractus cerebello-tegmenti. Forced positions of the head to the side of the lesion have been described. Ocular implications rarely occur, although nystagmus has been observed. (See Midbrain Lesions.)

Lesions within Cerebellum Itself.—The most important of these are ageneses or aplasias, scleroses or atrophies, hemorrhage, softening, inflammation, abscesses, and tumors.

Aplasias of Cerebellum.—These are congenital, and represent a vast array of different conditions; total lack of cerebellum, absence of the lateral (in old sense) lobes, absence of vermis, unilateral loss, irregular defects, and general congenital smallness of the cerebellum

[1] Claude et Lévy Valensi: In Gilbert and Carnots, Traité de med., 1922, **32**, 1. André Thomas: Étude sur les blessures du cervelet, 1918.

[2] Adler: Die Symptomatologie der Kleinhirnerkrankungen, Wiesbaden, 1899, has collected the cases. Claude and Lejonne: Rev. Neur., 1911, **2**, 15.

[3] Babinski and Jumentié, Soc. de Neur., January 2, 1911.

[4] Marie et Foix: Soc. de Neur., July 4, 1912. Sem. med., January 8, 1913; March 26, 1913. Thiers: Thèse de Paris, 1915.

[5] Claude et Loyez: Rev. Neur., July 15, 1912.

and cerebrum. A consistent symptomatological grouping is not yet possible. Mingazzini[1] has attempted it. His grouping of the conditions is as follows:

1. Pure unilateral ageneses and atrophies.
2. Pure bilateral ageneses and atrophies.
3. Cerebellar atrophies associated with
 (*a*) Disease of the cerebrum.
 (*b*) Disease of the spinal cord.

1. *Unilateral loss* of a lateral (in old sense) lobe may be present without any symptoms according to present developed modes of testing. Few of these cases have been tested by more recent methods. In certain instances unilateral atrophy has been associated with epileptiform convulsions, or retropulsion, when the cortex of the affected hemisphere is atrophic; if the atrophy involves a part of the vermis, slight motor signs, such as slowing of the gait, have been observed.

2. *Pure Bilateral Agenesis.*—In some of the cases reported no symptoms have been observed (old cases). The commonest symptoms are difficulty in standing and walking. The patient, in high grades of atrophy or aplasia, is unable to stand, or sometimes even to sit; in the milder grades the station is wobbly, the feet placed far apart, and walking is possible only with assistance. The gait is then the classical drunken stagger. There is marked asynergia of the trunk and lower extremities. Tremors, ataxias, incoördination (asynergias) of the upper extremities are also present. Hypotonus, muscular weakness, slow, irregular, hesitating or explosive speech are also present. Nystagmus may or may not be present, there is usually adiadokokinesis, the knee-jerks are usually normal, or even slightly exaggerated at times, even in the presence of hypotonus. Bilateral atrophies show similar symptoms.

Combined Aplasias of the Cerebellum and Brain.—Combettes' (old period) patient, with absolute absence of the cerebellum had from birth epileptiform attacks, was able to walk but fell often. Many of these patients are idiotic and imbecile, and show symptoms similar to those enumerated in the previous paragraph.

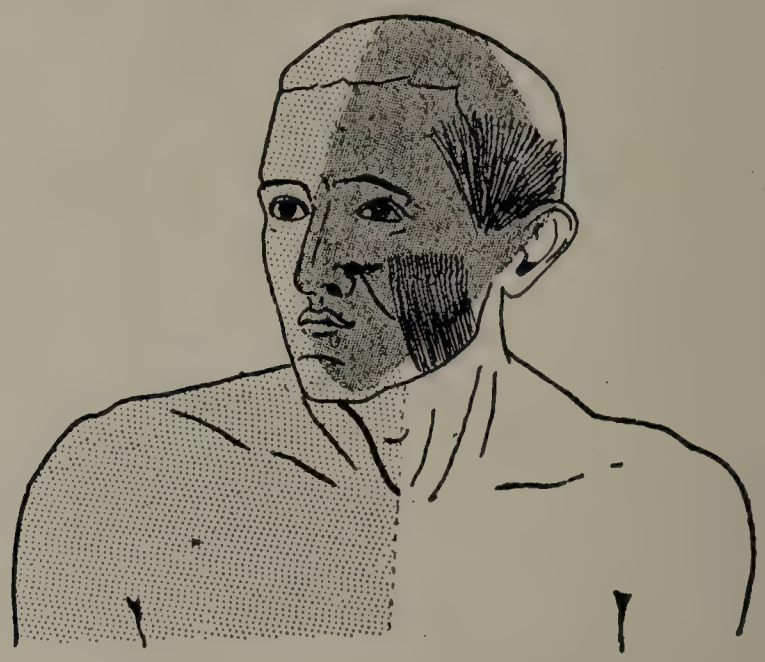
Mingazzini includes the olivo-ponto-cerebellar atrophies here, but these are discussed later. Holmes[2] calls these cases "congenital smallness of the central nervous system, with cerebellar symptoms."

A number of conditions may be grouped here. Some of Marie's so-called hereditary cerebellar ataxias are best referred here. Irregular staggering gait, Romberg, disorder of speech, nystagmus, and ataxias of limbs are the chief symptoms. These patients have shown small cerebellums with apparently intact tracts in cerebellum and cord. Claude's recent discussion may be consulted to advantage.

[1] Monatschr. f. Neur. u. Psych., 1906, **18,** 76. See Brun: Schw. Arch. f. Neur. u. Psych., vol. **1, 2, 3,** for complete modern discussion. Also see Mingazzini e Gianulli. Real Accad. l. Lincei., Rom. San. 1918, **12,** fasc. 13.

[2] Brain, 1907, p. 546.

Olivo-ponto-cerebellar Atrophy.—This type was described by Thomas and shows a fairly definite syndrome.[1] Anatomically there is atrophy



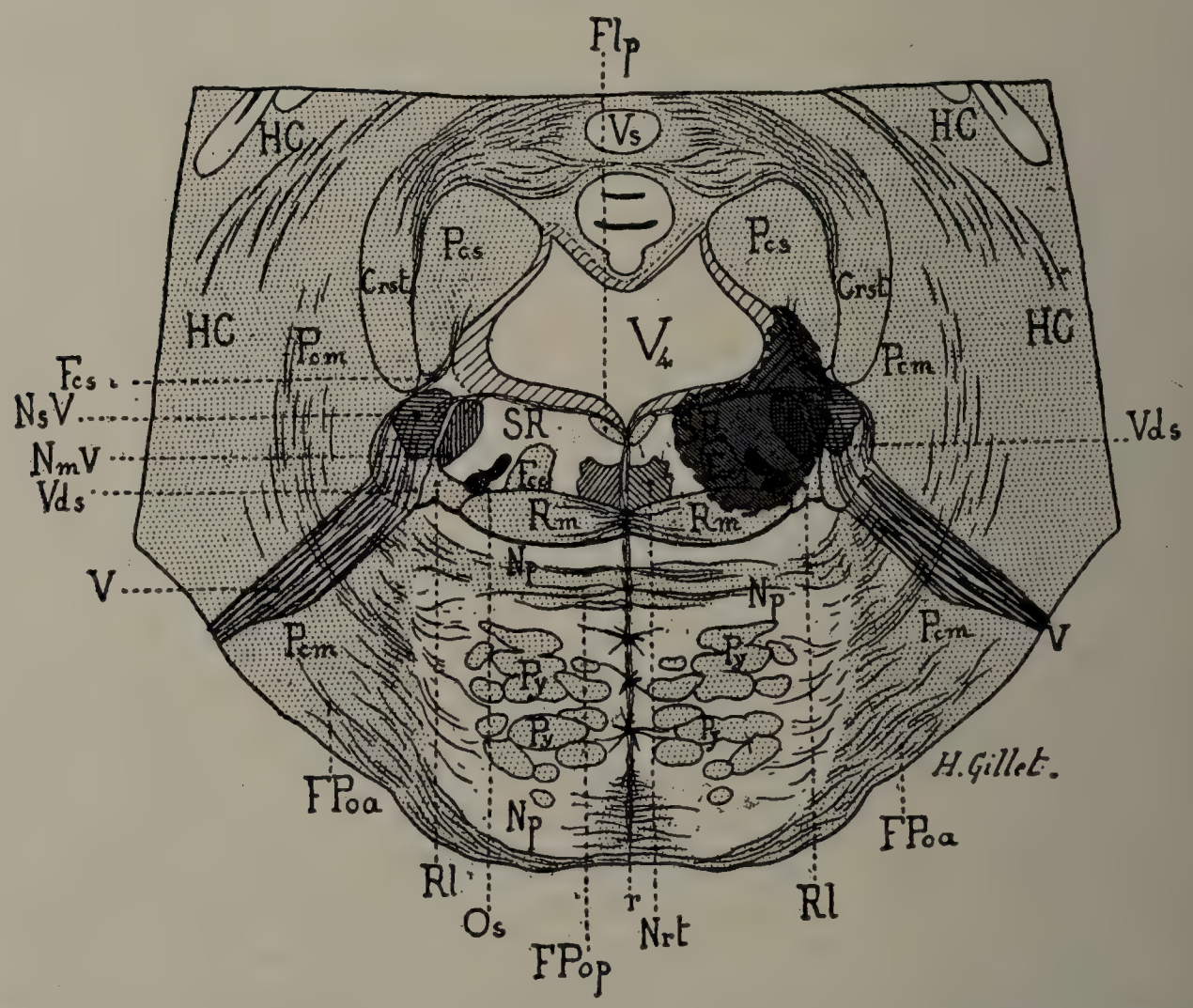


FIG. 342.—Posterolateral pontine syndrome. Lesion of the lateral part of the left pontine tegmentum, involving the nuclei of the trigeminus, the crossed secondary sensory paths of the tegmentum, partially involving the superior cerebellar peduncle (*Pcs*) and the median lemniscus (*Rm*), and not involving the anterior portion of the pons.

On the right there is hemianesthesia of the extremities of the syringomyelitic type, above all for pain and temperature sense.

On the left there is paralysis of the muscles of mastication (pterygoid, masseter, temporal) by lesion of the motor nucleus of the trigeminus. There is slight anesthesia in the trigeminus region (sensory nucleus *V*) and choreo-athetoid movements of the extremities from involvement of the superior cerebellar peduncle. (Dejerine.)

[1] Dejerine and Thomas: Nouv. iconog. de la Salpêtière, 1900, p. 350. Lejonne and Lhermitte: l'Encéphale, 1909, No. 6. Taft and Morse: Jour. Nerv. and Ment. Dis., September, 1914.

of the cerebellar cortex, of the bulbar olive, and of the gray matter of the pons. There is total degeneration of the middle cerebellar peduncles, partial degeneration of the inferior cerebellar peduncles, and a relative integrity of the cerebellar nuclei. It is not necessarily hereditary, familial nor congenital. It comes on at an advanced age and progresses slowly. Clinically there is great defect in equilibration in standing and walking—drunken gait. Romberg is absent. Some irregular intention tremor, usually nystagmus, and also scanning speech is present.

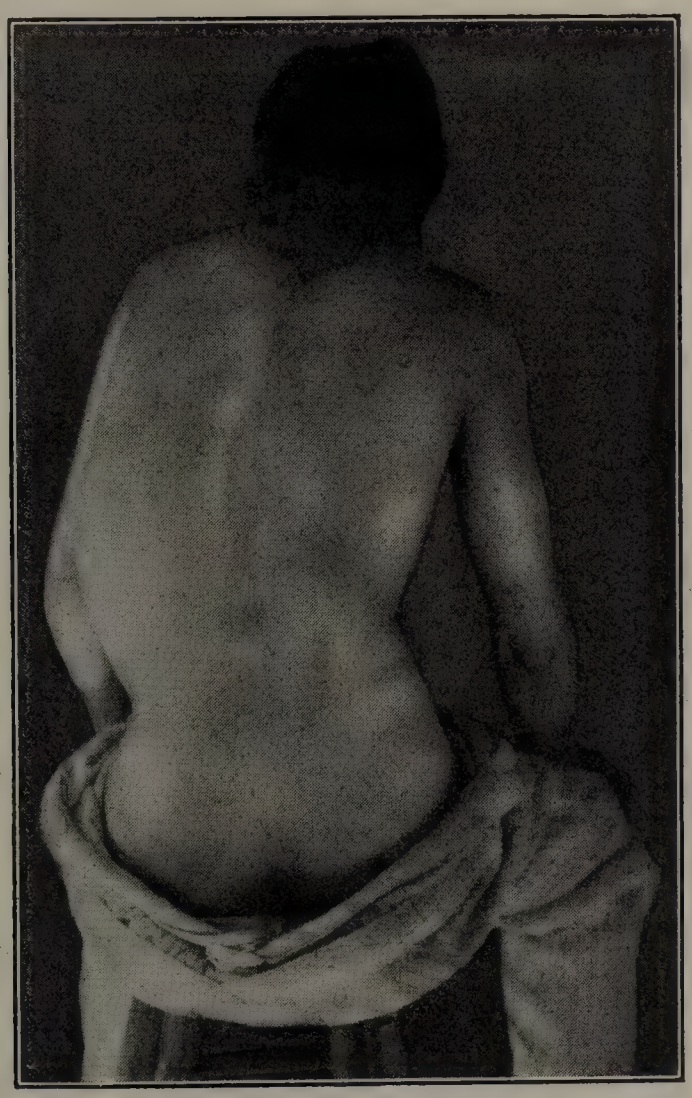


FIG. 343.—Scoliosis of Friedreich's ataxia.

Cerebellar and Cord Atrophies or Chiefly Hereditary Aplasias.[1]—Here also clinically a motley group is on record. These cases will vary greatly in proportion to the varying degree of the lesion in the cerebellum and in the cord. Some authors are disposed to refer Friedreich's disease to this group also, and to claim that between these two disorders one finds every gradation from the Friedreich type, with mostly cord and little cerebellar change, to Marie's cerebellar atrophies, with more cerebellar and less cord change. This, it must be emphasized, is the clinical aspect of the situation. From an etiological viewpoint, more important, but still very difficult of analysis, the

[1] Holmes: Brain, 1907, p. 466; Loc. cit., for literature. Thomas: Loc. cit. Kappers: Jour. Nerv. and Ment. Dis., 1927, **65**, 113.

hereditary uniformity of the Friedreich group and the possibly less uniform Marie group must be distinguished.[1]

The chief signs here[2] are ataxias of the leg, arm, facial, ocular, head, laryngeal and pharyngeal muscles. Cerebellar reeling, no Romberg. Later palsies and contractions. Exaggerated tendon phenomena. In most of these cases the disorder is predominantly in the cord.

In other cases with degeneration of the spino-cerebellar tracts with normal or only small cerebellum one finds patients with staggering gait, scanning, explosive, slow speech, nystagmoid jerkings of the eyes, muscular cramps, fatigability of muscles, normal or exaggerated knee-jerks. As mentioned, Friedreich's disease properly belongs to this rubric.



FIG. 344.—Friedreich's ataxia, showing the permanent extension of the great toe.

Primary Parenchymatous Degeneration.—Holmes[3] has described this condition. It usually sets in about middle age and progresses slowly. Staggering or reeling gait is an early symptom, then asynergia of the upper extremities, and later hesitating, scanning, or explosive articulation, nystagmus, tremor of the head and limbs. Tendon reflexes are normal or slightly exaggerated. No clonus, no Babinski. Sphincters intact and normal psyche. Most of the cases have shown a familial character.

[1] See Harnhart's discussion of these types. Schweiz. med. Wchnschr. 1923; Schweiz Arch. f. Neurol. u. Psychiat., vol. **13**.

[2] Brown: Brain, 1892, **15**, 250.

[3] Brain, 1907, p. 466.

Cerebellar Tumors.[1]—Cerebellar tumors are relatively frequent. Almost one-third of the brain tumors occurring in childhood and adolescence are of cerebellar origin. They may be divided into: (1) Primary and (2) secondary tumors. Among the primary tumors one can distinguish: (*a*) Cystic and vascular tumors; (*b*) hard tumors.

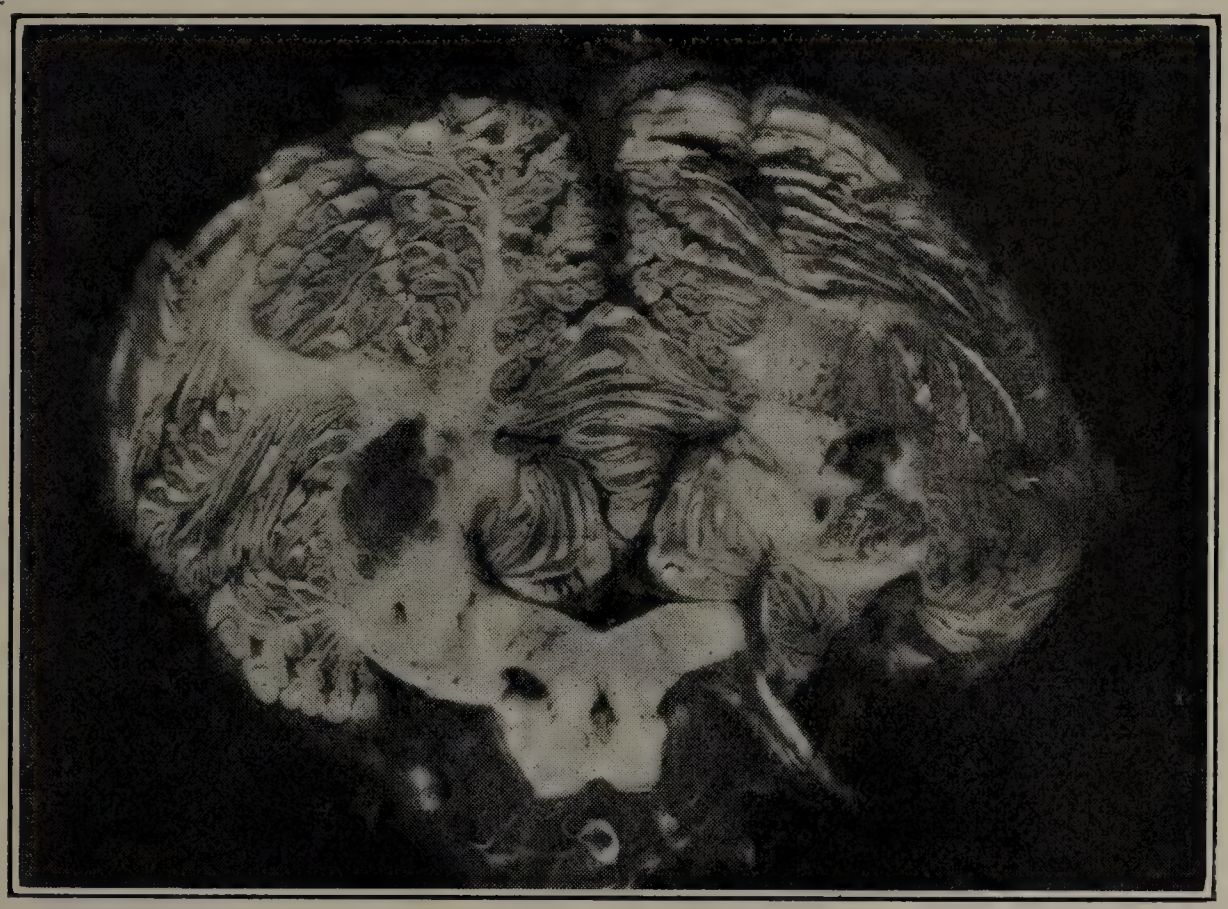


FIG. 345.—Bilateral softening of the cerebellum. (Schaller.)

(*a*) Cysts and cystic tumors are not always distinguishable. Together they form a small part of tumor formations in the cerebellum (5 to 10 per cent.). The symptoms of cerebellar cysts are practically identical with those of cerebellar tumor, but the operative outlook is much better.[2]

(*b*) The hard tumors consist of gliomas (70 per cent.), sarcoma, psammoma, lipomas, myxomas, cholesteatomas, endotheliomas. The secondary tumors are chiefly tuberculous or syphilitic.

The chief general symptoms of cerebellar tumors are headache, usually severe, occipital or frontal, and apt to be confined to a sagittal plane; papilledema and later optic atrophy, which is rapidly progressive; vomiting, vertigo, and tenderness to percussion over the occiput. Tumors in the hemispheres, not involving the central tracts or the intrinsic nuclei, may give rise to no localizing symptoms. But there is usually an implication of these contiguous structures with added symptoms.

These are the classical cerebellar syndromes of gait, and attitude,

[1] Stewart and Holmes: Brain, 1904, **27**, 522. Claude and Lévy Valensi: Loc. cit., 1922. Thomas: Loc. cit. Balduzzi, C.: Tumors and *X*-rays, Ztschr. f. d. ges. Neurol. and Psychiat., 1926, **106**, 653. McKendree: Arch. Neurol. and Psychiat., 1927, **11**, 44

[2] Williamson: Rev. Neurol. and Psychiat., March, 1910, for literature.

asynergia, ataxia and adiadokokinesis of the same side, with hypotonia of special character already described, and motor paresis of the same side. To these are usually added nystagmus and eye deviations already noted. The nystagmus is apt to be pronounced only on looking to the affected side, and is usually slower and coarser than the nystagmus of labyrinthine origin, or of involvement of the vestibular tracts. As these latter are frequently impinged upon, it is useless to insist upon too fine distinctions in the character of nystagmus. Unless the pyramidal tracts are influenced by pressure, the arm and leg tendon reflexes are not markedly exaggerated, nor are they lost, and the Babinski, Oppenheim, Schaefer and Remak signs of pyramidal tract involvement are not present. The abdominal reflexes are usually unmodified.



FIG. 346.—Tumor of cerebellum. (Goodhart.)

Finger-pointing tests are of special value in localization in the initial stages.[1]

As the tumors increase in size there are added symptoms due to encroachments or pressure upon contiguous structures. These are usually the signs of involvement of the pyramidal tracts, eye palsies, and of the cranial nerves—from the fifth to the twelfth. These all show on the opposite side of the body. The two lower branches of the facial are involved, the tongue protrudes to the paralyzed side and is without atrophy or R. D. Occasionally the medulla is pressed upon and one finds all branches of the facial involved with atrophy of the tongue. Homolateral anesthesia of the cornea may be present, due to trigeminus involvement. Homolateral affections of the ears, deafness, buzzing and homolateral pain to pressure on the mastoid may aid in diagnosis. Percussion should never be neglected. Oppenheim has called attention to the cracked-pot sound often present in cerebellar tumors.

Unilateral signs are apt to pass over into bilateral signs as the pressure increases, with dysarthria, dysphagia, continued vomiting, and finally cardiac and respiratory signs.

[1] Rothmann: Neur. Centralblatt, 1914, No. 1.

Lumbar puncture may give important information in clearing up a diagnosis of meningitis-serosa or hydrocephalus.

Dyssynergia Cerebellaris Progressiva—Chronic Progressive Cerebellar Tremor.[1]—As dyssynergia cerebellaris progressiva, Ramsay Hunt has, described a chronic progressive tremor disturbance of cerebellar origin.

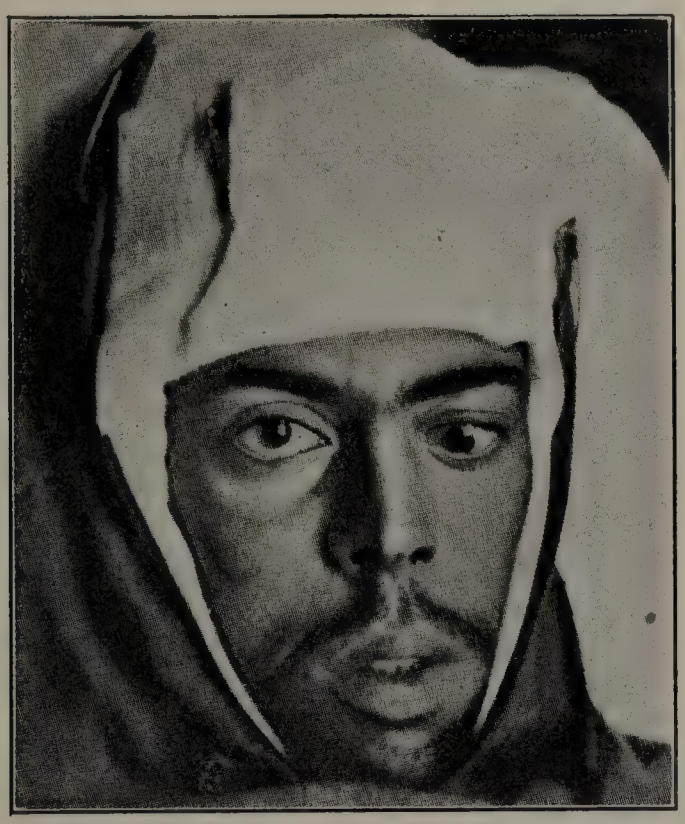


Fig. 347.—"Skew deviation" after removal of a tumor from the left lateral lobe of the cerebellum; the left eye is directed downward and inward, the right eye upward and outward. (Holmes and Stewart.)

This affection is characterized by generalized intention tremors, which begin as a local manifestation and then gradually involve in varying degree the entire voluntary muscular system. The tremor which is extreme when the muscles are in action, ceases entirely during relaxation and rest. This disorder of motility is associated with disturbances of muscle tone and of the ability to measure, direct and associate muscular movements; the clinical manifestations of which are *dyssynergia, dysmetria, hypotonia, adiadokokinesis* and *asthenia.* All of these symptoms, including the volitional tremor, which is an extreme expression of the underlying disturbance of muscle tone and synergy, indicate a disorder of midbrain and cerebellar mechanisms.

These cases are further distinguished by the absence of true nystagmus, objective vertigo cerebellar fits, vestibular seizures and disturbances of equilibrium, symptoms which are so frequently associated with gross lesions of the cerebellum.

Wounds of the Cerebellum.—The World War has increased greatly the possibilities of study of cerebellar function. These studies are too numerous to be here reviewed. A syndrome of irritation due to cerebellar substance loss has been described by Goldstein and Reich-

[1] Brain, 1914, vol. **36.**

mann[1] and several important contributions have been made by Thomas,[2] Levi[3] and others. Goldstein and Reichmann's irritation syndrome is characterized by overvaluation of weights, of spatial and of time relations, by increased sense of heaviness and of pseudospontaneous movements. Deviation of finger test with conservation of cerebellar center stimulation and increase of labyrinthine deviation. There is adiadokokinesis, excess of Holmes-Stewart contraction test and increased pressure and muscle sensation.

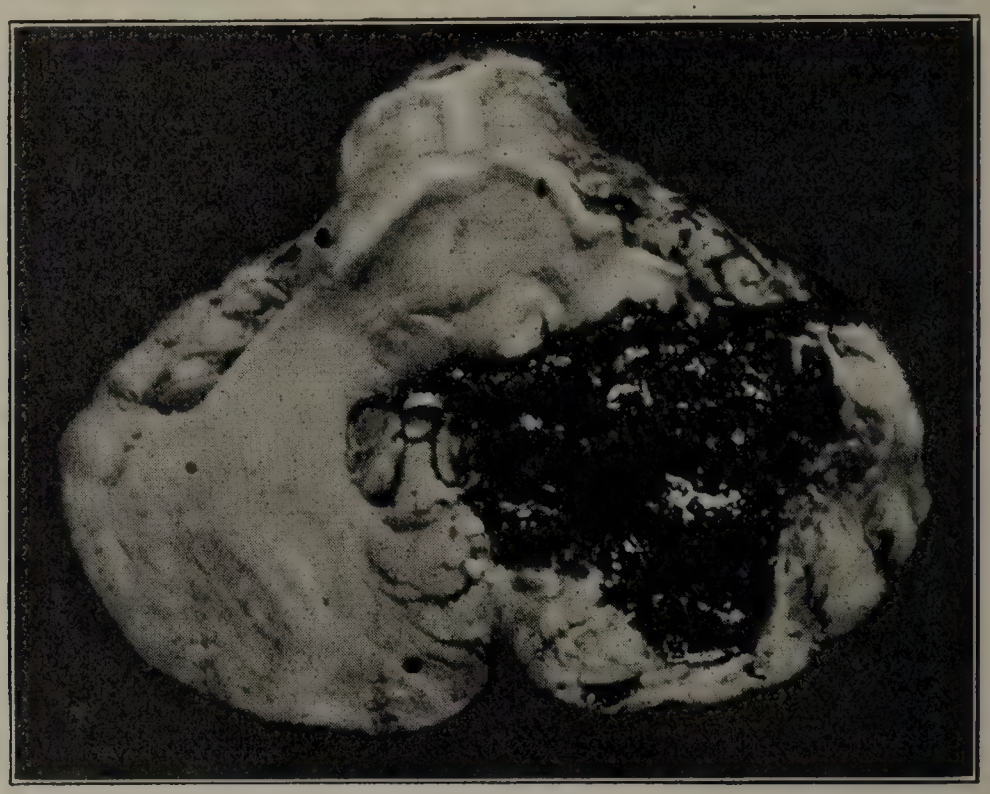


FIG. 348.—Hemorrhage of cerebellum. (Larkin and Jelliffe.)

Hemorrhage of Cerebellum.[4]—Spontaneous cerebellar hemorrhage is extremely rare. Hemorrhage from wounds of war have been comparatively frequent in the world war. Its symptomatology will depend largely on its size and the location of the effusion. The superior cerebellar artery is oftenest involved, thus implicating certain portions of the dentate nuclei. Extension into the fourth ventricle is to be borne in mind.

The onset of the symptoms is usually sudden, or preceded by pain in the back of the head, with slight giddiness, or forced position of the head. There is usually marked vertigo, recurring on attempts to move, and usually persisting in non-fatal cases. Cerebellar gait, forced position of head, depending on location, nystagmus, especially on lateral movements. Operative interference is generally useless in the spontaneous types. War wounds may be localized according to the principles laid down by Bolk and Bárány (*loc. cit.*) especially when

[1] Arch. f. Psychiatrie, 1916, lii, 2: Ibid., 1917, vol. **57.**

[2] Etudes sur les blessures du cervelet, 1918.

[3] Bull. Acad. de méd., May 8, 1917, No. 19. Claude et Lhermitte: Soc. med. d. Hôp., May 26, 1916.

[4] Starr: Med. Rec., May 2, 1906. Thomas: Loc. cit.

made by smaller missiles and after the acute symptoms have subsided. The finger-pointing tests are especially valuable in localization.[1]

Cerebellar Abscess.—These are relatively frequent, and originate mainly from middle-ear infections, either by way of the temporal lobes or the mastoid, and wounds, from trauma, which latter may have occurred a long time previously.[2] Occasionally abscess may result from thrombi due to abscess of the lungs, ulcerative endocarditis, etc.

These abscesses vary considerably in size from that of a pea to an apple, and their development is either acute or chronic.

The characteristic symptoms are headache, usually occipital, and radiating into the neck region, producing marked stiffness of the neck, at times resembling the pain of a cerebrospinal meningitis. General unrest, nausea, vomiting, and stupor are present. Hyperthermia may be added, but a cerebellar abscess may run a course of several months without temperature. Of special cerebellar symptoms ataxia, nystagmus and rotatory vertigo are characteristic. This vertigo is usually rendered worse and vomiting is induced by movement of the body; hemiparesis and hemiasynergia are usually present on the side of the lesion, but these are not constant signs.[3]

By an increase in the size of the abscess, symptoms of pressure, precisely similar to those mentioned under Tumor, may develop. Papilledema, with optic atrophy, is not infrequent in large abscesses.

Brain puncture by special aspiration needles is advisable to clear up the diagnosis in complicated cases. Lumbar puncture is useful in excluding meningitis.

Associated Posterior Fossa Complications.[4]—*Occipital Lobes.*—These may be pressed upon antero-superiorly by a foreign body growing upon or within the superior lobe of the cerebellum. Hemianopsia and mind-blindness may then occur.

Corpora quadrigemina.—Ocular palsies of a nuclear character, occur, first on one side, then upon the other. The oculomotor and obducens are oftener involved than the trochlear. There is less apt to be a paralysis of accommodation or changes in the pupils, and the loss of conjugate motion of the eye is rarely found. Implication of the posterior corpus, and of the middle geniculate causes deafness, usually bilateral, and if the lateral geniculate be pressed upon, amblyopia, without papilledema. Pineal tumors may cause the same picture.

Cerebral Peduncles.—If these structures are markedly impinged upon, the Weber-Gubler syndrome of alternate hemiplegia and oculomotor palsy may be found. From milder irritative pressures one obtains the Benedict syndrome, oculomotor palsy, with tremor of the

[1] See Jones: Equilibrium and Vertigo, loc. cit. Stiefler: Ztschr. f. d. ges. Neur. u. Psych., 1915, 24. Friedländer: Neur. Centralblatt, November 21, 1915. Bauer: Wien. kl. Wchnschr., 1916, 29.

[2] Combier: Thèse de Paris, 1911.

[3] Thomas: Soc. de Neur., January 8, 1914.

[4] See paragraphs on cerebellar peduncles.

opposite side. If the lemniscus is impaired there is crossed anesthesia and ataxia. (See Midbrain Syndromes.)

Pons.—Here one finds a number of syndromes added to the initial cerebellar symptoms. Crossed hemiplegia with facial palsy (Millard-Gubler) and R. D. Occasionally from a more anterior pressure there may be homolateral facial palsy without R. D.

Crossed Hemiplegia and Abducens Palsy.—Both are usually associated with hypoglossal involvements. Conjugate deviations to the opposite side of the lesion are observed in these cases when the abducens nucleus is involved. Such conjugate palsies speak more for tumors within the pons (*q. v.*). (See Midbrain Syndromes.)



Fig. 349.—Tumor of fourth ventricle bringing pressure on vestibular nuclei, causing nystagmus. (Schaller.)

Crossed hemiplegia and trigeminus involvement are occasionally found, and also alternate hemiplegias with cochlearis symptoms. Here the hearing defect is due to destruction of the intrapontine fibers of the cochlearis; or to pressure on the tuberculum acousticum.

Tumors of the Fourth Ventricle.—These may be considered here because of the cerebellar symptoms induced. These tumors are for the most part glioma, sarcoma, psammoma, carcinoma. They give rise to symptoms due to pressure on the medulla and pons and almost always cause a marked hydrocephalus with advancing stupor and confusion. Cysticercus may also be found here. Bruns has called attention to the following features: Alternating periodicity of headache, nausea, vomiting, vertigo, changes in pulse and breathing, with sudden let-up of all these symptoms. The vertigo and vomiting are set up by changes in position of the head; sudden movement of

the head may cause immediate unconsciousness. Cerebellar ataxia, mild nystagmus and occasionally diplopia are other signs.

One more word may be said about cerebellar tumors and their diagnosis. They may, in the presence of few signs only, be mistaken for affections of the frontal lobes (fronto-cerebellar paths), parietal lobes (implications of central sensory components) and of the optic thalamus.

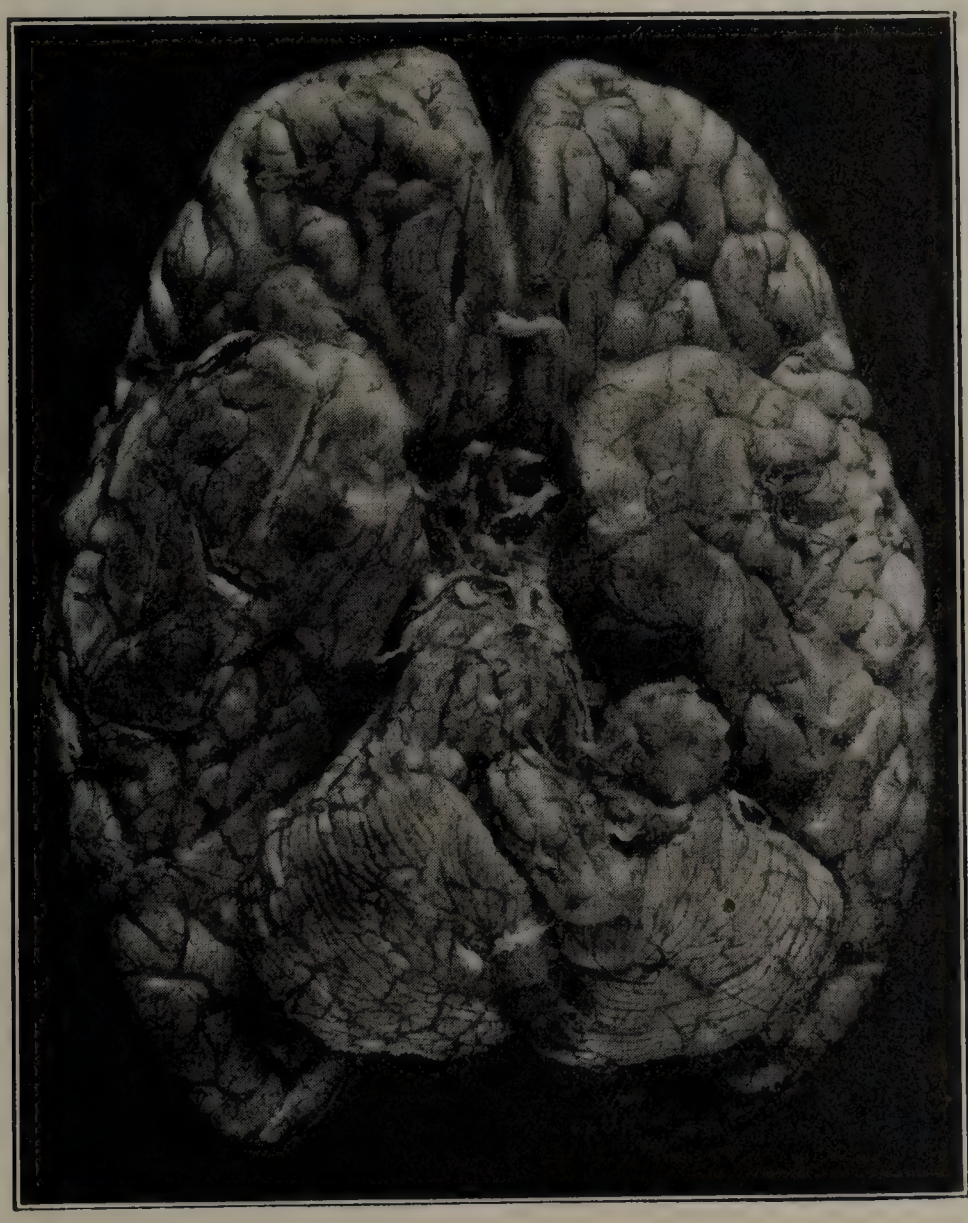


FIG. 350.—Tumor (neurofibroma) of cerebellopontine angle. (Larkin and Jelliffe.)

In frontal lobe tumors special intelligence defects are usually found, if carefully examined for by the methods of Ziehen, Sommer, and Kraepelin. The tremor is apt to be very fine and rapid, hemiparesis, if present, is crossed, and shows spastic phenomena; the speech disturbance is aphemic; the conjugate deviations are irritative and not paralytic. Then anosmia, apraxia, witzelsucht and aphasia are often added. Skew deviations, and hypotonus are not known for frontal tumors.

Parietal lobe disease only occasionally offers difficulties, while the characteristic sensory disturbances and central pains of optic thalamus involvement should exclude this structure.

Cerebello-pontine Angle Tumors.[1]—These should be discussed here because of the symptoms of cerebellar pressure and of vestibular involvement. Two main types of tumor come under review. Those from the pia of the cerebellum, and those growing on or about the acoustic nerve. Fibromata, myomata, and sarcomata are the most frequent.

These tumors press upon the pons and middle cerebellar peduncle and the symptoms vary slightly, according to the variations in pressure on these two structures. The eighth nerve is usually involved early; buzzing and deafness are observed. Facial palsy is usual with corneal anesthesia from pressure on the fifth; trigeminal pains are frequent. Ptosis may appear. Pressure on the cerebellum causes the typical gait and the ataxia, homolateral paresis, and hypotonus. A contralateral paresis from pressure on the pyramidal tracts usually develops. This gives the usual signs of a pyramidal tract involvement. Homolateral static tremor is frequent, also a sense of subjective rotation toward the side of the lesion.

Treatment of Cerebellar Conditions.—Gummata must be attacked by the usual antisyphilitic treatment, otherwise surgery offers the only opportunity, and this is limited to the attack upon cysts which may give fortunate results. The results of operations for abscess are improving, as are also those for tumors. But as each case is a law unto itself, it is futile to generalize.

[1] Henschen, F.: Ueber Geschwülste der hinteren Schädelgrube im besonderen des Kleinhirnbrückenwinkels, 1911, for full literature to 1911; also English literature. Stewart and Holmes: Brain, 1904. Weisenberg: Jour. Am. Med. Assn., 1908. Starr: Jour. Nerv. and Ment. Dis., 1910. Lewandowsky: Handbuch der Neurologie. Cushing: Tumors of the Acoustic, 1918. Hermann and Troplan: Ztschr. f. d. ges. Neurol and Psychiat., 1924, **93**, 528. Schaller: Arch. Neurol. and Psychiat., 1927, **17**, 609.

CHAPTER X.

INJURY AND DISEASES OF THE MENINGES. FRACTURE OF THE SKULL, ETC.

HERE diseases of the dura, the arachnoid, and the pia are to be recognized. Under the first, various types of meningeal hemorrhage and inflammation—pachymeningitis—are found. Under the latter—various forms of leptomeningitis and cerebrospinal meningitis.

FRACTURE OF THE SKULL: MENINGEAL APOPLEXY.

1. **Meningeal Apoplexy** (*Traumatic Meningeal Hemorrhage. Fracture of the Skull*).—**Etiology.**—Trauma from blows, instruments, violence of various kinds, causes either a rupture of the branches of the middle meningeal artery, the veins, or of the dural sinuses. Such hemorrhages may be found at the site of the injury, or at the point opposite.[1]

The hemorrhage may be extradural, between the pia and dura, epidural, or between the pia and cortex, arachnoidal hemorrhage The bleeding may be diffuse or circumscribed. At childbirth such hemorrhages with partial destruction of the brain substance itself are extremely frequent. Here the bleeding is almost always exclusively venous.

Pathology.—Macroscopically such hemorrhages resemble hemorrhagic pachymeningitis, but microscopically they differ, especially in the absence of new elements—vessels, plasma cells, etc.

Not infrequently in skull fractures the brain substance is also involved.

Symptoms.—*Fracture of Vault.*—These will vary according to the mechanism of the producing lesion, the presence or absence of a communicating wound, the form of the fragments and the location. Bursting, bending, simple, compound, fragmented or perforating fractures are the types met with in war neurosurgery. Basal fractures are apt to be linear, cortex fractures are comminuted with displaced fragments and much laceration. In severe injuries there are signs of shock and concussion in addition to the symptoms of pressure. Unconsciousness becomes increasingly deeper, the pulse is slow in the beginning, then becomes rapid, vomiting takes place, urination and defecation are involuntary, irregular respiration, with increasing temperature, and death often results unless operation relieves the pressure.

[1] A. Mayer: Zentbl. f. Gyn., 1915, No. 46. Cushing: Keen's Surgery, 1917. British Jour. Surg., 1918. Guillain: Presse méd., September 5, 1918, for meningeal hemorrhages in war. Nouvean Traité, Roger *et al*, 1927.

In less severe lesions the initial symptoms of concussion with varying grades of stupor partially clear up for an hour or more, even twenty-four, or a few days.[1] Then compression symptoms develop which Kocher has divided into four phases: (1) Stage of compensation compression through the cerebrospinal fluid shifting. Headache, mental dulness and slight focal signs characterize this stage. Then (2) increased headache, vertigo, irritability, restlessness, blanching of cerebral veins, it may be, with delirium. Stage (3) shows increasing compression when the pulse commences to slow down, the splanchnic vessels are constricted and there is is a rise in blood-pressure. The respiration is stertorous and coma is present with dilated pupils, it may be, and loss of all reflexes. With increasing compression (4) and death

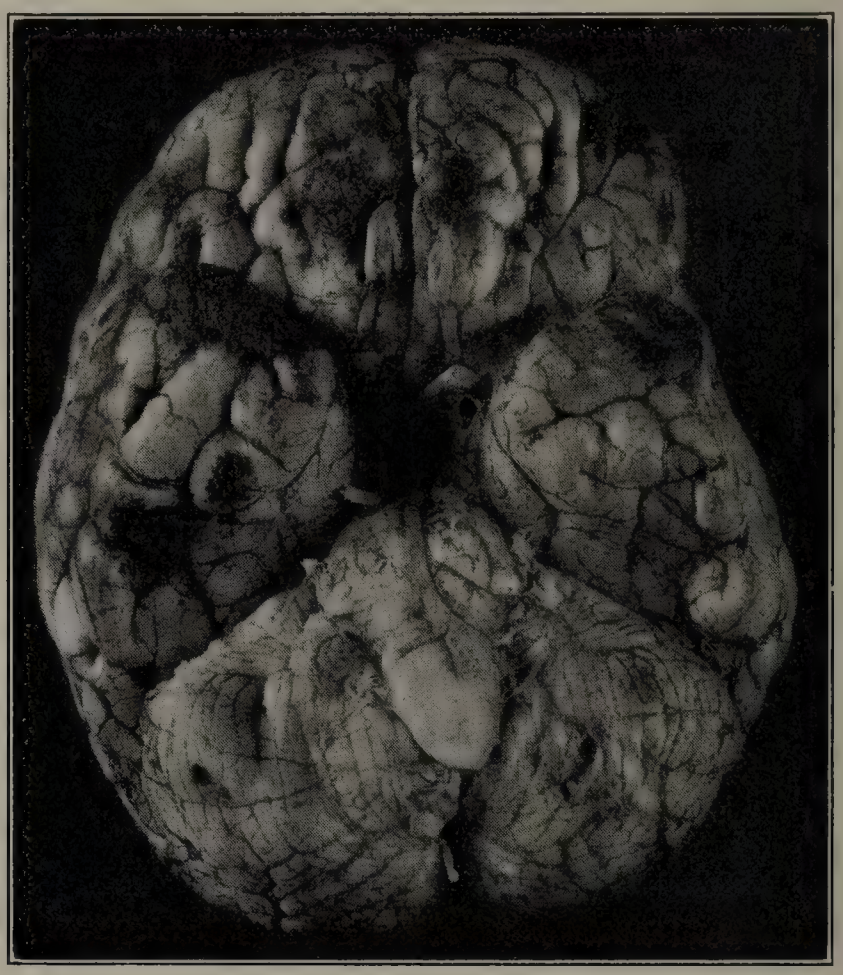


FIG. 351.—Pia-arachnoid hemorrhage from contra coup. (Larkin and Jelliffe.)

there is circulatory failure, rapid pulse, deep stupor and decreasing blood-pressure. In most head injuries there is an initial lowering of the temperature followed by a rise, the height of which is more or less in proportion to the severity of the head injury. High temperatures seem more associated with contusions than with simple hemorrhage. Temperatures of 104° F. and over are ominous. The focal symptoms, depending on location, are of considerable importance. There may be spasms, epileptiform convulsions, often of Jacksonian type, monoplegias, hemiplegias, the arm often suffering the worst. The hemiplegia is usually on the side opposite to the site of injury, but in about 5 per cent. of the cases is found on the side of the lesion (uncrossed

[1] Connell: Free Interval in Meningeal Hemorrhages, Surg., Gyn. and Obstet., March, 1906.

pyramids or contra coup). Aphasias are not infrequent in left-sided injuries. The tendon reflexes are usually increased on the paretic side, while the skin reflexes are usually diminished. The Babinski phenomenon is frequent on the paralyzed side, and occasionally present on the side of the hematoma. Occasionally hemianesthesia and hemianopsia can be made out. Guillain calls attention to the slight biliary discoloration of the skin as a special sign in severe meningeal hemorrhages from war shocks, with or without cranial trauma.

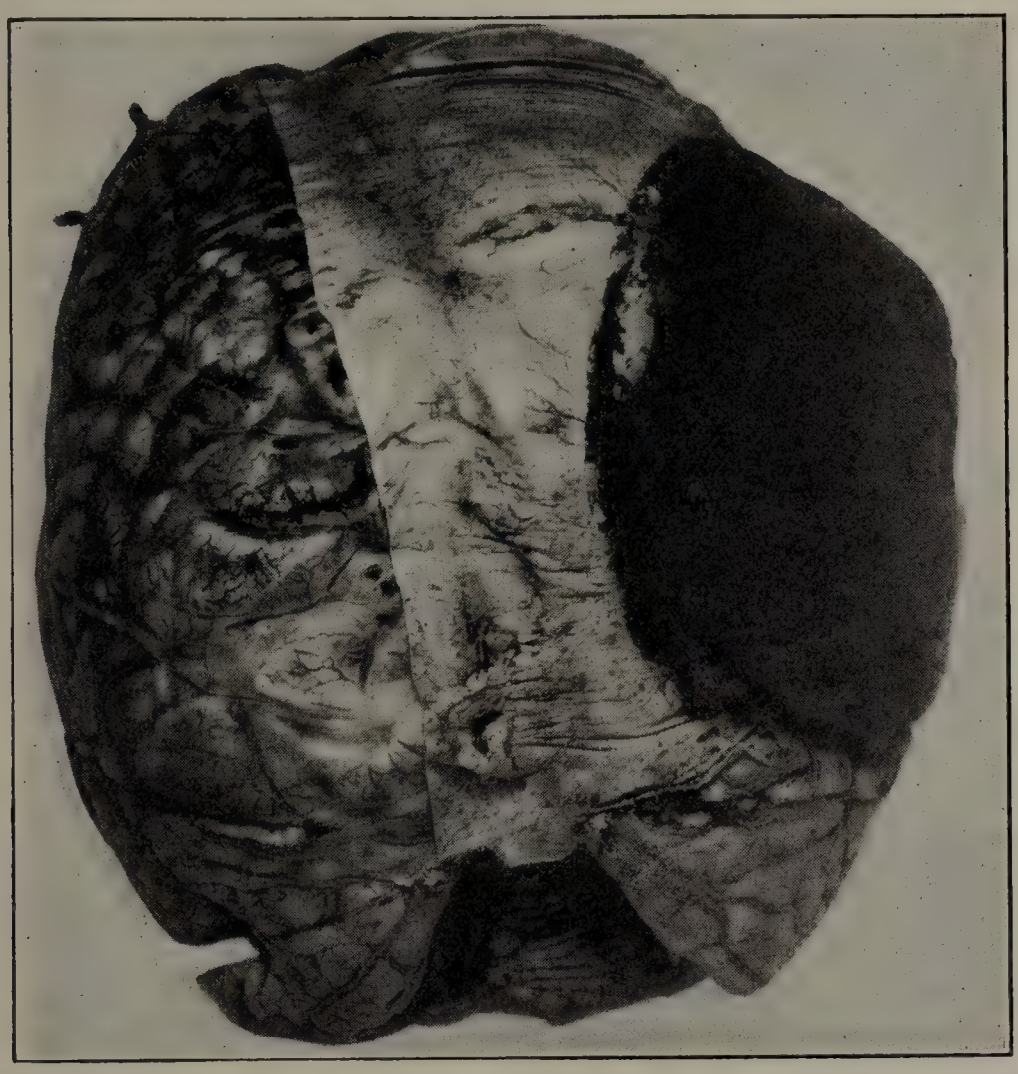


FIG. 352.—Traumatic supradural hemorrhage. (Larkin and Jelliffe.)

In severe meningeal hemorrhages from brain injuries or compression the pupils vary greatly. Wiesmann has shown in 70 cases, that in 39 both pupils were dilated and immobile, in 20 there was dilatation on the side of the hemorrhage, in 7 both pupils were small, and in 4 there was dilatation on the side opposite the lesion. The great war studies show great variability in the pupillary symptoms. They are so variable as to be of small diagnostic value for purposes of localization. In about one-half the cases it may be said that the larger pupil corresponds to the greater intracerebral pressure Widely dilated and non-reacting pupils offer a sinister prognosis. The fundus findings are of great value in determining intracerebral pressure especially in old cases, less so in acute ones, as they rarely develop within twenty-four hours.[1]

[1] Menninger: The Pupils in Coma, *loc. cit.*, Optic Nerve.

Fracture of Base.—Basal fractures may simply show the signs of concussion, prolonged coma, with gradual recovery or death. Not infrequently there is bleeding from the ears or nose, or there may be subconjunctival ecchymoses. Cranial nerve injury is not an infrequent accompanying symptom. It may be the auditory, or the sixth, or the facial. At times all three may be involved. The resulting and resultant clinical pictures vary greatly. When the fracture injures the auditory and facial, facial palsy and deafness are present in varying degrees. The facial palsy usually clears up. The deafness remains. Taste modifications also occur. In rare instances a vagus, facial and acoustic syndrome may develop.[1]

The war brought into prominence a number of severe skull injuries with basal fractures and nerve injuries. They cannot even be summarized here. Some have been discussed in the chapters on the cranial nerves. Among the various nerve injuries were the glossopharyngeus, apart from the vagus, where it is separated by the inferior petrosal sinus. Vernet's curtain sign here is seen. (See Cranial Nerves.) The war injuries also seemed to show that the accessory or eleventh nerve is a caudal extension of the vagus. Cervical sympathetic fibers were frequently injured. Lesion of the sixth nerve caused the syndrome of Gradenigo, originally described as a diplopia developing after middle ear suppuration (mastoid). Paralysis of the pharynx, larynx and shoulder have been observed in war basal fractures. Epileptic seizures are special complicating possibilities.[2] Late results of lasting importance should never be lost sight of.[3]

The syndrome of the foramen lacerum posterior is one that has come into prominence from war injuries. It was described by Vernet in 1916. There was loss of voice, difficulty in swallowing solids, cough, excessive saliva and mucus. No paralysis of tongue. The soft palate was paralyzed so that the uvula appeared deviated to the left and in phonation and swallowing the palate was drawn upward and to the left. Nasal regurgitation took place and a nasal twang was imparted to the voice, with hoarseness; a vocal cord being paralyzed. Choking sensation of right side of the soft palate, pharynx and larynx. Taste diminished to quinine. Back of affected side and facial, auditory and accessory unchanged.

A combined posterior lacerate or jugular and condyloid syndrome as an extension of Vernet's syndrome is described also. Bullet wounds in the right temporo-maxillary region just below the ear cause it. Lower facial, glossopharyngeal, pneumogastric, bulbar roots of accessory and hypoglossal are implicated. Impaired taste, cough, salivation, hyperthesia of soft palate. Pain in side of throat and shortness

[1] Ulrich: Schweiz. med. Wchnschr., 1921, No. 21. Stookey: Jour. Nerv. and Ment. Dis., June, 1923, No. 6.

[2] Reichmann: Ueber Entstehung und Haufigkeit epileptischer Krämpfe nach Schädelbrüchen an der Hand 603 Fällen, Deut. zt. Nerv., 1927, **96**, 260.

[3] Stone and Brams: Ultimate Results in Severe Craniocerebral Injury, Jour. Am. Med. Assn., 1927, **88**, 1056. Hall: The Relation of Psychiatic and Neurotic Disturbances to Head Injury, Illinois Med. Jour., October, 1925.

of breath are present. Also paresis of soft palate and superior constrictor, thick voice and paralyzed vocal cord. Paralysis right half of tongue.

A retroparotid space syndrome is also added to the list by Villaret. It is to be distinguished from the syndromes of Avellis, Schmidt, Hughlings Jackson and Tapia. (See Midbrain Syndromes.) There is hemiparesis of the glossopharyngeal, the pneumogastric, the accessory, the hypoglossal and cervical sympathetic.

A number of other complicated skull base war injuries have been described, but they must be sought in special discussion.[1]

Birth Hemorrhages.—In birth hemorrhages, Seitz has shown that subtentorial hemorrhages behave differently from convexity hemorrhages. In the former the children frequently show no sign of asphyxia, then after a few hours respiratory disturbances set in, the breathing becomes irregularly quickened, with spasms and cyanosis. Then spasms of the eyeballs occur, less often facial spasm. If the pressure is directed downward toward the medulla, opisthotonos and muscular rigidity develop, and not infrequently priapism.

In convexity hemorrhages the child shows considerable restlessness, refuses to suckle, and then develops signs of brain-pressure, pressure in the fontanelles, respiratory disturbances, drowsiness to unconsciousness, with some slowing of the pulse. Localizing symptoms may then show themselves, spasms of the opposite arm and leg, increased tendon reflexes, and slight hypertonus. In children the late results bring about various syndromes. When the hemorrhage and destruction occupies the motor areas alone, the type of Little's disease is present. Slight variations in localization of the hemorrhage and destruction bring about variant forms of Little's disease with sensory involvement. Little's complete syndrome *when present at birth* is usually due to other antecedent degenerative factors. Cerebellar localizations bring about anomalous cerebellar diplegic types.[2] According to the localization of the hemorrhage and to antecedent developmental arrests the following cerebral types of *birth palsy* may be distinguished:

1. Atonic types with mental defect—frontal lobe.
2. Spastic types—Little's disease, motor area (antecedent fetal degenerations necessary).
3. Sensory types—speech defects, posterior central.
4. Pseudobulbar palsies.
5. Cerebellar diplegic types (Batten, Clark, L. P.).

Diagnosis.—In birth palsies it may be extremely difficult to determine whether one has to deal solely with a pure meningeal apoplexy, or whether there is also intracerebral disturbance. If there is a definite free interval after the initial signs of concussion, with the gradual or sudden onset of compression signs, the probabilities are for

[1] See Medical Science Abstracts, 1921, p. 35. Vernet: Paris méd., 1916 and 1917. Villaret and Faure: Beaulieu, Presse méd., 1918, **26**, 591.

[2] Clark, L. P.: Jour. Nerv. and Ment. Dis., 1915; Trans. Am. Neurol. Soc., 1916. Hunt, J. R.: Loc. cit. Batten: Brain, 1913. Anglade and Jacquin, 1909.

hematoma (80 per cent). The length of free interval offers no certain criterion as to site of hemorrhage. Choked disk, often transitory, also speaks for hematoma. Bloody spinal fluid, which does not clot, speaks for intradural hemorrhage, occasionally extradural. Neisser's brain puncture and lumbar procedures often help in clearing up a diagnosis. Lung intervals speak for abscess. The presence of Little's syndrome (1861) at birth is evidence that something more than a meningeal hemorrhage[1] has occurred. Some developmental intra-uterine factor or factors (heredity, endocrine, syphilis, etc.) are operative.[2]

The diagnosis of war fractures is usually obvious. Roentgen ray for type of fracture is imperative. In a certain percentage of cases sugar appears in the urine. Lumbar puncture offers many important diagnostic signs in hidden fractures of the base.



FIG. 353.—Before operation. FIG. 354.—After operation.
FIGS. 353 and 354.—Little's disease. Spastic diplegic type from motor area destruction. Posterior root operation. (Kamamura.[3])

Therapy.—This is surgical for vault and comminuted fractures and should be immediate. The exact procedure must be determined largely by the symptoms. The prognosis is apt to be good if the patient lives forty-eight hours, those dying after this usually succumbing to abscess or meningitis. The general prognosis is bad, about one-third fatal. Even the intracranial hemorrhages of childbirth may be controlled by skilled surgical measures. The results of surgical interference are three times as good as leaving the patients alone.

In the late cases surgical section of the proper posterior roots may be of great service in overcoming the spastic states. (Figs. 353 and 354).

[1] McNutt, 1885.
[2] Freud, 1897. Collier: Brain, 1924, **47**, 11. Sachs and McCarrison: Lancet, 1908.
[3] Surg., Gynec. and Obst., 1918, **27**, 131.

Traumatic late apoplexy is a special variety in which degenerative processes complicate the picture. The patients may develop the signs of hemorrhage, usually intracranial, several hours or days after the injury (larvated apoplexy.) Such cases are to be diagnosed with much caution. Senility, pronounced arteriosclerosis, and syphilis should be excluded.[1]

2. **Inflammation of Dura** (*Pachymeningitis*).—(*A*) **Pachymeningitis Externa.**—Following severe trauma, purulent processes of the frontal sinuses, the middle ear, mastoid, erysipelas, caries, or osteomyelitis, one occasionally observes an inflammation of the external surface of the dura of the cerebrum. It is usually localized. A similar process may be present in the spinal dura.[2]

Symptoms.—These are usually hidden in those of the causative lesion and vary with the acuteness and extent of the process. Localized cerebral or spinal pains, muscular twitching, spasms, marked scalp tenderness to percussion, and slight rise in temperature are the chief signs. At times symptoms of localized pressure of the cerebrum or of the cord, resembling tumor, are observed.

Treatment.—The treatment is that of the causative factors.

(*B*) **Pachymeningitis Interna Simplex.**—This may consist of a localized or circumscribed purulent exudate, giving in the main the symptoms of a brain abscess or a brain tumor. In rare instances (pneumonia, etc.), a secondary pseudomembranous productive inflammation occurs.

(*C*) **Pachymeningitis Interna Hemorrhagica.**—This consists of a chronic congestion in the internal layers of the dura, associated with repeated small hemorrhages and the organization of a new membrane which tends to extend and thicken. It is by no means infrequent[3] and gives rise to chronic psychoses of an illy defined type.

History.—Morgagni noted the affection in the eighteenth century. Baillarger[4] followed the older authors in assuming it to be a primary hemorrhage, with new membrane tissue formation. Heschl and Virchow[5] first pointed out the inflammatory nature of the productive inflammation, and the consequent hemorrhage due to the rich formation of new bloodvessels. Jores and modern authors support these views.

Occurrence, Pathology.—Usually a disorder of advanced years, hemorrhagic pachymeningitis may be found in children.[6]

It is extremely frequent in general paresis, and Blackburn has

[1] Delayed apoplexy, see: Stadelmann: Deutsch. med. Wchnschr., 1903. Allen: Jour. Nerv. and Ment. Dis., 1909. Auvray: Bull. de l'Acad. méd., 1926, **96**, 401. Schmidt: Arch. klin. Chir., 1927, **142**, 475. Singer: Loc. cit.; Ztschr. f. d. ges. Neurol. and Psychiat., 1922, **75**, 127.

[2] Pincoffs: Trans. Am. Phys., 1926, **41**, 247.

[3] Blackburn: Government Hosp. Autop. Repts. Cathala and Wolf: Bull. Soc. Pediat., 1926, **24**, 321.

[4] Maladies du Cerveau, 1854.

[5] Würzburger Verhandlungen, 1856, p. 134.

[6] Foppert: Jahrb. f. Kinderh., 1905, **61**, 51. Freund: Monatsschr. f. Kinderh., 1909, vol. **7**.

found it accompanying chronic excited and chronic depressed states, in patients running a course resembling senile dementia. It is frequently an alcoholic complication. It is seen in some chronic choreic affections. Tuberculosis, nephritis, leukemia, scorbutus, Barlow's disease, and hemophilia have seemed to stand in causal relations in some instances.

In the initial stages a productive inflammation causes the formation of a thin, delicate, yellowish-brown membrane on the inner surface of the dura. The base is rarely affected, the temporal regions more often. New bloodvessels form, then their walls give way, giving rise to extravasations of blood. The process of new membrane formation and bleeding goes on hand in hand until the whole membrane may be one or more centimeters thick, compressing the brain, with which it is usually closely united by new connective-tissue formation, and new bloodvessels, and causing atrophies, degenerations, softening, or scleroses of the near-lying portions of the cortex.[1] The process may extend to the spinal cord.

Symptoms.—The disorder may be present for years without symptoms. In paresis it may not add any definite symptoms to the underlying disorder, or it may cause a number of complicating pressure pictures. When the process has advanced to a definite extent both general and local symptoms show but are so indefinite as to defy diagnosis. Gradually increasing head discomfort, headache, often severe, some nausea or vomiting, irregular periods of confusion or distress in the head, with at times mild delirious excitement, are among the more characteristic earlier signs.

With rapid extravasations, acute pressure symptoms may develop, with epileptiform convulsions, hemiparesis, comatose states with slow pulse, aphasias, irregularities in gait of ataxic, diplegic nature. It is very characteristic that these may be transitory. Persistent monoplegias, with Jacksonian attacks involving irregularly distributed muscle groups may be early signs. Often persisting stereotyped muscle movements, chewing, automatic arm, hand, or leg movements may be the signs of a local irritative lesion. Constantly putting the hands to the head was considered suggestive of pachymeningitis by Fürstner. It is a frequent sign in paresis. The eye muscles are rarely involved (conjugate deviations, nystagmus), and still more rarely one finds involvement of the cranial nerves at the base.

Papillitis, or choked disk, is often present. The pupils are not infrequently irregular, occasionally myotic in the early stage, dilated in coma, and at times immobile to light and accommodation. Argyll-Robertson may be present with or without positive Wassermann. Increased tendon reflexes show the presence of irritation of the motor cortex, clonus and the Babinski reflex being occasionally present, and at times coming and going. Irregular but inconstant temperature changes are often present.

[1] Jores and Laurent: Ziegler's Beiträge, vol. **29**. Putnam: Jour. Nerv. and Ment. Dis., 1927, **65**, 260.

Course.—This varies considerably, is usually chronic, shows remissions and exacerbations. At times the patient recovers completely; again the disorder is progressive and may bring about death after a long psychotic period of irregular excitement or depression.

Diagnosis.—It must be separated as a primary and as a secondary process in alcoholism, paresis, cerebrospinal syphilis, etc. Brain tumor, abscess, hydrocephalus, sinus thrombosis, leptomeningitis, apoplexy are to be borne in mind. Traumatic meningitis must be excluded if an antecedent trauma, even of mild grade, such as falling from the bed, in bath tub, hard crack on the head from running into door, etc., should have occurred.

Neisser's procedure of brain puncture is often desirable in those patients that give signs of brain tumor, brain abscess, etc.

Therapy.—In acute progressions, local bleeding is advisable. Hydrotherapy with stimulation of elimination is useful. Brain puncture has been of service, also lumbar puncture in children. Abstinence from alcohol is imperative. Mercury in the positive Wassermann cases is indicated. The pains are often relieved by analgesics, and by warm baths.

DISEASES OF THE ARACHNOID AND PIA. LEPTOMENINGITIS.

1. **Acute Leptomeningitides** (*Cerebrospinal Meningitides*).—The studies of recent years particularly those which have come from the camps during the mobilization of troops have shown a host of causes for acute inflammation of the cerebral and spinal arachnoid and pia. These vary very widely as to severity—simple meningism to the gravest forms of general meningeal involvement—epidemic cerebrospinal meningitis, epidemic polio-myelo-encephalitis, general syphilitic meningomyelitis, etc. It becomes impossible to present a logical classification of these disorders, either from the etiological, pathological, or clinical standpoint. In general only the more circumscribed types of meningitis will be considered here.[1]

Causes.—These are many. Trauma is one of the most important and the meningitides from skull and brain wounds were common during the war 1914–1918. Such trauma may act either through direct infection, as by compound fracture, or secondarily bring about a septic meningitis, through hemorrhage, thrombosis, and subsequent infection.

Traumatic early and late meningitides appear. Many tuberculous leptomeningitides arise from trauma. Occasionally one meets with late purulent meningitides, due to old encapsulated abscesses, old projectiles (bullets, splinters, etc.), old fractures of lamina cribrosa.

Infection from suppurative processes in the neighborhood is one of the most frequent causes of the type of meningitides under con-

[1] Valuable monographs are by Schultz (Nothnagel) and by Finkelnburg (Handbuch der Neurologie, **1**, 1090, in both of which complete bibliographies are to be found. Also see Southard in White and Jelliffe: Modern Treatment of Nervous and Mental Diseases, 1915, vol. **1**. See Quart. Cumulative Med. Index for enormous literature.

sideration, and chief of these is otitic meningitis. Suppurative otitis may lead to intra- or extracranial abscess, sinus phlebitis, thrombosis, direct infection, infection through labyrinth, through mastoid, etc. Serous meningitis may also have an otitic origin.

Nasal and frontal sinus infection gives rise to a small number of these meningitides; they may be purulent or serous. Operations upon the nose may be complicated by meningitis.

Facial erysipelas occasionally is a cause; rarely facial furunculosis.

Specific organisms give rise to specific types of meningitis. Among these are measles, mumps, scarlet fever, varicella, typhoid, gonorrhea, syphilis, pneumonia, diphtheria. The influenza bacillus is an extremely important organism in this respect, often giving rise to severe epidemics of meningitis. Malaria, yellow fever, anthrax, leprosy, actinomycosis, pneumonia, whooping-cough each may be the exciting agents. Septic extension from acute articular rheumatism is a factor. Tuberculosis is a large item. The epidemic type due to the *Diplococcus intracellularis* is one of the most characteristic of the types. Old abscess formations in the lung, liver, bladder, prostate, teeth pockets, etc., all may give rise to a meningitis.

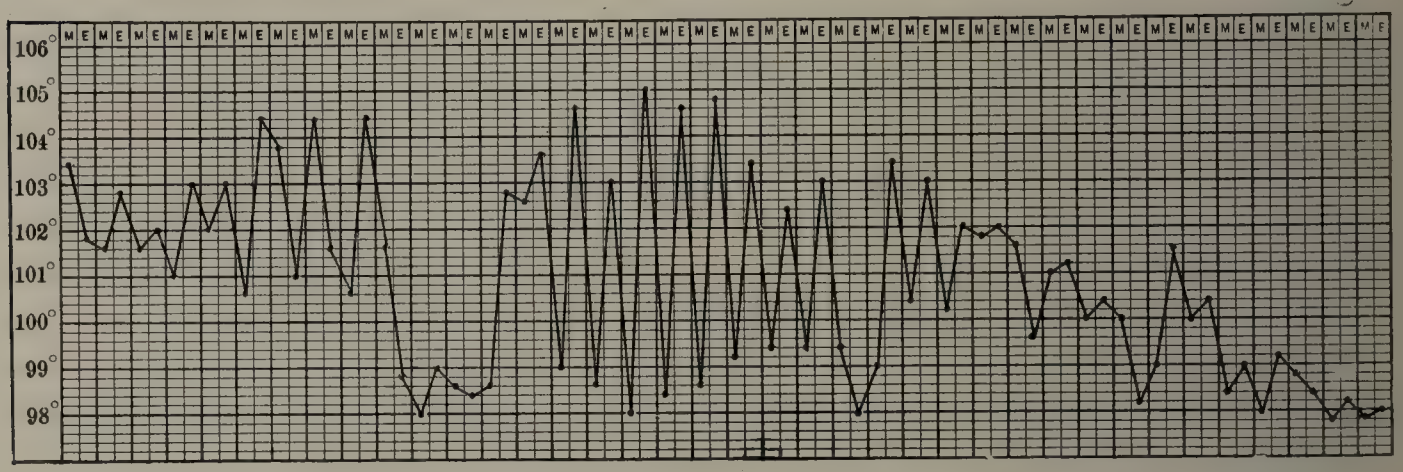


FIG. 355.—Temperature curve showing irregularity and wide excursions. (Osler.)

Occasionally one ascribes certain meningitides to chemical poisons, diabetes, lead, gout, are among these.

Symptoms.—These show certain variations according to the mode of infection and the type of infecting agent. Since simple purulent meningitis, tuberculous meningitis (usually a mixed infection) and epidemic cerebrospinal meningitis are the chief infections, the following description will apply to these and no attempt will be made to cover the symptomatology of the entire group.

Headache.—This is one of the earliest signs, and is frequent, usually increasing in intensity as the disease progresses. It is mostly diffuse, but may first appear in the occipital region, or in the forehead. The slightest movement, percussion, etc., increases it.

Stiffness of Neck.—This is a striking symptom and develops early, sometimes preceding the headache. Such early stiffness may or may not be apparent to the patient, but comes out on careful examination; and is not infrequently accompanied by painful pressure points over

the cervical vertebræ. When well developed the least movement of the neck is painful and the attitude of the patient on movement is striking. In young children it is not infrequently absent, and in adults in chronic cases. It is a further characteristic that this stiffness and painfulness shows considerable variation even during the twenty-four hours.

Hyperesthesia.—The skin, the muscles, the joints, can hardly bear the slightest touch or pressure, and sensitiveness to light and to sound are early signs of meningeal irritation. Occasionally the sense of smell is extremely acute.

Nausea and Vomiting.—These are common initial symptoms (80 per cent.). Occasionally the vomiting persists. Early vertigo is frequent.

Mental Symptoms.—These occur early, particularly in children, and more especially in tuberculous meningitis. The patients become peevish, throw their toys away, are capricious, their attention varies. They not infrequently have mild delirium early, especially at night, or are confused. Very young children, one to three years, often show less mental involvement. Older patients are excited, sleep badly, are restless, mutter in their sleep, or have well-marked delirium. Later a semicoma may gradually develop, with variations in intensity from slight confusion to a confused delirium. Marked ups and downs characterize the epidemic forms, but some degree of confusion or coma is more or less constant.

Temperature.—The patients practically always show a rise in temperature. High temperatures (104° to 106° F.) usually characterize the purulent meningitides; often preceded by chill. Such temperatures may remain high, or not infrequently show remissions. The tuberculous meningitides usually show a lower curve.

Motor Irritation, or Paralytic Phenomena.—Cramps and epileptiform convulsions occur more often in the early stages with children than with adults. They sometimes are very persistent. Circumscribed spasms, Jacksonian in type, are not infrequent. Occasionally there are choreic, athetoid, or tremor movements, which come and go, or are continuous.

Muscular rigidity is an early and persistent sign, showing early in the neck, later in the back, with opisthotonos, or stiff lordosis postures. These muscular rigidities are often subject to considerable fluctuation with the degree of internal or external hydrocephalus. Lumbar puncture often influences them greatly.

Kernig's sign is frequent, and is based upon this generalized muscular rigidity (reflex, spasmodic contraction of rectus femoris).

Paretic signs, monoplegia, or hemiplegia, are less frequently met with, and then more in the late stages. Such indicate localized purulent processes, with abscess formation in the motor area, and are usually accompanied by convulsions. Spinal pareses (paraplegias) are rare.

Speech disturbances, usually cortical, and aphasias are occasionally observed, more particularly in tuberculous meningitides.

Cranial Nerve Signs.—Disturbances of the cranial nerves are among the most characteristic signs in the meningitides. The pupils are often unequal; frequently markedly myotic in the beginning, they show irregular widening later. With increasing coma they usually widen, and react sluggishly or not at all. Convergence reactions are less easily tested, but also show slowness.

Eye palsies are frequent. Mild ptosis, unilateral or bilateral, divergent and convergent strabismus—the abducens is particularly prone to disturbance—and double vision is often present. These eye palsies vary from hour to hour in extent, and in intensity, becoming permanent in the long protracted cases particularly. Protrusion of the eyeball is a rare sign, nystagmus frequent.

Papillitis is very frequent, and early; optic atrophy is common (18 to 25 per cent.). Permanent blindness, however, is rare. The trigeminus is rarely involved.

Facial palsies are very frequent, but are usually incomplete, vary from day to day, and are rarely permanent.

Hearing is frequently affected. Complicating otitis, and labyrinthitis often leave these patients deaf. It is a frequent complication in the second and third weeks of the disease.

The vagus involvement causes pulse and respiratory anomalies which are also influenced by central processes. The pulse is initially hastened in practically all forms, and usually remains rapid in the later stages, save in those forms of less acute progress, notably in tuberculous meningitis, where it is often slow, especially after the second week of the disease (75 per cent.—Heubner). Marked irregularity of the cardiac rhythm is conspicuous.

The respiration is also irregular and Cheyne-Stokes type is frequent in the severe purulent and tuberculous types toward the end.

Reflexes.—The tendon reflexes are usually somewhat exaggerated, particularly in the early stages, but may be missing from the beginning (medullary and ventricular pressure). With the advance of the disease they may be missing. Marked variations and irregularities are to be expected, thus lost knee-jerks may be associated with ankle-clonus and Babinski. This latter is a very frequent early sign and later disappears. It should be remembered that it is normally present in young children up to six to eight months of age, and hence is to be neglected in diagnosis in young children.[1]

Lumbar Puncture.—This is of primary importance. The pressure is initially increased. Later this is not marked as the fluid is purulent. In the ordinary purulent meningitides the fluid is usually cloudy, sometimes only microscopically so, again markedly purulent, in which the specific organisms may be found by proper methods. In tuberculous meningitis the fluid is less apt to be cloudy, especially early, but by proper technic the bacillus is found (33 per cent. early stages, 50 per cent. with pressure signs, 100 per cent. in paralytic stages —Pfaundler).

[1] See Göppert for careful study of reflexes; Klin. Jahr., vol. **15**, 423; Berl. klin. Wchnschr., 1905, 21, 22.

Cytologically[1] one finds that in purulent meningitis there is a preponderance of polynuclear leukocytes, while in tuberculous meningitis the lymphocytes are increased. This is not an absolute rule and there are variations during the course of the disease. The cytological findings should be checked up with the clinical ones.[2]

Irregular Symptoms.—Herpes is not infrequent (75 per cent., save in young children) in the epidemic form, and usually appears within the first week (two to five days). It is most frequent about the nose, lips, and forehead. It does not persist long as a rule. Other skin eruptions are not infrequent, especially the rose-colored spots of the diplococcus types. Eruptions resembling measles, scarlet fever, urticaria are occasional, while erythemas and hemorrhagic spots are rare.

Gastric disturbances are frequent, especially constipation. Diarrhea may be an initial symptom in young children.

Tonsillar and pharyngeal redness and soreness are not infrequent in the epidemic types, and bronchitis is often present in the later stages.

Course and Prognosis.—Whereas most of the types of leptomeningitis show much the same general symptomatology, it is more particularly in their developmental course that the differences appear. These variations may be viewed as fundamentally due to specific differences in the microörganisms in question, although it may be said that in a number of instances the only differences observed by the best clinical observers have been those of the microscopic slide, or the test-tube. Hence it may be stated that under certain circumstances the pathological fact that the patient has a meningitis governs the entire picture and all forms are precisely alike, but in the main it is true that specific variations exist, and should be sought for when facing any particular case of leptomeningitis.

In general, fairly sharp distinctions can be made between (1) the infectious, (2) the epidemic, (3) the tuberculous, and (4) the serous forms.

1. *Infectious Meningitis.*—Here the initial disturbance in the ear, nose, mastoid, frontal sinus, fracture, etc., precede, and its symptoms often hide those of the suppurative meningitis that follows. The onset is usually acute. Headache, local or diffuse, is early. The temperature usually mounts rapidly to 104° F. or 105° F., with initial chill; the pulse and respiration are rapid. There is photophobia and hyperacusis within a few days. The mental signs come on soon. Confusion, somnolence, or coma are frequent, often punctuated by active delirious intervals. Lumbar puncture usually shows a purulent fluid. The tendon reflexes are usually increased and the cranial nerve signs are marked within the first week. No special skin eruptions are noted. Neither tâche cérébrale nor dermographism are marked.

With irregular septic temperature, increasing coma or delirium, increasing signs of local pressure or destruction, convulsions, and

[1] Schönbron: Klin. Vort., 384; Med. Klin., 1906, p. 593.

[2] Consult Rehm. u. Schottmüller's valuable atlas of cerebrospinal fluid findings, 1913.

paralyses the more serious cases end fatally within two to five days in children, or one to two weeks in adults.

Other patients show less grave symptoms, run a subactive course and recover in three to four weeks, but in general the prognosis is unfavorable. Those that get well probably have a circumscribed process.

2. *Epidemic Cerebrospinal Meningitis.*—Known for centuries, first recognized as epidemic in 1805 in Switzerland, in 1806 in Massachusetts, this form has been extensively observed the world over.[1] Its epidemic onset is usually very insidious, and spring and winter are the months of predilection in the north temperate zones. It is now endemic in large cities. Children under ten are more prone to the infection. The exciting agent is the *Meningococcus intracellularis* of Weichselbaum[2] of which a series of types have been isolated varying

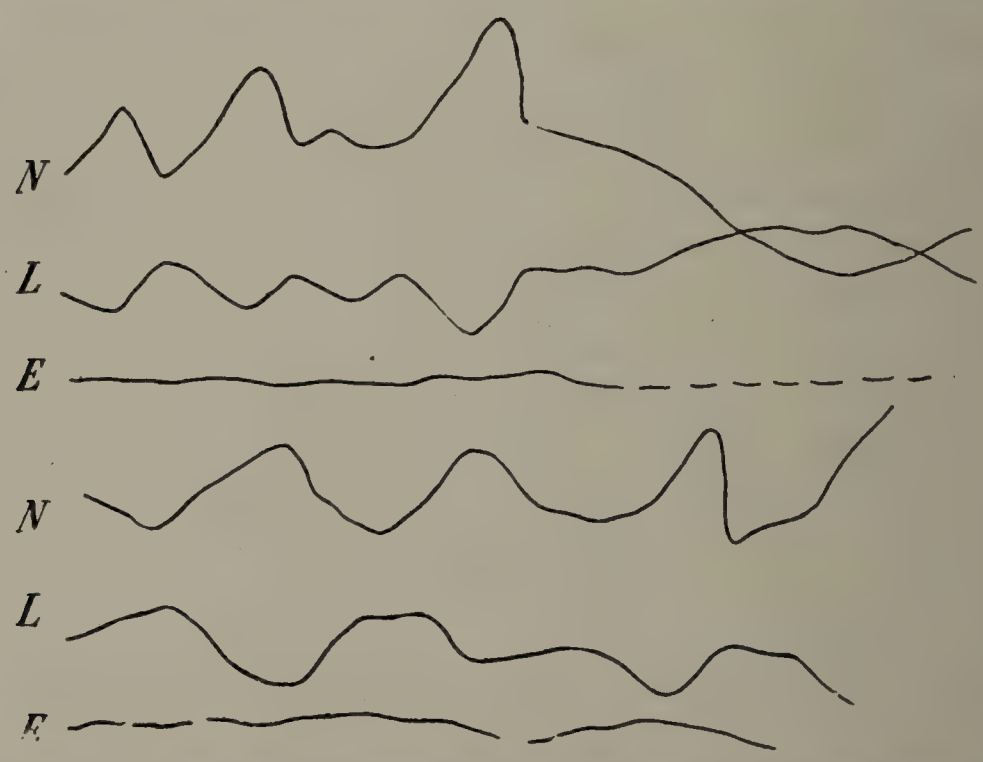


FIG. 356.—Trend of blood curve in cerebrospinal meningitis in favorable and unfavorable cases. *N*, neutrophiles; *L*, lymphocytes; *E*, eosinophiles. (Rusca.)

in their virulence. The disease is contagious, the contagion being possibly carried in the nasopharynx.[3] Abortive, subacute, acute, chronic, and fulminating cases are recognized, with every possible intermediary type. Studies among the armies in Europe and in the United States have contributed very widely to the knowledge of the possibilities of this disorder which has been prevalent in many of the large camps. See Index Medicus, War Supplement, 1914–1917, p. 60.

The malignant or fulminating cases may end fatally in twenty-four hours, with headache, nausea, vomiting, delirium, coma, convulsions,

[1] See Report of State Board of Health, Massachusetts, 1898, for extensive review with literature to that date. Articles of Finklenburg, loc. cit., for later literature and current literature in Jour. Nerv. and Ment. Dis.

[2] Fortschritte d. Med., 1877, p. 622.

[3] See Fildes and Baker: British Med. Jour., October 5, 1918.

stiff neck, high temperature, rapid pulse, Cheyne-Stokes respiration and death from acute toxemia.

Abortive cases, which are more often found in adults than in children, show rudimentary meningeal signs. Headache, backache, nausea and some vomiting with marked stiffness of the neck occur, or the patients may only have slight vertigo, malaise, and nausea, and keep on with their work. Fever is usual and occasionally deafness develops in these mild attacks.

The usual subacute or acute types show a latent period averaging three to five days, consisting of irregular backache, headache, slight vertigo, and sweating, with signs of a nasopharyngitis. Then a chill with slight rise in temperature accompanied by vomiting indicates the onset. Children become restless, cry, and are very irritable. Usually within twenty-four hours meningeal signs appear. Stiff neck, headache, mental confusion, are present; not infrequently convulsions appear in children. Photophobia, hyperacusis, excessive sensitiveness to pressure, and hypertonicity then show themselves with Kernig's sign and increased tendon reflexes. The cranial nerve signs then develop. Herpes labialis is frequent from the second to the fifth day, and a few, macular eruptions (spotted fever) may appear. Other skin eruptions develop in the first week. The cerebrospinal fluid shows a characteristic picture. (See Rehm and Schottmüller.) The blood findings afford some clue as to the prognosis. The leukocytosis is characterized more or less definitely by an increase in the polymorphoneutrophile cells which in the patients with a good prognosis tend to diminish and are accompanied by an increase in the lymphocytes, with a slight increase in eosinophile cells. In the unfavorable cases this crossed curve does not take place and the eosinophile cells disappear entirely.[1]

The coma continues, shows considerable variation in intensity, and the patient gradually improves, or shows increasing signs of pressure, convulsions, palsies, and dies within seven to fourteen days, after ineffectual attempts at maintaining nourishment.

Other patients show a much more chronic course, lasting weeks or months, with considerable variation in the intensity of the headache, coma, or delirium, with eye palsies, mono- or hemiplegia, marked muscular rigidities, which later may leave obstinate rigidities with or without arthritis, constant emaciation, and variable temperature until death takes place, often with increasing size of the head—hydrocephalus and signs of increased cerebral pressure.

In other more favorable cases, periods of clearness or of betterment become longer and more pronounced, intermingled with the severer symptoms already outlined. The variability in clinical course is enormous, but the prognosis, up to the time of the introduction of the Flexner serum, was distinctly unfavorable, the mortality ranging from 50 to 80 per cent. Apparently mild cases often die and some extremely ill children recover. Recovery with defect, either deafness,

[1] Rusca: Deutsch. Arch. f. klin. Med., 1911, 103.

blindness, cranial nerve palsies, monoplegias, hemiplegias, or mental defects, are not infrequent.

Treatment.—Since a specific antimeningitic serum has been elaborated by Flexner[1] the prognosis has been much improved. The mortality has fallen to 25 per cent. in some of the recent epidemics.[2] The best results are obtained in children of from five to ten years.

The effects of serum treatment are often seen very soon—twenty-four hours after injection. The Kernig sign and stiffness of the neck persist, however. The attenuation of the symptoms is very marked in many of the cases, as well as shortening of the disease. Lysis is the usual mode of recovery of non-serum-treated cases. Crises occur much more often in serum treated cases (25 per cent.). Furthermore, the permanent sequelæ of the disease are markedly reduced by the serum treatment. In the meningitides of the camps 1917–1918, polyvalent sera were widely employed to advantage as at least four meningococcus types may be present in differing proportions.

The general treatment will be considered with that of the other forms of meningitis.

3. *Tuberculous Meningitis.*—This form was first definitely isolated about 1830 (Rilliez and Barthez), although one can see its chief features in the writings of Robert Whytt (1768) and in the *Nosologie of Sauvage*, written in 1763. In this form the onset is characteristically subacute or chronic, one or two weeks, exceptionally months, and is almost invariable secondary to tuberculosis in other organs. General symptoms, such as loss of appetite, irritability, loss of flesh, general malaise, with loss of desire to play, fretfulness and ready fatigability are the precursors. Headache and dizziness, with irregular fever movement is then observed, and occasional dreamy states, during which the patient's manner is peculiar.

Then gradually, or suddenly, the patient's manner becomes much changed; mild delirium or coma develops, and convulsions appear. The patients lie in bed, are restless, rolling from side to side with sharp cries or whimpers, and frequent placing of the hand to the head. Passive motions of the head forward invariably bring out resistance and pain. The sensitiveness of the skin to pressure or touch is marked, and hypertonus with muscular twitchings and Kernig's sign are present.

The temperature ranges from 102° F. to 104° F., and is usually less marked than in the suppurative or epidemic types. Lumbar puncture in the first week usually gives a clear fluid under pressure, with exceptionally the tubercle bacillus or pus elements.

The cranial nerve signs may then develop in the second to third week, and show more ups and downs than is usually present in the other types. Localized pressure signs with hemiplegias or monoplegias then develop and, not infrequently, the previously increased tendon reflexes diminish or are lost.

The patients gradually get worse, convulsions are frequent, and

[1] Jour. Exp. Med., 1907.

[2] Flexner: International Clinics, 1909; Jour. Am. Med. Assn., October 30, 1909.

rigidity is followed by flaccidity, and the patient dies with signs of marked exhaustion, sometimes with agonal rise of temperature immediately preceding. The whole attack may terminate within a comparatively short time, two or three weeks, or may run for months.

Atypical forms are especially frequent in adults such as apoplectiform onsets, with aphasia, monoplegia, or hemiplegia. The picture may be that of a toxic delirium. The prognosis is bad.

4. *Serous Meningitis.*—Dietl, in 1846, first isolated this type, which is of uncertain origin, and characterized by varying grades of edematous exudate with infiltration of round cells. Influenza, epidemic encephalitis, are among the causal factors. The symptoms are usually those of a mild meningitis. Headache, stiff neck, marked sensitiveness are constant, whereas convulsions and signs of pressure are less frequent. Again the disorder may be acute and very severe, with high temperature and signs of cranial nerve involvement. The usual course is less stormy Papillitis is a frequent symptom, and should be borne in mind in those patients in whom the serous exudate is more or less circumscribed, giving the general impression of a brain tumor. Lumbar puncture shows increase in pressure with some lymphocytes.

In the serous meningitis of alcoholic origin (*wet brain,* meningoencephalitis), there is usually a busy delirium, excessive hyperesthesia, with marked twitching of the muscles. This type is comparatively infrequent but is seen often in the large cities—Bellevue Hospital service particularly—in all drinkers of long standing in the thirties. It is seen following a very hard drinking spell and often is accompanied by delirium tremens (*q. v.*). The patients become semiconscious, in a muttering, fairly busy delirium with hallucinations of sight and hearing which come and go. There is marked hyperesthesia and the pupils are contracted. The coma is apt to deepen, the temperature rises, the tongue is very heavily brown-coated. Involuntary evacuations may occur. Increasing temperature, 104° F., rapid, irregular and feeble pulse, loss of tone of the skin and muscles all point to a lethal termination. Others slowly improve and recover without or with (Korsakoff) defect.

5. *Syphilitic Meningitis.*—See Syphilis of the Brain.

Differential Diagnosis.—The chief disorders that may be confounded with the meningitides, especially in the beginning of the disorder, or in mild cases, are as follows: Internal pachymeningitis, which rarely gives temperature or lumbar puncture findings. Encephalitis, and encephalomyelitis; in the former localizing symptoms develop early, and the spinal fluid is clear; in the latter flaccid palsies rapidly develop, and the fluid is not purulent. Brain abscess may be complicated by meningitis, or localized, in which latter case the localizing symptoms, septic course, and clear spinal fluid are of aid.

Infectious sinus thrombosis may resemble meningitis very closely. There is a greater tendency for the lower cranial nerves to be involved, particularly the vagus, hypoglossal and spinal accessory. The spinal fluid is usually clear.

Delirium tremens shows an active occupation delirium, optic hallucinations, marked fine tremors and little temperature. Hysteria rarely shows temperature, and many of the organic signs described by earlier authors as found in hysteria are better referred to as organic brain disease with hysterical epiphenomena.

Treatment.—This must first be prophylactic, and involves general hygienic precautions in tuberculosis; prompt aural treatment in otitis, mastoiditis; surgical asepsis of wounds in all head operations. The isolation of the epidemic types is advisable, and nasal antisepsis imperative. Quarantine measures are important and are best controlled through cultures of the nasal and pharyngeal mucus. The active treatment is surgical for most of these forms, especially if the symptoms show early signs of being circumscribed, or when it seems possible to get at an initial focus of infection, as in the various septic infectious forms—ear disease, etc.

In diffuse general meningitis the responsibilities are great, and it is difficult to decide in the individual cases.

Epidemic types are best treated by serum. Surgical treatment of tuberculous meningitis has not yet met with sufficient success to warrant its advocacy.

Lumbar puncture has given very brilliant results in some patients, in others it has been of little service. With the development of acute hydrocephalus it is indicated, and it is in general a harmless procedure. The punctures may be made frequently, and 25 to 40 cc. of fluid removed. The amount removed should be controlled by the pressure. Hess (*l. c.*) speaks of the Rusca leukocyte curve as affording a criterion for lumbar puncture. The drop in eosinophile cells and the failure of the lymphocytes to mount, with increased fever, these are indications for puncture.

Of the sera, the Kolle-Wassermann in septic cases has given good results. Flexner's serum for the epidemic types has been mentioned. In using this serum a lumbar puncture is first made, and from 10 to 50 cc. of fluid allowed to drain off, the amount being determined largely by the pressure. One drop in from three to five seconds is a general rule for determining this. Then from 10 to 50 cc. of the antimeningitic serum is slowly injected by gravity. The serum may be used every three or four days. Control smears of the organisms in the fluid afford some clue to the frequency of use of the serum. With numerous bacteria, active cellular exudates indicate more frequent use of serum. In the alcoholic types, prompt and thorough catharsis should first be used. Diaphoresis by hot packs is of service. Supporting and tonic treatment is then required.

General treatment consists in keeping the patient quiet. Bromides, chloral, veronal and other mild hypnotics are of service. Hypodermic use of morphine or hyoscine may be necessary, but other things being equal, is undesirable. The continuous hot bath is especially valuable in delirious patients.

In the beginning a prompt use of calomel is called for. The room

should be darkened and as quiet as possible. Pain may be controlled as far as possible by analgesics, and local counter-irritants.

Particular attention should be given to the nourishment and rest of the patient. Continuous fussing and overnursing are to be avoided in these cases. There are no specific remedies. Urotropin in large doses may be tried, as it is partly broken down into formaldehyde in the cerebrospinal fluid. The colloidal silver salts are disappointing.

In the long, tedious convalescence of many of these little patients one's ingenuity is taxed to stimulate the appetite, provide the proper amount of outdoor play, and to strengthen the paretic or paralyzed muscles.

2. **Chronic Leptomeningitis.**—This is usually a secondary affection in paresis, senile dementia, chronic lead poisoning, and chronic pachymeningitis; it is rare as a primary affection save as syphilitic or tuberculous.

Chronic tuberculous leptomeningitis is usually of the convexity, generally anterior, involving the frontal and motor areas. It behaves as a low, mild-grade meningitis, often with intermittent circumscribed symptoms, simulating those of a brain tumor.

Traumatic cases often show chronic vertigo, pains, epileptiform convulsions, aphasias, nausea and vomiting, and gradual mental involvement. In order to establish a diagnosis of trauma these signs should stand in direct relationship to the injury, and should not be complicated by signs originating at a distance from the site of the injury or from other causes (Argyll-Robertson from syphilis, for instance).

Chronic meningeal inflammations are occasionally found in children, giving the signs of a posterior basilar meningitis. They are often accompanied by hydrocephalus. Opisthotonos is frequent. Many are due to syphilis, as the Wassermann tests show.

Hydrocephalus.—An accumulation of fluid within the cranial cavities takes place in a variety of affections. It is invariable in the acute meningitides, in greater or lesser degree; in tuberculous meningitis it is often extreme and comes on with great rapidity—acute hydrocephalus being practically synonymous with a tuberculous basilar meningitis.

External hydrocephalus is often synonymous with serous meningitis; as an entity it is infrequent.

Hydrocephalus is a very frequent complication in tumors of the brain, particularly in tumors of the third ventricle, the pineal, the corpora quadrigemina, and those causing pressure upon the aqueduct. These conditions are discussed under Brain Tumor. Ventricular hydrocephalus often results from such structural anomalies as cephalocele, and spina bifida.

In children one may find a primary chronic hydrocephalus of as yet unsettled pathology; but there is usually a chronic ependymitis present of possible hereditary luetic origin.

One to five pints, even quarts, of fluid may accumulate. The fluid is clear, slightly alkaline with sp. gr. 0.1005, containing the earthy

chlorides, albumin, phosphates, and occasionally a sugar-reducing substance. The presence of high percentages of albumin and many cellular elements indicates a more active inflammation. As a result of the pressure the ventricles widen, the septum lucidum is displaced, the cortex thins, the infundibulum dilates, the optic chiasm is pressed upon. Extreme distention may convert the pallium into an enormous cyst, with the smallest vestige of a cortex. The thalamus may be flattened, even the pons.

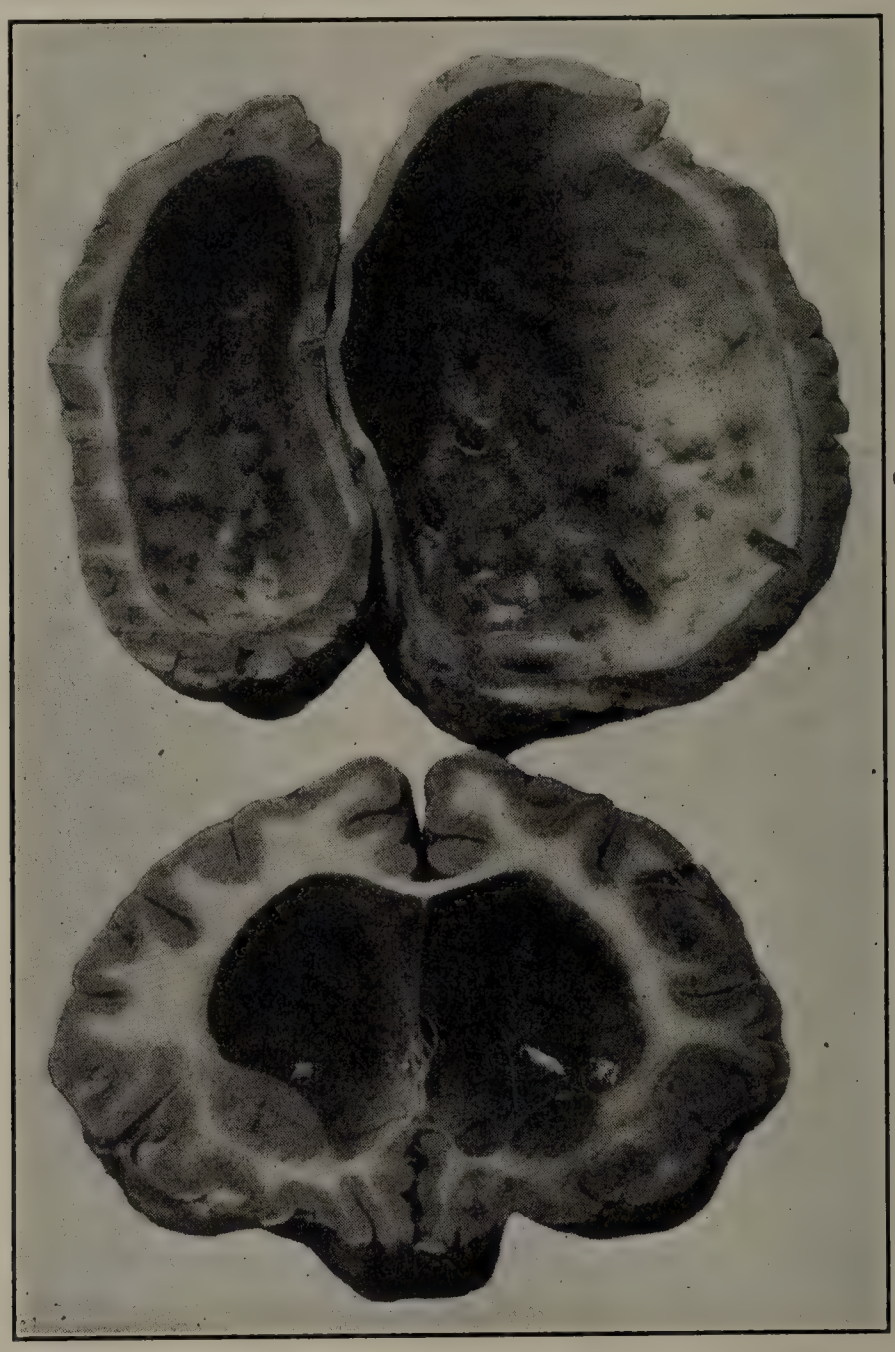


FIG. 357.—Showing marked hydrocephalus. Clinically the patient showed epileptic attacks and was markedly feeble-minded. (Munson.)

The cranial bones may become separated. The head enlarges, usually symmetrically, at the vault and at the base. In older children, however, hydrocephalus may exist without these changes in the position of the cranial bones taking place. The average cranial circumference at birth is fourteen inches; at one year eighteen to nineteen inches. In hydrocephalic heads this is increased. As a rule the congenital types show the largest heads. These patients are rarely born alive, or they live a short time only.

Symptoms.—Two or three or more months after birth it is noted that the child's head is increasing in size with more than the usual rapidity. Somnolence and lethargy are frequent; the child may not be able to hold the head up. A whiny, peevish, irritability, with frequent sharp cries is the rule. With fairly rapidly increasing internal pressure the coma is marked, showing great variability, however; vomiting is frequent, the hearing is affected, also the sight; choked disks, spasticities, with usually symmetrically increased reflexes. The pupils are usually contracted and sluggish to light early. With severe grades of pressure, there may be extreme dilatation. Convulsions appear and death results in from three to six months, with signs of emaciation.

In the cases of more gradual increase of intracranial pressure a marked degree of accommodation takes place. The symptoms are those of dulness or stupidity, the children are usually less bright, the choked disk may be very little marked, or may be excessive if the bones have not permitted distention, and may be followed by atrophies; but many cases recover with only a slight degree of mental reduction —weak-mindedness or only stupidity. There is a proverbial cheeriness in these patients. Some few make total recoveries, and may show brilliant mental capacities.

In the hydrocephalus of brain tumor in adults, with its up-and-down course, somnolence and headache, choked disk, dilated pupils, somewhat inactive to light, are characteristic. A cracked-pot percussion note is often very characteristic in the young and also in many adults.

Therapy.—Many cases are hopeless from the onset. A Wassermann test should always be made, both of the blood and cerebrospinal fluid, for the double purpose of determining syphilis or other inflammatory disorder. Many cases of ependymitis are syphilitic in origin. For these prompt mercurial treatment is indicated; salvarsan or enesol are useful. Inunctions are also valuable in the less rapidly advancing cases. Iodin therapy is advantageously combined.

Cerebral irritative phenomena need hot baths and bromides.

Tapping is frequently of service, but not always. Puncture of the corpus callosum may prove valuable. Lumbar puncture is not practicable to relieve pressure, as frequently the aqueduct of Sylvius is blocked; a dry lumbar tap is of diagnostic service.

SUNSTROKE.

This is a condition brought about by exposure to the direct rays of the sun or to great heat from any source.

The severe cases are marked by profound unconsciousness, very high temperature—108° to 110° F., perhaps by convulsions, delirium and death. The milder cases are so-called cases of heat exhaustion, with general prostration, little or no rise in temperature, and they recover spontaneously. These patients not infrequently show marked cardiac, renal, or other organic disease which undoubtedly conditioned the onset

of the symptoms by rendering the individual especially susceptible to this particular type of trauma. In the severe cases coma is profound, there may develop paralyses, hemiplegia, or other symptoms of focal brain injury—meningitis or encephalitis may follow, and if the patient lives, convulsive phenomena, definite psychoses, neurastheniform conditions, focal disturbances, ocular disturbances and deafness, and general conditions of weakness, may ensue.

Therapy.—The patients with hyperpyrexia should be placed immediately in cold water, the temperature of which is kept down by ice. The drop in the bodily temperature will determine the length of time of immersion. In the heat exhaustion cases a supportive line of treatment is indicated.

CHAPTER XI.

DISEASES OF THE BRAIN.

ENCEPHALITIS—ABSCESS OF THE BRAIN.

ENCEPHALITIS is an inflammation of the brain substance. There are a number of forms, but the term is here restricted to the more acute processes, due to bacterial or toxic action. Primary idiopathic encephalitis does not exist. General paresis is a diffuse encephalitis, with exudations and proliferation of new glia and bloodvessels; multiple sclerosis is a type of disseminated encephalitis, etc., but the discussion is here limited more particularly to acute exudative inflammations of the brain substance proper. Abscess is a frequent secondary result.

Historical.—The encephalitides were for years included with the meningitides, possibly under the term phrenitis, which to the ancients meant any excitement, accompanied by fever. In Hippocrates the typical description of phrenitis, however, is a typhoid delirium. Traumatic encephalitides were known to this author.

Just how long this conglomeration of cerebral affections remained an entity in nosology cannot be determined. Separation of types is going on at the present time, and it must be recognized at best that a heterogeneous group must be included for the present under the symbol encephalitis, a term used as early as 1554 A.D. by Actuarius.

Leaving aside the older works, the history of encephalitis practically begins with the works of Rostan (1820), Bouillard (1826), Lalleman (1830), and Fuchs (1835), in which processes of softening, of an inflammatory or non-inflammatory character were commencing to be recognized. Cruveilhier, in 1829, distinguished an apoplectic softening, suppurative softening, and softening with disorganization without pus or inflammation. Virchow's (1846) work on thrombosis and embolism threw much light on the subject, while the studies of Durand Fardel (1849), Haye (1868), and Huguenin (1876) commenced to give a modern touch. Then Wernicke (1881) described a toxic form, chiefly alcoholic, and Strümpell (1884) opened up the large study of the infective types. Finally, the modern study of epidemic poliomyelitis, by Medin and Wickmann, the recognition of syphilitic types by Freud and Plaut, the work of Councilmann and his students and the 1917–1920 epidemic of encephalitis (v. Economo et al.) have served to widen out the conception of the infectious types of Strümpell. Recent monographs of value are by Oppenheim and Cassirer (1907), Vogt, in Lewandowsky's *Handbuch der Neurologie* (1912), Hunt in Osler's *Modern Medicine* (1928) and McNalty's *Epidemic Diseases of Central Nervous System* (1927) and an enormous literature on epidemic encephalitis, q.v.

Etiology.—Infections and intoxications are many and various. Among the former are found the organisms of influenza, streptococcus,

typhoid, *Diplococcus meningitides*, pneumonia, scarlet fever, measles, tuberculosis, syphilis, trypanosomiasis, malaria, rabies, poliomyelitis, etc. The commonest acute forms are due to the influenza bacillus, pyogenic organisms (external and internal infections), including mastoid, etc., poliomyelitis; among the more common chronic types are tuberculosis and syphilis, which latter, as Plaut[1] has well shown, is responsible for many of the infantile cerebral palsies. An acute *epidemic encephalitis* of uncertain etiology, which under the title lethargic encephalitis, has given rise to at least 5000 larger and smaller studies from 1917–1928 as an epidemic extension throughout the entire world. This type has been known for centuries. The involvement of the interbrain, midbrain and basal ganglia give to this general form a special syndromy.[2] As for the intoxications, alcohol plays the chief role.

Acute Encephalitis.—Symptoms.—As so much depends upon the seat of the inflammatory process, it is natural that great variations in the clinical picture should be expected. The type of infecting agent also introduces a variant. The infecting agent of a polioencephalomyelitis is different from that of a syphilitic encephalitis. Although similar structures may be involved and in a manner very closely related pathologically, yet nevertheless the mode of development will be different. For this reason some of the more pronounced types will receive separate consideration. Any attempt to generalize on the whole group results in a descriptive monstrosity untrue to any clinical type.

1. *Influenza Encephalitis.*—Wickmann and others showed great care in attempting to separate this type from poliomyelitis. In the true influenzal type it was held that the disorder is apt to develop in young adults some days, or even weeks, after an acute influenza. Oppenheim includes other infectious types here. The "cold in the head" seems to be clearing up, when most intense headache—often in the occiput—nausea, vomiting, apathy, or drowsiness commences to indicate something more than the usual depression of an influenzal attack. Sometimes there is a rigor. Confusion and mild stupor are frequent, the patient being aroused with some difficulty after a few days. Symptoms of meningeal irritation are not prominent, and a clear cerebrospinal fluid will separate this condition from the closely related picture of cerebrospinal meningitis. There is usually much febrile irregularity. The pulse is very variable, being not infrequently slow.[3]

Focal symptoms develop irregularly. If the pyramidal region is involved there is premonitory weakness of the limbs, then paresis, then paralysis. Convulsive seizures may be present. The extent may be that of a monoplegia only, or a hemiplega, which may not show in the coma. Various aphasias may occur, pseudobulbar palsies, or

[1] Nervous and Mental Disease Monograph Series, 1911.

[2] See Strauss and Abrahamson: New York Med. Jour., 1919, vol. 1. Wilson, S. A. K.: Lancet, July 6, 1918, and literature cited under Epidemic Encephalitis.

[3] Jelliffe: Influenza and Encephalitis, New York Med. Jour., 1918. Crookshank: Influenza, London, 1922. Leichtenstein: Influenza, Nothagel's System, and many recent studies on the differentiation of influenzal from other types of encephalitis.

oculomotor palsies. Sensory anomalies are frequent, but are difficult to detect because of the mental state.

Many patients recover absolutely, others are left with slight motor defects, monoplegias, or severe hemiplegias. In a few abscess may develop. In others there is a complicating ependymitis with hydrocephalus, and frequently there are psychotic complications. Many cases are indistinguishable clinically from epidemic encephalitis.

2. *Polioencephalomyelitis* (Strümpell's acute encephalitis of children in part).—This has been discussed under the cerebral forms of polioencephalomyelitis (Heine-Medins' disease). This disorder shows the usual acute onset of this disease. The cerebrospinal fluid shows the characteristic bodies described by Hough and Lafora; the Wassermann reaction of the cerebrospinal fluid should be negative. The residuals here may be purely in the cerebrum, the midbrain, pons, medulla, and spinal cord escaping entirely. Various forms of infantile palsy (so-called Little's disease), idiocy, imbecility, monoplegia, etc., are to be encountered. Epileptiform convulsions are an infrequent residual of a motor zone focus. The prognosis is usually good. Many patients recover entirely, or with mild residuals. As a rule there are pontine, bulbar, or spinal complications.



FIG. 358.—Infantile cerebral palsy. (Frauenthal.)

3. *Polioencephalitis Hemorrhagica Superior*.—Wernicke called attention to a special form of diffuse encephalitis, with pronounced involvement of the midbrain (ophthalmoplegias). In one sense this is not a true inflammatory reaction. In the alcoholic and syphilitic patients the course of the affection resembles that of the Korsakoff syndrome, with pronounced eye palsies. There is usually an initial delirium, sometimes appearing later. Headache, nausea, and vomiting precede the development of an irregular type of ophthalmoplegia, apparently nuclear, possibly neuritic. Ptosis, nystagmus, irregular palsies, optic neuritis are frequent. The gait is cerebellar; the speech is slurring; the mental disorientation for time and place and confabulation are marked. Somnolence and loss of sensory functions are also frequent, and point to the involvement of the thalamus. There is no fever, no leukocytosis, and a doubtful cerebrospinal fluid. The pulse is usually rapid—80 to 120 often found. The nerve trunks and muscles are usually tender in the alcoholic types. Many of these patients clear up

to a marked degree, but there is usually a residual mental defect, showing in a lack of initiative and mild deteriorated states.

The lesions are those of a diffuse neuritis, with hemorrhagic foci.

A non-alcoholic variety has been described in which somnolence is more marked than delirium. There is ptosis, complete ophthalmoplegia, with vertigo and ataxia, disorientation and frequently confabulation.

Occasionally poisoning by mussels, by fish, bromides bacillus botulinus or from rabies shows similar clinical pictures.

4. *Pyogenic Types.*—Here a multiplicity of affections is to be reckoned with. Age is no bar. Traumatism bulks high in the percentage; also ear disease. The onset is usually acute, particularly in the younger patients. Trauma, or middle-ear, labyrinthine, sinus or other pyogenic infection is followed by malaise, headache, nausea, and vomiting with advancing stupor, or convulsions and increasing coma. Great restlessness, with increasing delirium, usually develops within from three days to two weeks. Leukocytosis is usually present. Lumbar puncture is usually negative, except when meningeal symptoms are also present.

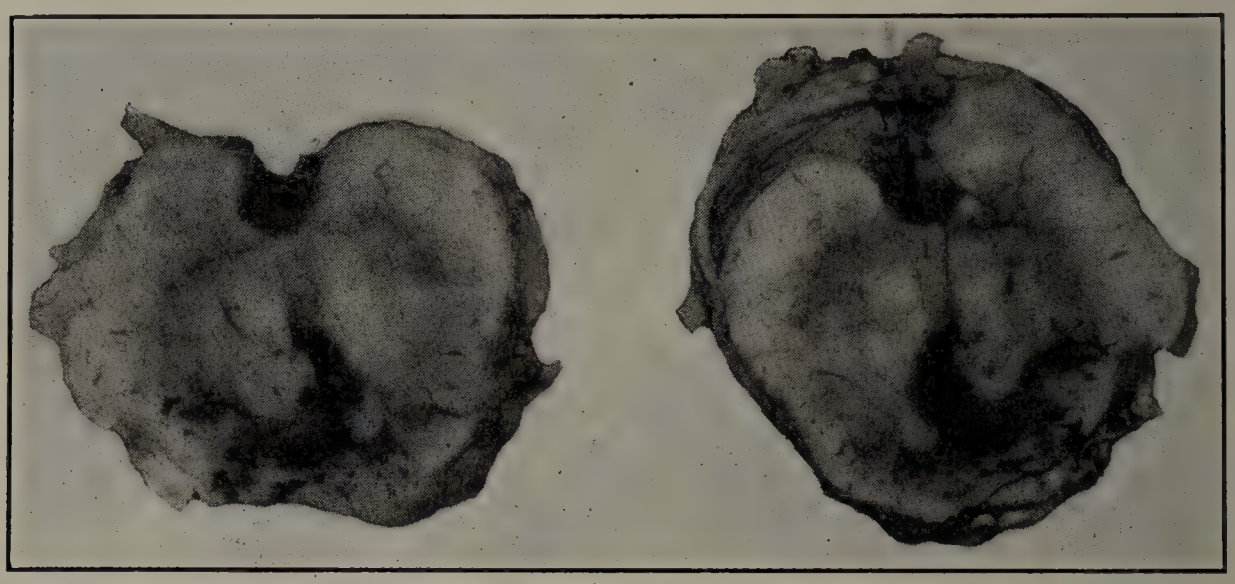


FIG. 359.—Midbrain lesions in paresis with Korsakov syndrome; Wernicke's polioencephalitis superior.

Topical signs are very frequent. These are monoplegia, hemiplegia, epileptiform convulsions, cranial nerve palsies. If the delirium clears up, one may find aphasias, pseudobulbar palsies, various midbrain syndromes, hemianopsias, and bulbar palsies.

Many patients die in coma. Others recover with marked mental defect (cerebral atrophies of childhood, idiocy, imbecility, debility). In others the general signs of brain abscess (*q. v.*) become apparent. Again others clear up with hemiplegias, monoplegia, diplegias (so called Little's disease). Finally, others recover entirely, or show small focal residual lesions.

Epidemic Encephalitis.—Under this title is included a host of cases, which have been observed throughout the entire world as an epidemic since Cruchet in 1916 first and v. Economo later (1917) more definitely described them. They are characterized by the acute involvement by

a general infectious process with characteristic localizations in the interbrain, midbrain and basal ganglia. Such epidemic types of encephalitis have been recorded for several centuries,[1] although attention to the striking features of the syndromy as now known is due to Cruchet and v. Economo.[2] In different epidemics different organ systems have been involved. Why this is so is an unsolved problem. The new features, picked out by observers as new, have been the involvements of structures whose functions medicine has just begun to understand, and hence notice. Thus in Leichtenstein's classic, *Influenza*, most of the present-known syndromes of epidemic encephalitis are recorded. The literature of epidemic encephalitis now composes at least 5000 titles (1928). Ocular syndromes were described in 1685.

Etiology.—In discussing poliomyelitis, attention has been called to the great difficulty in separating these encephalic syndromes. Those of influenza, poliomyelitis and epidemic encephalitis, one of us (Jelliffe) believes to be often *clinically* indistinguishable. The causative agent which *French* observers[3] believe they have isolated is an ultramicroscopic filtrable virus of variable virulence. It is claimed to be identical with herpes virus. Flexner has disproved this claim. Rosenow and Freeman claim to have found a bacterium. Jahnel, who has analyzed very minutely all of the claims, comes to a negative standpoint. No generally accepted specific agent has been isolated up to 1928.

Clinical Pictures.—The student might refer to the chapter on Poliomyelitis to see all of the possibilities of epidemic encephalitis, but as in this epidemic the bulbar, pontine, interbrain, midbrain, basal ganglia and cortex were more affected than in the poliomyelitis epidemic—which picked out the spinal cord more flagrantly, special attention should be devoted to these forms. Particularly as many important problems of neuropsychiatry have been entirely revolutionized by the study of epidemic encephalitis.

The clinical picture is that of an acute or subacute infection. Anorexia, nausea, headache, vomiting, subfebrile course with slight rapidity of the pulse. Then one of two main types may develop. (1) The lethargic type. This is the one first picked out by v. Economo. The patients fall asleep at times with transient states of confusion. This form which is often fatal is usually accompanied by diplopia, athetotic signs and rigidity. Lethargy and akinesis remain the leading symptoms. (2) The second type is the hyperkinetic one with sleeplessness. Mild or severe deliria are frequent. They often resemble severe acute chorea-minor. Hyperkinetic movements with twitch-

[1] Crookshank, F. G.: Influenza, London, 1922. Luger: Grippe and Encephalitis, Wien, klin. Med., vol. **39**. Sauvages: Albrecht v. Hildesheim (1685).

[2] Die Encephalitis Lethargica, Deuticke, 1918; translated into Italian, Il Policlinico sez. med., 1920, **27**, 93. Also see monographs of Tilney and Howe, Achard, Reys, Stern, Ricaldoni and numberless others. For chronic types see Wimmer.

[3] Levaditi, Harvier and Nicolau: Ann. de l'Inst. Pasteur, February, 1922. Also Perdreau: British Jour. Exp. Path., 1925, **6**, 123. Rosenow: Jour. Am. Med. Assn., 1922, **79**, 443. Jahnel: Ztschr. f. d. ges. Neurol. and Psychiat., 1925, **99**, 253; for literature and complete discussion of etiology. Kling: Wien. Arch. f. inn. Med., 1923, **6**, 101.

ings, myoclonias, especially of the diaphragm and abdominal muscles (hiccough types) are usual. These cases also frequently die.

In rarer instances acute Parkinsonism may be present from the beginning. In children acute excitements and pronounced behavior disorders may inaugurate the picture.

Other detailed symptoms may now be considered. These vary from patient to patient.

Ocular palsies then set in. They are inconceivably numerous (Cords)[1]. Diplopia is the chief one. Any and every variety have been described. Ptosis, unilateral or bilateral is frequent. These ocular palsies may be quite intermittent. Hypotonia of the muscles is characteristic. Complete internal and external ophthalmoplegia is rare, but has been observed. Ciliary muscle paresis and iris paresis are both present. Argyll-Robertson pupil has been seen (Jelliffe, Economo, Guillain, Lortat-Jacob). Loss of convergence is the commonest residual.

Sleep comes on at about the same time, or it may precede the ocular palsies. It is a peculiar type of lethargy varying from simple sleepiness to a suppressed busy delirium with coma. Patients walk around with it. They may lean against a wall and fall asleep, are prodded, go on, sit down and fall asleep. The variations defy description within the limits of a text-book. Sometimes there is an agitated delirium instead of a lethargy (maniacal cases).[2]

Reflexes are very variable. Usually average, not infrequently exaggerated, rarely diminished. Clonus and Babinski are not rare—especially the latter. Abdominal and cremasteric reflexes are usually unchanged, but may be.

Sensation to gross tests is unchanged. Anesthesiæ have been recorded. Visceral esthesiæ are markedly diminished. All incoming stimuli seem to be interfered with somewhat (thalamus).

Paralyses are very variable. Hemiplegia, diplegia, monoplegias, etc., are not infrequent. Neuritic cases are known (Kennedy) and cases resembling brain tumor (Kennedy, Jelliffe). They cannot be detailed here.

Eye grounds, these are usually average, but choked disc and hemorrhages are recorded. (Kennedy, Jelliffe, Cords.)

Motility Changes.—These have varied enormously. Hiccough has been an interesting occurrence. Myoclonias have been frequent. Tics, Parkinsonian syndromes, choreic,[1] choreo-athetoid, myoclonic tremors, intention tremors and multiple sclerosis syndromes, spasmodic laughing, crying, simple hypertonus of the muscle system with electrical hyperexcitability and increased chronaxia (Bourguignon). This muscular hypertonus may be general or localized to limited segments. Speech variations are frequent, pseudobulbar, Parkinsonian, explosive, trembling, dysarthyric, monotonous, etc. Epileptiform convulsions have been observed. Respiratory phenomena, tachypnea, etc., are frequent. (Lévy, Jelliffe, Turner and Critchley.)

Mental changes are too numerous to catalogue. These may be distinguished as: (*a*) Early changes; (*b*) during the illness, (*c*) after

[1] Cords: Zentralb. f. d. ges. Ophthalmopl., 1922, **5**, 225.

[2] Babonneix: Chorea and Encephalitis, Paris méd., October 6, 1923. Salmon: Paradoxical Kinesia, Riforma méd., 1923, No. 6.

the illness and (*d*) residuals. The early stages resemble those of a toxic infectious delirium with confusion and disorientation. Later the picture may continue and show atypical catatonic, hebephrenic,[1] or paranoid-like syndromes. Acute maniacial states are met with. Defect states develop, or prolonged anxiety syndromes, with the myotonic features in the muscles, remain for long periods. A vast variety of behavior disorders occur in children in the chronic stages. Criminal acts may be performed which are of considerable medico-legal significance.

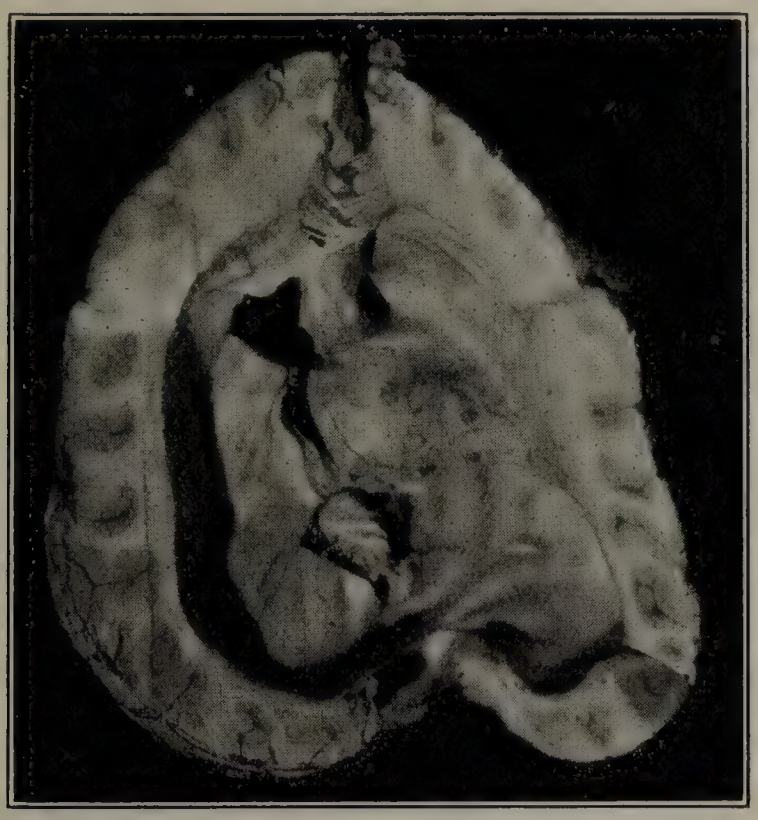


FIG. 360.—Internal hydrocephalus secondary to epidemic encephalitis—meningoencephalitis. (Courtesy of S. C. C. Turner, Memphis, Tenn.)

Reviewed briefly, the following trends or types are more or less frequent: (1) Somnolent ophthalmoplegic types; (2) paralytic, akinetic or hypokinetic types; (3) amyostatic syndromes (Strümpell, Boerstrom); (4) hyperkinetic types, myoclonias, tics, choreatic, multiple sclerotic, athetotic, dystonic, epileptiform, narcoleptiform; (5) bradykinetic types (Cruchet, Verger, Hesnard,[2] Steck); (6) hyperalgic neuritic, radiculitis types (Kennedy); (7) tabetic types, with or without ataxia; (8) cerebellar types; (9) anomalies of conduct, psychoses, resembling schizophrenia—manias; (10) various vegetative disorder types; (11) respiratory types.

This is but a crude summary of the almost limitless situations that have been described from cases all over the world. Thus soldiers brought the disease to India and it spread to China and Japan, and wherever found similar very diverse syndromes were observed.

Cerebrospinal Fluid.—The changes here vary considerably. At times there are no new findings. Lymphocytosis is not infrequent.

[1] Jelliffe: Encephalitis and Schizophrenia, Am. Jour. Psychiatry, 1927, **6**, 413. Bibliography.

[2] Verger and Hesnard: d'Etal mental des bradykinetiques encephalitiques, Jour. d. méd. de Bordeaux, September 25, 1922.

Albuminosis is rarely met with. Actual blood may be infrequently found from meningeal hemorrhage (Jelliffe, Achard). Hyperglycorachia is recorded.

Chronic Phases.—Wimmer[1] and Lévy have given well-documented monographs that no student of these syndromes can neglect. The mortality in the acute cases has varied in different epidemics (10 to 50 per cent), usually from respiratory failure (Flatau), but what is more important and serious is that the vast majority of those who do not die develop in from a few weeks to four to six years, either with apparently free intervals, or as following an infection (often influenza-like), chronic phases of the disease. In from 50 to 80 per cent. of these a Parkinsonian picture develops or one of the numerous dyskinesias already spoken of under the section of Midbrain, Interbrain or Striatal Diseases. Others have developed endocrine defects, others disease of the viscera.

Glandular changes are numerous and vagotonic and sympathicotonic reactions result from implication of the adrenals, thyroids and pituitary.

Metabolic disturbances from central involvements are very frequent. Hyperglycemia, azoturia, modifications of water excretion, salt loss, obesity, Fröhlich syndrome, loss of flesh, hypothermia, hyperthermia, blood changes, etc. Herpes is frequent; also seborrhea in the Parkinsonian cases.[2] Sialorrhea is frequent, either central or from local parotitis (Levaditi).

In children anomalies of conduct have been extremely frequent as sequels or as evidences of a chronic process. These are extremely diverse and vary from what moronic observers deem naughtiness, bad temper or hysteria, to definitely indefinite psychotic pictures. (See Kraepelin, Kirby, Hohman and numerous others.) Some of these are schizophrenic-like. (See Hauptmann, Schilder, Meyer-Gross, Jelliffe[3] and others.) Chronic respiratory types are striking but infrequent.[4]

Differential Diagnosis.—Often the delimitation is quite simple, again it may be impossible. Syphilitic encephalitis and meningitis should first be ruled out. The Wernicke type of alcoholic encephalitis also. Cerebral concussion at times may be confusing. Two patients personally seen had fallen, had blood in the C. S. F., optic nerve compression signs, and resembled fracture of base cases, but were encephalitis cases. A group of brain tumor-like cases has been recorded.[5]

Poliomyelitis shows in its bulbar, mesencephalic and cortical forms precisely similar features. Whether the neutralization reaction of Netter or the immunity reaction of Amoss will permit a separation is still under discussion. Botulism is to be separated. The early cases in England were diagnosed as botulism. Other types of myatonia, myoclonus, paralysis agitans, electric chorea, tic, etc., need a close

[1] Wimmer, A.: Chronic Epidemic Encephalitis, London, Heinemann, 1924. G, Lévy, Paris Thesis, 1922.

[2] Steifler: Wein klin. Wchnschr., 1924, **37**, 334; Krestin: Quart. Jour. Med., 1927, **21**, 177.

[3] See Jelliffe (Am. Jour. Psychiat., January, 1927) for discussion of literature of these types.

[4] Lévy, G., Turner, Critchley, Jelliffe: Postencephalitic Respiratory Disorders, 1927.

[5] Heinrich: Arch. f. Psych., 1926, **78**, 643.

scrutiny as to etiological factors. As has been emphasized frequently in this book all these titles are but syndromes due to different agents. They are not things in themselves. All levels must be studied.

Pathology.—Macroscopically there is meningeal congestion, sometimes hemorrhage. Exudative inflammatory foci, large or small, may be isolated or widely disseminated through the cerebrospinal axis. In the so-called classic cases the striatum, locus niger, midbrain and hypothalamic regions are the seat of the diseased foci. The foci show the vascular dilatation, diapedesis, exudative, proliferative changes similar to the acute infectious encephalitides—as in influenza, poliomyelitis, rabies, etc.

Observers are divided as to the specificity of the disease.

Treatment.—This is still largely symptomatic. Urotropine given intravenously has helped some—not others. Lumbar puncture repeated has seemed to aid a number of patients. Arsenic has been of some service for the motility disturbances.[1]

Certain principles stand out.[2] The disease is infectious, though but slightly contagious, and is probably caused by a pleomorphic organism that gains entrance to the nervous system by the blood stream from foci of infection about the nose and mouth. It is particularly liable to develop in individuals who have been under prolonged strain with insufficient periods of rest. Its prophylaxis, therefore, includes the maintenance of optimal health, physical and mental. During the period of acute manifestations salicylates are the medicinal preparations of choice for their direct action on the supposed cause, and colloidal materials for their non-specific effect on the immune bodies. Attempts at serotherapy are promising, but the question of specificity is not yet solved. Blood transfusion may be a life-saving measure. With the inception of convalescence, foci of infection are to be eradicated, and it is possible that active immunization with killed streptococci will forestall the late recurrences. Up to the present time the indications have been favorable. With the development of the chronic progressive manifestations, similar therapeutic aids are invoked in arresting the course of the disease, and protein shock treatment, intravenous use of sodium iodide (Farnell) are to be considered.

For the chronic stages every conceivable type of therapy has been devised to little purpose. Some recover spontaneously, but a large number go into chronic invalidism in spite of every form of therapy; thousands are hospitalized at the present time. Various drugs of the belladonna type are of doubtful value. They only temporarily hold up some of the motor symptoms and create an illusion of doing something.[3] They are used by the business man chiefly.

[1] The literature on epidemic encephalitis comprises several thousand titles. The interested student is referred to v. Ecnonomo's initial contribution, Vienna, 1917. Howe and Tilney's small monograph, 1920. Achard's, 1922. Reys, 1922. Stern, 1922, monographs. Monograph of Research Association, 1921. See chapter in Sachs and Hausman: Nervous and Mental Disorders, Hoeber, 1926. Bibliography, Jour. Nerv. and Ment. Dis.; Rev. neurol.; Ztschr. f. d. ges. Neurol. u. Psychiat; Index Medicus.

[2] Freeman, W.: Am. Med. Assn., October 15, 1927.

[3] See discussion of psychotherapy for some of the chronic cases in Jelliffe's monograph, Postencephalitic Respiratory Disorders, 1927.

DIAGNOSIS OF THE ENCEPHALITIDES.

It is impossible with present methods to clearly distinguish all of the various encephalitides. Many are not diagnosed during life, the course being so rapid, and facilities for laboratory research limited. At the present time (1928) any attempt at presenting the problem is only a patchwork.

Wernicke's type is characteristic. It is, however, frequently reserved for the psychiatrist to make the diagnosis, as the mental symptoms so mask the physical signs. Ophthalmoplegia, neuritis, disorientation for time and place, with confabulation, progressing to delirium or coma, the almost typical facies, closely resembling the Korsakoff facies, tachycardia, and no hyperthermia are the leading features. Here the entire series of the alcoholic syndromes must be reviewed. (See Alcoholism.)

Polioencephalomyelitis is to be suspected during an epidemic. The acute onset, frequently with gastro-intestinal or nasal respiratory predecessors; the frequency of spinal and bulbar involvements; usual absence of signs of neuritis; lost knee-jerks, spinal fluid findings are suggestive.

Mesencephalic syndromes in influenza are infrequent but they do occur.[1] There are many etiological causes for lethargic encephalitis. Measles, mumps, syphilis and others are among them. The symptoms are solely the result of the location of the process.

Meningitis often calls for diagnosis. Here lumbar puncture is of great service, since many of the initial clinical signs are identical. Increased pressure, presence of cellular elements and pus are present in purulent meningitis. In epidemic cerebrospinal meningitis there is the characteristic organism.

Herpes is quite frequent in encephalitis; the neck and muscle hyperesthesia usually less. Eye-muscle palsies are often absent in the pyogenic types of encephalitis; they are not infrequent in meningitis. In syphilitic types the Wassermann test is of great service.

Therapy.—Practically only in the malarial and syphilitic encephalitides is there any specific therapy. In the pyogenic forms, with abscess, surgery is demanded. The poisonings require withdrawal. Bromide poisoning is not to be overlooked.

All these patients require bed treatment in a darkened room. The toxic cases should have hot sheets and elimination encouraged. Ice-bags to the head give comfort and counter-irritation; they do not affect internal temperatures. Active catharsis is desirable, calomel and salines being advisable.

In the influenzal types, salicylates are indicated. Otherwise the treatment is largely symptomatic. The residual symptoms call for their special therapy.

[1] Jelliffe: Influenza and the Nervous System, loc. cit.

ABSCESS OF THE BRAIN.

History.—Brain abscesses have been recorded for many years: Cruveilhier's and Carswell's illustrations are classic. The steps that led up to the present-day conception of brain abscess are partly outlined in the section on Encephalitis. The history of these two groups is almost identical up to the appearance of Wernicke's *Lehrbuch d. Gehirnkrankheiten,* 1883, when a division into infectious and non-infectious abscesses was foreshadowed. Macewen's (1893) studies gave a great impetus to the understanding of the ports of entrance, while Korner's (1902–1908) studies have set in clear relief the importance of aural disease. Detailed monographic or critical studies are those of Oppenheim and Cassirer (1909), Brissaud and Soques, in Bouchard and Brissaud's *Traité de Medicine* (1904), IX. Lewandowsky, in Lewandowsky's *Handbuch der Neurolgie* (1912). Southard, in Osler's *Modern Medicine* (1915), and Warrington: *Quarterly Journal of Medicine,* January, 1918. Schlesinger: *Kraus. Brugsch.,* vol. 10, p. 2. Eagleton: *Brain Abscess,* 1922.



FIG. 361. — Infantile cerebral palsy. (Frauenthal.)

Etiology.—Cerebral abscess is almost exclusively a secondary phenomenon due to infection. Many organisms have been found in cerebral abscess. They are derived from many sources; external trauma with or without fracture, the venous sinuses, and otitis media being among the commonest. Otitic infection supplies about a third, the usual communication being through the tegmen of the antrum. Nose infections and pyogenic processes in the accessory sinuses play important roles. Metastatic abscesses, from pyemia, tuberculosis, osteomyelitis, abscess of lung, liver, female pelvic disease, etc., are among the curiosities. Streptococcus pyogenes is the most frequently found bacterium. S. aureus citreus, B. pyocyaneus and pyogenes fetidus are frequently found. Multiple infection is the rule and a great many different organisms have been isolated.

Symptoms.—There is always some antecedent disorder, although such may be overlooked or be silent, the acute miliary tuberculosis of cardiac disease being an example. The symptoms will vary greatly with the exciting cause, the site, and the size of the abscess or abscesses. Certain traumatic abscesses run their fatal course in three to five days, whereas some recorded cases have persisted for years. Intermediary types of every variety are the rule.

There are enough cases described to show that, speaking in general

terms, one may recognize (1) a stage of irritation, (2) a stage of remission or latency, and (3) a stage of paralysis or of acute progression.

1. *Primary Stage.*—In the initial stages, general rather than localizing signs predominate. The patient usually has an acute rise in *temperature;* it is frequently slight, insidious, and variable, and often absent after a few days. The *pulse* is often slow. *Headache* is rarely absent but is very variable in intensity. It is most frequently dull, but gives a sense of tension in the head. There may be some vomiting.

Such a tense headache, with rise in temperature and slow pulse following an otitis, after the discharge may have ceased, is of grave import. The pain is situated usually over the side affected; but is not infrequently slight or absent in cerebellar cases (*q. v.*) or here may be frontal in character. Movements are apt to increase the headache. Anxiety, insomnia, restlessness are the rule, and in younger patients the sleep is often broken by loud cries. There may be some delirium or stupor in these initial stages, which occupy the first three or four days, occasionally a week. There is frequently some leukocytosis in this stage.

2. *Stage of Remission or Latency.*—A period of depression often sets in about this time. The headache diminishes, the fever diminishes the patients are fatigued, somnolent, indifferent. The pulse is apt to remain slow. Percussion at this time may reveal tenderness. The blood count is variable, with a tendency to polynuclear increase. This stage shows extraordinary variation in different cases, sometimes persisting weeks or months.

In this stage beginning choked disk is not infrequent, especially if the disorder has lasted over a week or ten days. It is apt to first appear homolateral with the abscess. There are indefinite signs of a cerebral affection; variable headache, occasional nausea, vomiting, fainting attacks, or convulsive seizures.

The pupils are frequently unequal, if there is pus, and the more dilated pupil is apt to be on the side of the abscess. With maximum equal dilatation, the localization is difficult from the pupils alone, and such dilatation argues for a large abscess. With increasing size, immobility appears. Contracted pupils are found, in a few instances, toward the end in severe cases.

3. *Stage of Advance.*—After a period averaging from one to three months the symptoms of an advancing cerebral pressure manifest themselves. There are delirium, often epileptiform convulsions, all of the symptoms of a generalized meningitis, with death.

Occasionally encapsulation occurs with no symptoms for from ten to twenty-eight years[1] (Nauwerck).

Course.—The course just described is, statistically speaking, the most frequent, but it is by no means universal. In many cases there is an acute progressive course, without any intermission or remission. This may also follow a latent period, or may develop acutely after a

[1] See Oppenheim, loc. cit., for literature of these rare cases.

trauma, or following a suppurative process in the ear or nose. In others there occurs an acute progressive course, with a remission which is incomplete. The chronic course is as described.

Forms.—The most marked are the traumatic, the otitic, the rhinogenic, and metastatic. In the *traumatic* cases the abscess is usually near the site of the trauma, and the symptoms of the abscess formation are apt to be complicated by the traumatic incidents. The symptoms of a leptomeningitis develop early, whereas those of a purulent destructive nature usually require from eight to fourteen days (Bergmann). The whole development is usually gradual, and the stages far from being sharply set off one from another. Headache, fever, vomiting, vertigo, confused delirium, these are the prodromal signs. The focal lesions occur several days later, either as an epilepsy, a cortical monoplegia, a speech or other motor or sensory defect.

Traumatic *late* abscesses are also known, weeks, months, even years go by, before complete evolution.

Otitic brain abscesses are frequent, yet in proportion to otitis are rare (Jansen, 7 in 5000 cases). Here the original disorder, the localization, and the complications are of moment. The development has already been sketched. It is very insidious in many cases. The localization is usually either temporo-sphenoidal or cerebellar. Heinman's[1] study of 818 cases showed that there were 539 cerebral and 279 cerebellar involvements. Of 645 carefully studied cases the localizations were as follows: Temporal 395, temporal and occipital 19, temporal and frontal 4, temporal and cerebellar 10, occipital 9, frontal 3, cerebellar 186, others scattering. The symptomatology of right- and left-sided temporo-sphenoidal abscesses or tumors offer considerable variations. A right-sided abscess may pursue its course with little or no localizing signs. Left-sided ones often give rise to aphasic complexes. Pressure upon the pyramidal tracts will give characteristic hemiplegic phenomena; there are apt to be disturbances of smell, direct or subjective, olfactory, or uncinate fits (Knapp, *loc. cit.*).

Rhinogenic abscesses are usually located frontally. In addition to the headache, there is frequently a certain degree of torpor, often associated with a tendency to joke (Witzelsucht), isolated choked disk, a cerebellar type of ataxia, when the cortico-rubro-cerebellar pathways are implicated, isolated tremor of the hand, and olfactory disturbances. When the abscess reaches farther back, motor symptoms occur.

Diagnosis.—Many differential points arise. The etiology is the most important factor to bear in mind. Traumatic purulent meningitis, apoplexy, late apoplexy (Bollinger), pachymeningitis, brain tumor, tuberculous meningitis, syphilitic meningitis, hemorrhagic encephalitis, meningitis serosa, psychogenic headache, sinusitis and sinus thrombosis are the chief disorders to be reviewed. The last often runs a very similar course, and is often associated with a purulent otitis, as a fundamental disorder. Here the fever is apt to be high and remittent, even

[1] Arch. f. Ohrenhk., lxxiii, 258.

on the same day, rigors, chills, and profuse perspiration, pulse usually rapid and irregular, convulsions rare, save perhaps in children, headache constant, eye-grounds, as in abscess, usually bilateral neuritis, more frequent than choked disk. Focal symptoms rare. Physical signs of swollen spleen, and metastatic invasions confirm the diagnosis of thrombosis.

A purulent meningitis may be more difficult to differentiate. It may be caused by similar factors; it is usually more acute and stormy in onset; it usually runs a shorter course, shows higher and more continuous fever, herpes, the pulse usually rapid, irregular, often slow in beginning as in abscess, initial unrest, irritability, confusion, delirium in contrast to a heaviness or coma in abscess, headache constant, vomiting frequent, convulsions common, general clonic and often muscular twitches, the eye-grounds are less often positive, meningeal irritation phenomena (Kernig, spasticity, clonus, etc.), common, local symptoms more confined to cranial nerves and basal signs rather than intracerebral focal signs, spinal symptoms frequent, whereas in abscess they are rare. Furthermore, the cerebrospinal fluid is more apt to contain globulin and show a lymphocytosis or even pus.

Prognosis.—When left alone, cerebral abscess almost invariably causes death. Calcification or external discharge occasionally occurs.

Treatment.—This is exclusively prophylactic in nose, throat, and otitic work, and surgical after the development of the abscess. The results of operative interference are highly satisfactory. The statistics vary with each new series of cases. In the hands of competent surgeons the risk from operation is *nil*. The temporosphenoidal and frontal abscesses can be readily reached and drained.[1]

[1] Starr: Med. Record, 1906. Halsted, in White and Jelliffe: Modern Treatment of Nervous and Mental Diseases, 1915.

CHAPTER XII.

DISEASES OF THE BRAIN (CONTINUED).

VASCULAR DISTURBANCES—CEREBRAL APOPLEXIES.

THE quantity and quality (viscosity) of the blood, the heart action, the vascular tension and blood-pressure are the chief factors which govern the general as well as the cerebral blood flow. Of all the bodily structures, the brain is probably the most highly vascularized, arguing for its great functional activity. Furthermore, there is marked plasticity evident in its compensatory regulations. Not only is there a continuous balance maintained between different vascular systems, especially between those larger groups, such as the portal system, the vessels of the skin, the muscles, and the brain, but within the brain itself different balancing reactions are constantly going on between speech areas, visual areas, auditory areas, motor systems, visceral areas, etc., which are thrown into use at different times in specialized occupations or activities, or in response to emotional stress.[1]

The anatomical structures used in regulating these compensating mechanisms are partly localized in the medulla and cord, as discussed in the chapters on the Vegetative Neurology of the Vascular Apparatus, but parts are within the vascular apparatus itself; at times within the walls, again within the circulating blood (viscosity disturbance).[2]

The smaller cerebral vessels, in conformity with the softness and plasticity of a developing organ, have less rigid walls, hence more easily overfill (congestion) and contract (fainting), are more liable to spontaneous rupture or thrombotic or embolic occlusion, and, furthermore, are peculiar in that they have few or no anastomosing branches. Thus special problems are connected with the cerebral circulation.

The avenues of blood intake are through the two internal carotids and and the two vertebrals. The course of the left internal carotid is more direct, and it has been taught that thrombi are therefore more common on the left side. There are no decisive facts to bear out this *a priori* conjecture.

The vertebrals unite to form the basilar, which bifurcates into the two large posterior cerebral vessels which supply the temporo-occipital lobes, the corpora quadrigemina, crura, and parts of the optic thalami. The posterior communicating branches, usually small and symmetrical and subject to great variation, pass forward to join the internal carotids, and are given off to the base of the brain. The internal carotids form the middle cerebral and the anterior cerebral arteries, an anterior communicating branch completing the circle of Willis.

[1] Cannon: Fear, Hate, Rage and Anger, p. 183. Sepp: Die Dynamik der Blut circulation in Gehirn, Springer, 1928.

[2] Hirschfeld: Ztschr. f. Neur. u. Psych. Ref., vol. **4**, 193.

The middle cerebral arteries lie in the Sylvian fissure, giving off many branches to the lenticular nucleus, the internal capsule, portions of the thalamus, and superficially irrigate the Island of Reil, Heschl's convolution, and much of the lateral aspect of the brain.

The anterior cerebral arteries supply the frontal areas, the olfactory apparatus, the upper margin of the frontal, parietal and occipital lobes on their mesial aspects, and much of the corpus callosum.

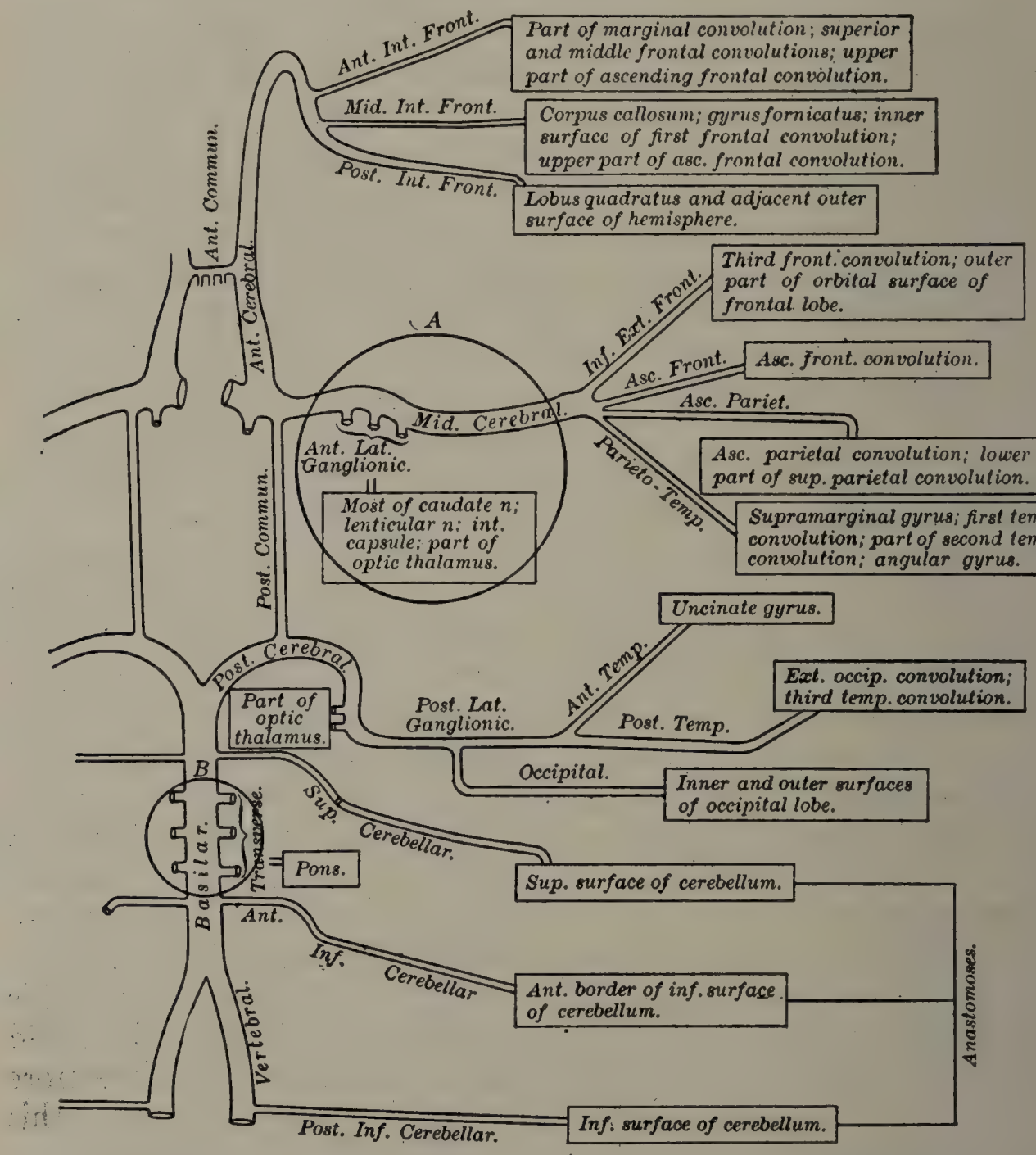


FIG. 362.—Circle of Willis and branches, with indication of distributions. Region *A*, *B* most frequent sites for hemorrhages. (Greenacre, from de Fursac.)

The cerebellum derives its supply from the vertebral and basilar arteries.

Partial compensatory balance of the circulation is brough about chiefly through the circle of Willis. This circle, as well as the branches from it, shows a vast number of anomalies, and these in part determine many anomalous cerebral disturbances, possibly related, as Blackburn, Windle, Bullen and others have shown, to faulty cerebral development, thereby laying the anatomical foundation for a neurosis, a psychosis

or some aberrant vascular disease (softening, deterioration, vicarious menstruation, etc.). Furthermore, such anomalies are of great surgical importance.[1]

The chief cerebral arterial disorders arise from (*a*) temporary vascular instability (shock reactions, cardiac irregularities, internal secretory disturbances); (*b*) protracted regressive changes (arteriosclerosis, with or without miliary aneurisms); (*c*) hemorrhage; (*d*) thrombosis; (*e*) embolism.

Venous changes are less frequent than arterial ones. Sinus thromboses are the most important.

(*a*) **Vascular Instability.**—This is a very variable factor and has been discussed under the sections on Vegetative Neurology. Vagotonia and vagotropia occur in the cerebral vessels as well as in the somatic vessels. The results were previously grouped under the concepts cerebral anemia and cerebral hyperemia. Chronic vagotonic conditions are known. They should not be called cerebral congestions.

Anemia (Shock; noci-association—Crile) results from loss of blood, paracentesis, surgical handling of intestines, cardiac weakness, or from marked vascular instability, often of psychogenic origin. The symptoms are faintness, dizziness, black spots before the eyes, buzzing in the ears, and it may be loss of consciousness with or without nausea and vomiting.

There may be a partial consciousness, apathy or semicoma, with contracted pupils and cold clammy skin, occasionally loss of bladder or bowel function.

The therapy is heat and bandaging of the extremities, the horizontal position, camphor, caffein and cardiac stimulants, alcohol and ether. The many problems of surgical shock must be sought for in surgical literature.

Hyperemia.—Hyperemia may be active or passive. The former may result from vascular instability, or is not infrequent after excessive eating or drinking. Sudden emptying of the peripheral and somatic vessels may occasion it. It is frequent in certain thyroidisms, notably in exophthalmic goiter and in natural and artificial menopause states, which latter often are accompanied by changes in the organs of internal secretion. (See Vegetative Neurology, Endocrinopathies.)

The symptoms of hyperemia are redness and congestion of the face and eyelids, pounding in the ears or neck or head, headache, confusion and usually contracted pupils. More severe attacks may lead to acute confusion, rise in temperature, and the general picture of an acute meningitis.

Signs of congestion of the face—flushing, a sense of fulness, etc., must not be taken to necessarily mean cerebral congestion. It should be borne in mind that the vessels of the face and those of the brain are controlled by separate mechanisms.

[1] Blackburn, I. W.: Jour. Comp. Neur., 1907, vol. **17**, 93; 1910, vol. **20**, 185.

Passive hyperemias result from mechanical obstruction to the jugulars, chiefly from new growths, emphysema and loss of compensation of heart disorders. Pressure of the head, abnormal wakefulness or sleepiness, dizziness, apathy or mild confusion, with anxiety, are the chief signs, combined with definite signs of passive congestion of the skin, face and mucous membranes.

Efficient therapy consists in ameliorating the causative factors whether active or passive. The hyperemias are purely secondary. Acute states may call for bleeding, mustard foot-baths, active catharsis.

(*b*) **Cerebral Arteriosclerosis.**—This is a widespread disorder occurring chiefly after forty years of age when with increasing years it tends to become more and more prominent. The arteriosclerotic process differs little in cerebral vessels from those located in other organs of the body, save in a tendency for a marked production of miliary aneurisms. These are largely conservative formations and are not to be considered as productive of hemorrhage, as taught by Charcot and Bouchard.[1] Cerebral arteriosclerosis is due to the same causes that produce arteriosclerosis elsewhere (heredity, syphilis, tobacco, chronic hyperadrenalemia from psychogenic, conscious and unconscious, or other factors, etc.), and may be the expression of a general disease, or may be sharply delimited to the cerebral vessels.

Symptoms.—The symptoms of early arteriosclerosis may be general or localized. The chiefly early symptoms are sleeplessness, restlessness, headaches, especially if there are dizzy attacks, and renal changes, neural fatigue, increased emotionalism and irritability. To this, at times is added hebetude, ready forgetting, especially of new impressions, increased blood-pressure. These signs are often loosely and improperly spoken of as neurasthenic. Local signs may be added, such as temporary lapses, marked somnolence, tingling or numbness or other signs of focal disease.

The chief neurological interests are focussed upon the distribution of the focal lesions and are here discussed according to the syndromes presented. The psychiatric features are only mentioned here, as they are considered in the special groupings under the senile, presenile and arteriosclerotic psychoses. (See Section III.) Thus only the more distinctly neurological features are accented in this place. (See tables in chapter on Midbrain.)

These chief syndromes are:[2]

I. Disorders due to disease of the twigs and terminal branches.
 - (*a*) Cortical branches; irritative complexes, Alzheimer's disease.
 - (*b*) Medullary branches; lacunar syndromes.
 - (*c*) Diffuse types (arteriosclerotic dementias).

[1] See Pick (Berl. klin. Wchnschr., February 21, 1910, p. 325) for bibliography.

[2] Lambert: States Hospital Bull., 1908, **1**, 475. Also see Kraepelin's Lehrbuch for complete discussion of arteriosclerotic disturbances.

II. Disorders of chief branches.
 (*a*) Anterior cerebral syndromes, crural palsies.
 (*b*) Middle cerebral syndromes.
 1. Palsies: hemorrhage, embolism, thrombosis, palsies, apoplexies.
 (1) Monoplegias: facial, brachial.
 (2) Combined palsies. Apoplexies in general.
 (3) Complete palsies: capsular and supracapsular.
 (4) Thalamic syndromes.
 2. Aphasias.
 3. Hemianopsias.
 (*c*) Posterior cerebral syndromes.
 (*d*) Inferior, superior, posterior, cerebellar syndromes.
III. Disorders of large trunks from obliterating lesions.
 1. Basilar syndromes.

CEREBRAL APOPLEXIES.

A tabular summary of all these is to be found at the close of the chapter on Midbrain Disorders (*q. v.*).

Cerebral Apoplexies.—Hemorrhage, Thrombosis, Embolism.—Clean-cut examples of these syndromes are rare. The patients present mixed conditions save in the few instances of embolism due to the closure of a main trunk. For this reason the general rather than the special type of apoplectic attack will be described. Special indications as to the localization of the region involved will be noted later. Medullary, pontine, midbrain, brain-stem, and interbrain localizations have already been discussed, as have also vascular disorders within the cerebellum.[1]

Distribution, Causation.—Men are more often affected (seven to five) than women, and four-fifths of the cases occur after forty years of age. Arteriosclerosis, as indicated, plays the more important role in hemorrhage and in thrombosis, and the smaller, rather than the basal vessels are responsible. Cardiac hypertrophy with increased blood-pressure (180 to 225 mm.) is the chief accompanying factor, and is closely associated with the arteriosclerotic process. Chronic kidney disease is also a frequent concomitant factor (30 per cent.). Exciting causes, such as great physical exertion, lifting heavy weights, coitus, vomiting, coughing, sneezing, etc., were formerly given a prominent place. It is doubtful if they have much importance. In a large series of cases studied by Jones, in a considerable number the stroke occurred within a few minutes after getting out of bed, so that the sudden change in blood-pressure on awakening and getting about was chiefly responsible. Many patients develop apoplexy during sleep, although, other things being equal, sleep is a protection. Psychogenic factors play a much larger role than is at present comprehended. These act chiefly through the maintenance of high blood-pressure states from emotional factors, conscious and unconscious.

[1] Consult recent Traité de Médecine: Gilbert et Thoinot. Handbuch der gen. Physiologie u. Pathologie, Springer, Berlin. Handbuch d. innere medizin, Berlin, Mohr v. Staehelin, etc.

The localization of the hemorrhage, thrombus, or embolus, varies greatly. Right and left sides are about equally involved. Morgagni's early dictum concerning the greater prevalence of right-sided hemorrhages has little support from extensive statistics.[1]

The chief syndromes are of the middle cerebral type. The branches of the lenticulo-striate arteries supplying the internal capsule, caudate nucleus, lenticular nucleus and optic thalamus are most frequently involved. Thus the most frequent syndromes are the combined palsies, arm and leg; arm, leg and face; arm, leg, face with sensory symptoms and aphasic complications.

Cortical hemorrhages are probably much more frequent than is usually supposed since many occur without the death of the patient. These result in more limited syndromes, such as apraxias, arm monoplegias (anterior cerebral syndrome).

Monoplegias of the leg, isolated aphasias, lower quadrant hemianopsias and the thalamic syndrome belong more especially to the middle cerebral syndrome, while mind-blindness and homonymous hemianopsias are the chief features of the isolated posterior cerebral syndromes.

Midbrain, pontine, medullary and cerebellar hemorrhages are comparatively infrequent, and have a special syndromy described elsewhere. (See Chapter VIII, Diseases of the Midbrain.)

The Apoplectic Attack.—Nausea and vomiting are the most frequent precursors of the apoplectic attack. In thrombosis or embolic occluding lesions twitchings or even convulsions are more frequent as precursors of the attack than in hemorrhagic cases. In hemorrhagic cases with convulsions the bleeding is more liable to have extended to the ventricles, and can frequently be demonstrated by lumbar puncture. This is not invariable, however.

The attack is usually abrupt. Dizziness, heaviness, anxiety, headache, paresthesiæ may be described by those who do not at first become suddenly unconscious, and yet in whom a gradually developing state of unconsciousness occurs, with paraphasia and gradual weakness of one side of the body. Many patients are able to describe the beginning symptoms with accuracy after recovery from even a profound coma lasting a week or more. Loss of consciousness is usual, however, especially in hemorrhagic cases, less often present in thrombosis or embolism, especially in the beginning. In Jones' series, 47.7 per cent. of cases of embolism showed loss of consciousness, impairment in 60 per cent. Thromboses show a similar percentage, while hemorrhage is accompanied by loss of consciousness in 75 per cent. of the cases.

Coma is then apt to develop and is accompanied by stertorous breathing, and by a slow, full and regular pulse. The patient is pale, or the face may be congested, the extremities are cold. The limbs are usually completely relaxed. Irritative phenomena are usually

[1] See 995 cases collected by Jones, Brain, 1905.

more indicative of cortical, basal or ventricular hemorrhages. Minute variations in the excursion of the two sides of the chest, irregularity of the usually dilated and light-inactive pupils, minor signs of irritability and reflex defense movements (Marie-Foix) may enable one to locate the side of the hemorrhage even in this comatose state. Other signs are loss of corneal reflexes, no reaction to painful stimuli, loss of reflexes in general, occasionally involuntary urination and defecation. Retention is more apt to occur.

In severe states of coma with marked rise in temperature, 103° F. to 105° F., with twitching or convulsive jerks, very slow heart action, later developing symptoms of irregular heart and breathing, the hemorrhage is probably very extensive, tends to flow into the ventricles, and death ensues in a few hours or a few days.

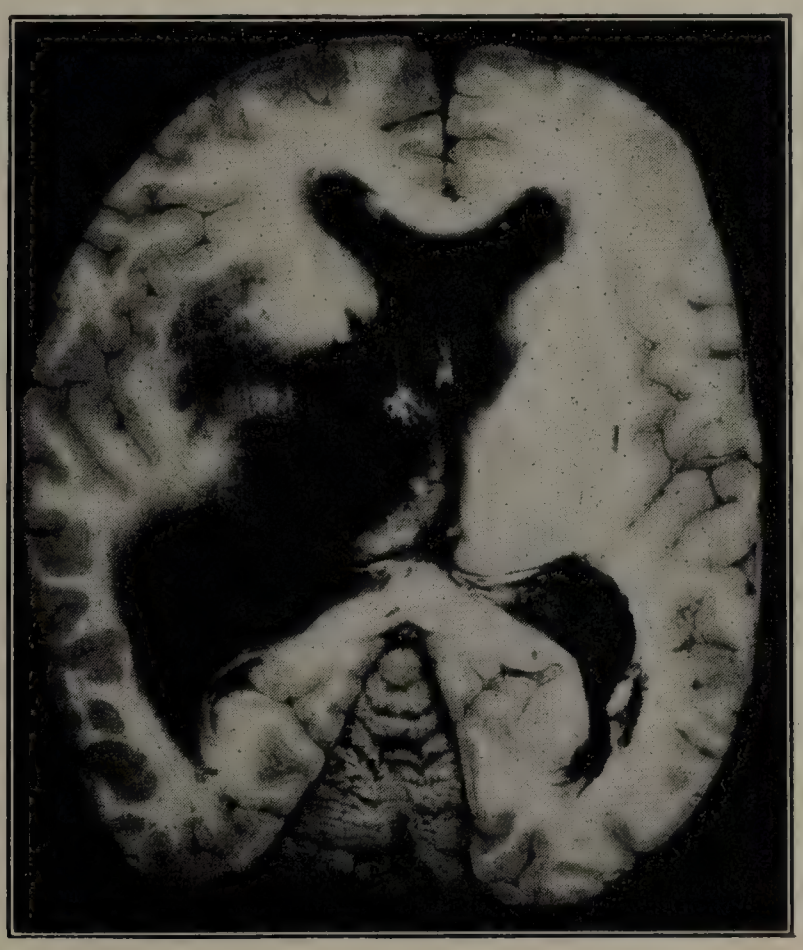


FIG. 363.—Cerebral hemorrhage within the ventricle. (Jelliffe.)

Thalamic cases are frequently attended with marked coma, which may be very protracted, three or four weeks, without definite hemiplegic signs.

Jacksonian convulsive attacks usually indicate meningeal bleeding. Some patients show a tendency to clouding of consciousness which comes and goes.

Recovery from the immediate attack may take place in a few hours or after weeks or more. The residuals found will vary according to the location and extent of the lesion. It is in this stage that a diagnosis of the localization and extent of the lesion can be made.

Anterior Cerebral Syndrome.—In emboli, thrombi and hemorrhage of the anterior cerebral arteries the lesion may be limited or be very extensive.

Mild or severe and acute confusions with variously colored mental defect states are frequently the result of minute or more extensive changes in the vascular supply of the frontal lobes, supplied by the anterior cerebrals. These patients not infrequently present lacunar symptoms when the medullary vessels are involved.[1] The clinical picture is protean, depending on the severity of the hemorrhage, or softening (from embolus or thrombus) and the distribution of these focal softenings. The course is more halting; epileptiform or apoplectiform attacks with acute confusions occur from which the patient is apt to make a clinical, not anatomical, recovery. The patient is usually in the fourth, fifth or sixth decade. There is increasing sense of incompetency. Vertiginous attacks occur, with headaches,

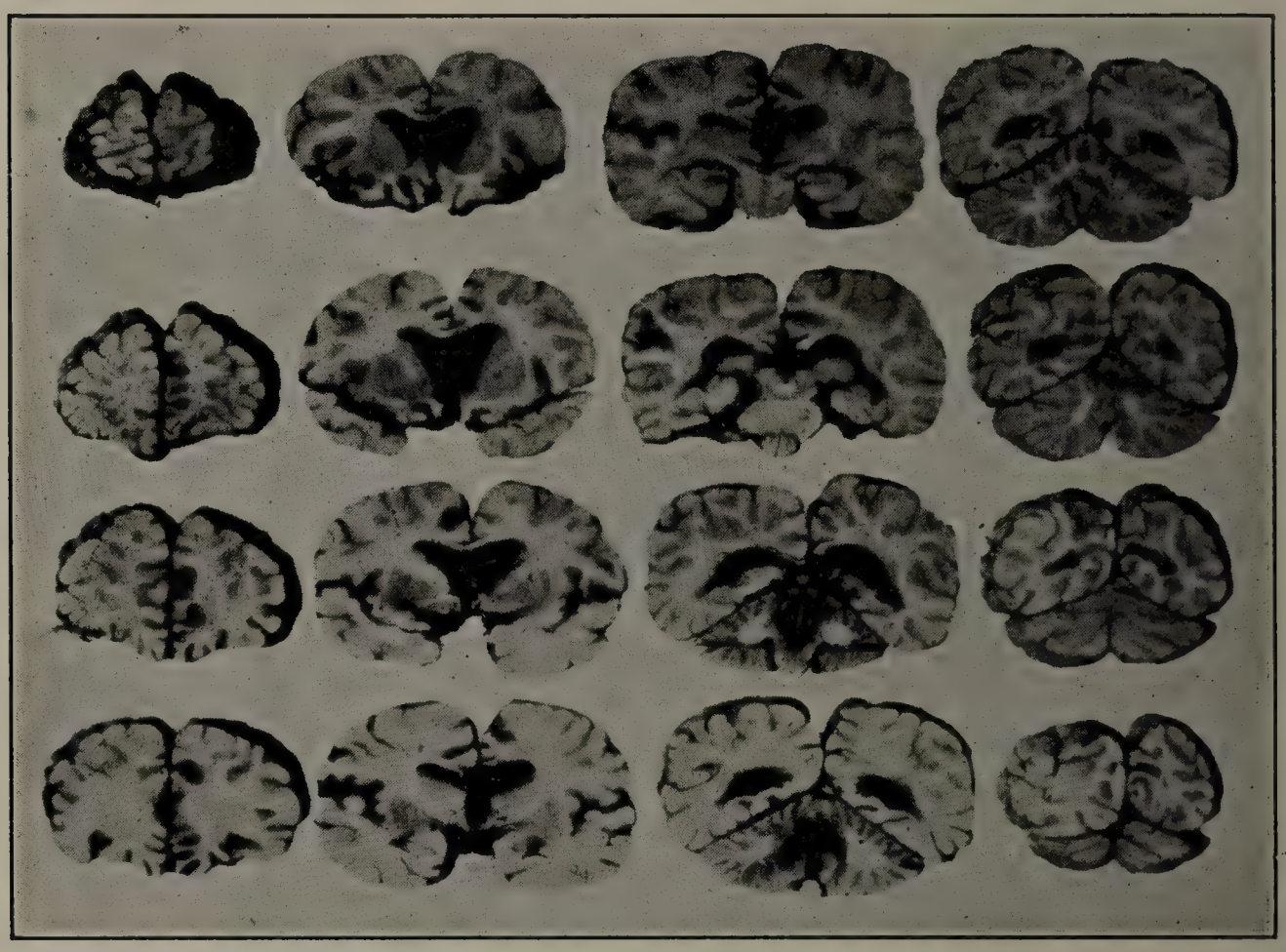


FIG. 364.—Extensive ventricular hemorrhage from rupture of anomalous (duplicated) anterior communicating artery.[2] (H. L. J.)

often migranous in character. Speech and thought associations are interfered with and minor monoplegias develop. Apraxia may be an isolated symptom, or there may be combined apraxia and crural monoplegia.

In more extensive lesions, softenings occur (lacunar), the patients seem to drop large portions of their mental life quite suddenly. These softenings usually show themselves at first as acute excited confusions, sometimes even manic-like in their general trend; others show so-called hysterical coloring; while in the later and more advanced cases apraxias, hallucinoses and more global defects appear.

[1] Lambert: State Hosp. Bull., 1909, **2**, 459.
[2] Tilney. F.: Neurological Bulletin, 1918.

General progression results in marked mental enfeeblement—arteriosclerotic deterioration—or a sudden new and more extensive lesion may cause sudden death.

Irritative complexes from cortical vascular disease develop not infrequently, even in younger individuals following various infections (thrombi, emboli of cortical vessels), alcoholism, syphilis, lead. Here transitory mild shock may usher in the disorder with twitching, jerky, choreiform accompaniments, or these motor disturbances may later develop as residuals, with or without epileptiform attacks. Paresthesiæ, astereognosis, weakness of an arm or leg, or mild paresis of the facial musculature may develop. True epileptic residuals may be permanent symptoms of certain of these cortical arteriosclerotic focal lesions.

The *middle cerebral syndromes* are the most frequent. Here *hemiplegia* is the most striking residual symptom. In total hemiplegia, usually capsular, supracapsular, the face, arm and leg of the same side are involved. The upper branches of the facial, wrinkling of forehead, closure of eye are not usually implicated. Nor are the eye movements modified unless the lesion is located lower down, crura or in the midbrain, when another syndrome is present.[1] The face is drawn to the healthy side, the tongue projects to the paralyzed side on protrusion. Dysphagia, from hemiparesis of the palate develops. The soft palate hangs lower on the paralyzed side. The neck may be involved, but is less apt to be, whereas the paralysis of the arm and leg are characteristic. (See Plate XII.)

The arm is flaccid, flabby, apt to be edematous, blue, cold and boggy. The reflexes which during coma are lost, gradually return and shortly become exaggerated. Chaddock's wrist phenomenon develops. There is little or no atrophy and the electrical reactions are normal. Later contractions may develop, or these may occur closely following the attack if the lesion involves only a portion of the arm fibers or encroaches upon the thalamus. The abdominal reflexes are diminished or lost on the paralyzed side.

The leg is flaccid, is thrown about the hip like a flail, may be edematous and flabby, and also shows later the signs of pyramidal tract, upper motor neuron disorder, *i. e.*, increased knee-jerks, ankle-clonus, Babinski and Chaddock reflexes, with increased muscle tone and normal electrical responses.

The grade of the paralysis varies greatly. With some it is a transitory weakness which passes in a few weeks or a few months, in others it is a complete, permanent and marked paralysis, with later developing contractures and total unilateral disability. Any intermediary grade may be expected. In the milder forms of hemiplegia the little signs—see Examination Methods—are of great value to clearing up the situation, since some mild hemiplegias may be confused with psychogenic palsies, especially in thalamic cases.

[1] Millard-Gubler, Benedict, etc., *q. v.* Maurice-Lévy: Les Ramolissements Sylviens, Paris, 1927.

Hemianesthesia may accompany the hemiplegia, or the patient may have a hemianesthesia without any, or with minimal hemiplegia. This argues for the implication of a portion of the thalamic fibers.

The patients complain of numbness, coldness, and, depending on the location of the lesion, may lose their stereognostic sense, may not be able to distinguish heat, nor cold, or may have subjective sensations, haptic hallucinations of the limb area.

Sensory disturbances from cerebral lesions are extremely diverse and of great importance in the diagnosis of brain disorder, particularly in arterial disease and in brain tumors. They are best discussed as parts of the thalamic or suprathalamic syndromes.

The involvement of the hypothalamic or lenticular regions may cause muscle vegetative signs, various tonic anomalies, etc.

The Thalamic Syndromes.[1]—If certain nuclear masses within the thalamus are involved with partial implication of related extrapyramidal tracts, a characteristic neurological complex results, termed by Dejerine and Roussy the thalamic syndrome. It is one of the middle cerebral artery combinations.

The chief features show usually after an apoplectic attack with the ordinary signs of a severe hemorrhage, thrombosis or embolism. In certain syphilitic cases the attack may be comparatively slight, or the syndrome may develop with no signs of an attack.

Practically the entire mass of sensory fibers carrying impulses of all kinds—the tests for most of which have already been outlined—have their synaptic junctions within the optic thalamus. These synaptic junctions *are* the thalamus. Only the most limited attention can be given here to the numerous fibers coming from the chemical receptors of the respiratory, gastro-intestinal, or genito-urinary tract, or to those from the organs of internal secretion. Some of these undoubtedly made their synaptic junctions in other than thalamic structures—globus pallidus, putamen, hypothalamus, locus niger, etc., for example. The thalamic (or related) synapses of these vegetative pathways have not yet been sufficiently worked out for didactic presentation. (See Lenticular Syndromes, Encephalitis Epidemica.)

The chief features of the thalamic syndrome as originally described are:

1. A persistent loss of superficial sensation of one-half of the body and face. This loss to touch, pain, and to temperature is more or less definite, subject to considerable variation and to partial recovery, but the loss of deep sensibility, deep pressure, postural sense, etc., is much more pronounced, and is more apt to persist. This latter is usually more marked distally and in many instances diminishes proximally.

2. There is slight hemiataxia and more or less complete astereognosis.

[1] Roussy, G. in Les Actualités Neurologiques, Paris, 1922; Jelliffe: The Thalamic Syndrome, Med. Record, February 1, 1910, for early references.

3. There are, in the complete syndrome, acute pains on the affected side, which are very persistent, coming on in paroxysms. They are frequently extremely severe and rarely controlled by the usual analgesics. These pains may involve a single member, may be limited to the side of the face, constituting a thalamic trigeminal neuralgia, or they may involve one whole side of the body.

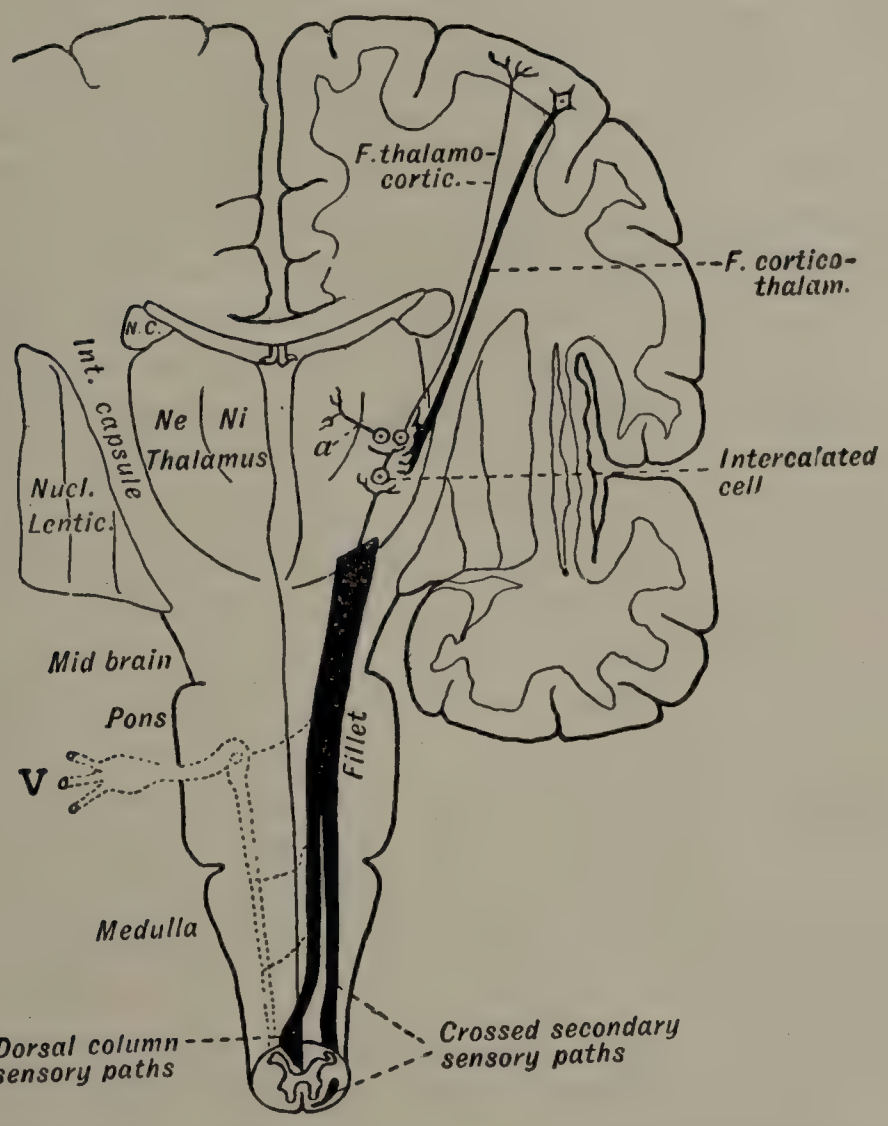


FIG. 365.—To show the position and relations of the optic thalamus in the central sensory path. Two distinct paths exist in the spinal cord: a crossed secondary path in the ventrolateral column which conveys impressions of pain, temperature, and touch, and a second uncrossed path in the dorsal column which also carries touch, and in which run impulses that underlie the sense of position, the appreciation of movement, the discrimination of two points, and the recognition of vibration, size, shape, form, weight, and consistence. This second path decussates in the lower part of the medulla oblongata, but runs separate from the first path, at least as high as the pons. All these secondary sensory fibers, now crossed, terminate in the ventrolateral region of the optic thalamus. The impressions they carry are regrouped here and, through intercalated neurons, are distributed along two distinct paths; the one carries impressions to the cerebral cortex, the other toward the more mesial parts of the optic thalamus. The corticothalamic fibers, which terminate in the lateral nucleus of the optic thalamus, are also shown. (Head and Holmes.) (Compare Plate XIII.)

4. There is usually a more or less distinct, though slight hemiplegia which, in the unmixed syndromes, rapidly clears up. Contractures rarely develop in the pure syndrome. In the mixed syndrome—with extension of the lesion to the internal capsule—contractures may be present.

5. Choreic, athetoid or paralysis-agitans-like movements may be present on the affected side.

These are the chief symptoms of optic thalamus disease, but in addition to these, Head and Holmes have pointed out an extremely suggestive series of affective reactions which are due to lesions which cut certain of the optic thalamus nuclei from their cortical connections. They have opened up an attack upon the analysis of the sensory content of emotional reactions. They show that in this general type of lesion there is a tendency to react excessively to pleasant or unpleasant stimuli. The prick of a pin, painful pressure, excessive heat or cold, all produce more distress than on the normal half of the body. Thus, in one of Head and Holmes's patients, if a pin was dragged lightly across the face or trunk, from the sound to the affected side, there was felt an excessive discomfort as it passed the middle line. The patient not only complained that it hurt, but the face was contorted with pain, and all this notwithstanding the fact that there was less ability to distinguish head from point, yet the prick was more disagreeable. This anomalous state of affairs is characteristic of a thalamic involvement.[1]

This excessive affective reactivity is present not only to pin-prick, but also to deep pressure, to extremes of heat and cold, to visceral stimulation, to scraping, roughness, vibration, tickling, to pleasurable stimuli and to ideational emotional states. Not all patients show all of these reactions, but in practically 90 per cent. of the thalamic cases examined by Head and Holmes, excessive affective response to one or more measured stimuli was found. For heat and cold and other forms of sensibility as well as for pain, the excessive response may be present, and yet the patients are unable to detect—*i. e.*, are anesthetic to—the stimulus itself. So far as the ideational affective reaction is concerned, these patients express themselves as follows: On hearing affecting music, "A horrid feeling came on in the affected side, and the leg screwed up and started to shake." The singing of a comic song left one patient absolutely cold, but a tragic song produced a very distinct unpleasant effect. One patient said, "My right hand seems to crave sympathy, my right side seems more artistic." In practically all of the cases the increased affective reaction was accompanied by actual sensory loss.

A more detailed study of the loss of sensibility in thalamic disorders made by Head and Holmes revealed the following: No sensory functions are so frequently affected as the appreciation of posture and the recognition of passive movement. The amount of this loss varies greatly from a scarcely measurable defect to complete want of recognition of the posture of the limbs of the abnormal half of the body.

Tactile sensibility is frequently diminished, but, excepting in a few cases, where all appreciation of contact was destroyed, a threshold could be obtained. It was always possible to show that increasing the strength of the stimulus improved the proportion of right answers

[1] Byrnes, J.: Jour. Nerv. and Ment. Dis., 1919; Alienist and Neurologist, 1919.

unless the observations were confused by the disagreeable tingling or other accessory sensations.

Localization of the spot touched was defective in half the cases where sensation was sufficiently preserved to carry out accurate tests. This inability to recognize the site of stimulation was equally great, whether the patient was pricked or touched. In cases where localization was gravely affected, the disagreeable sensation, so easily evoked, tended to spread widely on the abnormal half of the body. A prick on the hand may cause an extremely painful sensation in the cheek or side, and sometimes the patient simply recognized the stimulus as a change within himself, and did not refer the discomfort from which he suffered to the action of any external agent.

Sensibility to heat and cold may show all degrees of change from total loss to a slight increase of the neutral zone. Heat and cold are rarely dissociated[1] and if one form of sensation is lost, the other will be gravely disturbed. The apparent exceptions arise from a misinterpretation of the sensation evoked by high or low temperatures on the affected half of the body.

Not infrequently the compass test cannot be carried out because of the gross loss of sensation and inability to recognize contact; but whenever this method can be applied a threshold can be worked out, and widening the distance between the points increases the accuracy of the answers.

The power of estimating the relation between two weights is frequently disturbed on the involved half of the body. If the appreciation of posture and movement is affected, the patient can no longer recognize the identity or the difference of two weights placed on the unsupported hands. But so long as tactile sensibility is not diminished, the patient can still estimate the relation between weights applied one after the other on the same spot, and can recognize the increase or diminution in weight of an object already resting on the hand.

The appreciation of relative size is often disturbed in these cases. With care it is usually easy to demonstrate a difference in the threshold. Shape and form in three dimensions are frequently not recognizable on the affected hand. But, if tactile sensibility is not grossly affected, the patient usually retains an idea that the object possesses a form, and may give a considerable percentage of right answers.

Vibration of the tuning-fork is felt, in almost all thalamic cases, but the length of time during which it is appreciated is usually shorter, and sometimes the rate of vibration is thought to be slower on the affected half of the body.

Roughness, as tested with Graham Brown's esthesiometer is usually recognized, except in those cases in which the loss of all forms of sensation is unusually severe.

Partial syndromes are more frequent than the classical syndrome

[1] Tilney and Cornell, report a Thalamic Syndrome with Thermal Dissociation, Neurol. Bull., 1918, vol. 1.

described and more detailed analysis of which nuclear masses are involved is necessary to give a complete picture of the many thalamic syndromes which are possible. The thalamus is a highly complex switch board. To summarize: The main symptoms of the complete syndrome are:

1. Persistent hemianesthesia, more or less marked for superficial sensibility, tactual pain, temperature, most marked for deep sensibility and hence loss or diminution of postural sense and astereognosis.
2. Mild hemiplegia, usually regressive.
3. Mild hemiataxia, with choreo-athetoid movements either spontaneous or on an attempted movement. Adiadokokinesis at times.
4. Paroxysmal, shooting, neuralgic pains, often very persistent and severe and not helped by the usual analgesics.
5. Overresponse to affective stimuli, particularly on affected side, with emotional hyperactivity to varied sensory impressions.

Sensory Changes in Cortical Lesions, Suprathalamic Pathways.—The analysis of sensory stimuli in cerebral and thalamic disorders is of the greatest importance in cerebral localization, hence an extended presentation of contemporary work is desirable. In the chapter on Peripheral Nerves the chief sensory syndromes of the first sensory neurons were discussed. Those of the cord are presented elsewhere. Redistributions take place in the medulla and midbrain. Those disturbances due to lesions involving some of the thalamic nuclei have just been presented. Finally the thalamo-cortical pathways make a further regrouping of sensory qualities in the entire course of the sensory pathways. (See Plates XII and XIII.)

The analysis of these phenomena introduces complex factors, and it is necessary to abandon all generalizations, even, for instance, light touch, cutaneous sensation. The results of the test must be stated in terms of the tests employed. (See Sensory Examination Methods.)

Using graduated tactile stimuli, such as von Frey's hairs and the pressure esthesiometer, the chief results have been that a cortical lesion may reduce the accuracy of response from the affected part to graduated tactile stimuli. The form assumed by this defective sensibility differs from that produced by lesions at other levels of the nervous system. The affected part may respond to the same graduated hair as the normal part; but this response is irregular and uncertain. Increasing the stimulus may lead to no corresponding improvement, and even the strongest tactile hair may occasionally evoke less certain answers than a hair of much smaller bending strain. Moreover, a touch with the unweighted esthesiometer may be as effective at one moment as the same instrument weighted with 30 grams at another. In such cases no tactile threshold can be definitely obtained. This irregularity of response is associated with persistence of the tactile sensation and a tendency to hallucinations of touch. Where the sensory defect is not sufficiently gross to abolish the threshold, persistence, irregularity of response and a tendency to hallucinate may still disturb the records.

In all cases where tactile sensibility is affected, whether a threshold can be obtained or not, fatigue is induced with unusual facility. Although the patient may cease to respond to tactile stimuli over the affected part in consequence of fatigue, his answers may remain as good as before from the normal parts. The fatigue is local and not general.

With stationary cortical lesions, uncomplicated by states of shock or by "diaschisis," sensibility to touch with cotton-wool is rarely lost over hair-clad parts. Over hairless parts, stimulation with cotton-wool may produce a sensation which seems "less plain" to the patient, and his answers may show the same inconstancy so evident when he is tested with graduated tactile stimuli.

A pure cortical lesion leads to no change in the threshold to measurable, painful or uncomfortable stimuli. Nor does the patient express greater dislike to these stimuli on one side than on the other. A prick may be said to be "plainer" or "sharper" on the normal than on the affected side; but this is due to a defective appreciation of the pointed nature of the stimulus and bears no direct relation to the painfulness of the sensation evoked.

Temperature tests show that (*a*) the neutral zone, within the stimulus, was said to be neither hot nor cold, was considerably enlarged in comparison with that observed on similar normal parts of the same patient. (*b*) The patient complained that, although he recognized correctly the nature of the stimulus, it seemed "less plain" than over normal parts. His answers were less constant, and less certain; a temperature recognized without difficulty at one time seemed doubtful at another. (*c*) The power of discriminating the relative coolness of two stimuli or the relative warmth of two hot tubes may be diminished. Thus 20° C. may be said to be the same as ice, although both are uniformly called cold, and 40° C. may seem as warm as or even warmer than 48° C. The faculty of appreciating the relation to one another of two temperatures on the same side of the scale is disturbed.

Tests for *posture* and for *passive movements* show that (*a*) cortical lesions most frequently disturb the recognition of posture and of passive movements. Whenever sensation is in any way affected in consequence of a cortical lesion these two functions suffer. (*b*) In all their cases the disturbance in the faculty of recognizing posture and passive movements was greater toward the peripheral parts of the affected limb. (*c*) When a patient with unilateral disturbance of these faculties attempts to point to some part of his body, defective knowledge of its position causes greater error than want of recognition of posture and movement in the hand with which he points. (*d*) When testing the patient's power of appreciating passive movement, the answers are frequently uncertain and hallucinations of movement may occur. And yet the patient may be remarkably consistent and accurate when normal parts are tested.

Localization tests show that (*a*) the power of localizing the stimulated spot is not infrequently preserved, although sensation may be otherwise disturbed as a consequence of cortical lesions. (*b*) This faculty is independent of the power of recognizing the position of the affected limb; appreciation of posture may be lost, although localization is not in any way diminished. (*c*) If the power of localization is lost, the patient will be unable to recognize not only the position of a spot touched but also the position of a prick. (*d*) When localization is defective in consequence of cerebral lesions, the patient does not habitually localize in any particular direction, but ceases to be certain where he has been touched or pricked.

The *compass test* shows that (*a*) a cortical lesion may destroy the power of discriminating two compass points, both when applied simultaneously and consecutively. If this is the case, no threshold can be obtained for either form of the test; increasing the distance between the points does not constantly improve the accuracy of the answers. (*b*) This disturbance is not caused by changes in tactile appreciation; for it can be demonstrated equally well with two painful as with two tactile stimuli. (*c*) The condition of tactile sensibility and the accuracy of the simultaneous compass test are closely associated; a disturbance of the tactile threshold is usually accompanied by a raised threshold for the appreciation of two points applied simultaneously. (*d*) Should the power be preserved of recognizing two points when the compasses are applied consecutively, localization will be found to be intact. The patient's appreciation of the two points when they are separated by an interval of time is due to the recognition of the separate locality of the two spots touched.

Appreciation of weights shows that (*a*) the power of estimating the relation of two objects of the same size and shape is readily disturbed by cortical lesions. (*b*) Though the patient may retain sensations of contact when the weight is placed in his hand, all power of recognizing the relative heaviness of the object has disappeared. (*c*) This faculty is equally disturbed in most cases whether the weights are placed on the supported or the unsupported hand.

From these and related studies, Head and Holmes maintain that sensory impulses pass from the thalamus to the cortex in five groups:

1. Those concerned with the recognition of posture and passive movement. If these impulses are affected the power of discriminating weights on the unsupported hand may be also diminished.

2. Certain tactile elements; integrity of this group is necessary for the discrimination of weights placed on the fully supported hand.

3. Those impulses which underlie the appreciation of two points applied simultaneously (the compass test); on this group also depends the recognition of size and shape.

4. Those which underlie the power of localizing the situation of a stimulated spot. Recognition of the double nature of two points applied consecutively also depends on this group of impulses.

5. All thermal impulses are grouped together to underlie a scale of

sensations with heat at one end and cold at the other. At the level with which we are now dealing these impulses have already excited the affective center and are passing away to the cortex.

The functional integrity of the cortex enables attention to be concentrated upon those changes which are produced by the arrival of afferent impulses (Head and Holmes). When this is disturbed, some impulses evoke a sensation, but others, from lack of attention, do not affect consciousness. Attention no longer moves freely over the sensory field to be focussed successively on fresh groups of sensory impressions. Sensations, once evoked, are not cut short by the moving away of the focus of attention as when cortical activity is perfect. Hence arise persistent sensations and hallucinations which are so prominent a feature of lesions of the cortex.

The cerebral cortex is the organ by which attention may be focussed upon the changes evoked by sensory impulses. Such attention is to further the useful work in hand. A pure cortical lesion, which is not advancing or causing periodic discharges, will change the sensibility of the affected parts in such a way that the patient's answers appear to be untrustworthy. Such diminished power makes the estimation of a threshold in many cases impossible. Uncertainty of response destroys all power of comparing one set of impressions with another and so prevents discrimination. This interferes with function.

In addition to its function as an organ of local attention the sensory cortex is also the storehouse of past impressions. These may rise into consciousness as images, but more often, as in the case of spatial impressions, remain outside of central consciousness. Here they form organized models which may be termed "schemata." Such patterns modify the impressions produced by incoming sensory impulses in such a way that the final sensations of position, or of locality, rise into consciousness charged with a relation to something that has happened before. Destruction of such "schemata" by a lesion of the cortex renders impossible all recognition of posture or of the locality of a stimulated spot on the affected part of the body.

In daily life all stimuli excite more or less both thalamic and cortical centers, for most unselected sensations contain both affective and discriminative elements. But among the tests employed in sensory analysis, some appeal almost entirely to the one or the other center. The test for recognition of posture, as carried out by Head and Holmes, is purely discriminative; while the pain produced by squeezing the testicle, or to a less degree by the pressure algometer, appeals almost exclusively to the more affective center.

Sensory impulses arriving at the optic thalamus are regrouped in such a way that they can act upon both what Head calls its essential center and the sensory cortex. The essential organ of the thalamus is excited to affective activity by certain impulses, and refuses to react to those which underlie the purely discriminative aspects of sensation. These pass on to influence the cortical centers where they are readily accepted. In a similar way, the primary centers of the cortex cannot

receive those components which underlie feeling tone; in this direction they are completely blocked.

The work of Head and Holmes tends to show that the sensory cortex is the organ by which attention can be concentrated on any part of the body that is stimulated. The focus of attention is arrested at any one spot by the changes produced by cortical activity. These are sorted out and brought into relation with other sensory processes, past and present. Then the focus of attention sweeps on, attracted by some other stimulus.

All stimuli which reach as high up as the thalamic center have a high threshold. They must reach a high intensity before they can enter consciousness, but once they have risen above the threshold they tend to produce a change of excessive amount and duration, and this it is the business of the cortical mechanism to control. The low intensity of the stimuli that can arouse the sensory cortex, and its quick reaction period, enable it to control the activity of the cumbersome mechanism of the thalamic center.

This view of the sensory mechanism helps to explain many of the facts recognized by students of disorders of the nervous system. It enables one to understand how integrations can occur at all afferent levels of the nervous system, and make development possible even in the individual. An aim of human evolution is the control of feeling and instinct by discriminative mental activities. This struggle on the highest plane of mental life is begun at the lowest afferent level, and the issue becomes more clearly defined the nearer sensory impulses approach the field of consciousness. Finally, in terms of Bergson, "the cerebral mechanism is arranged just so as to thrust back into the unconscious almost the whole of our past and to allow beyond the threshold only that which will further the work in hand, *i. e.*, do *useful* work."

Cortical Sensory Disturbances.—Continuing his researches to the cortex, Head,[1] from an analysis of a large number of war injuries of the brain, first calls attention to the fact, that a gross lesion of the so-called "sensory cortex" does not abolish any of the four primary qualities of sensation, as does a lesion of the brain-stem or spinal cord, and yet in a case of such cortical injury the answers of the patient to tests show that these qualities are in some way disturbed. It is to the precise examination of these disturbances that Head has applied his experience, and he finds that if the sensibility of the hand, for example, be carefully tested, disturbances, perhaps confined to certain digits, will be revealed. These disturbances will affect, not crude recognition of touch, pain, heat and cold, but three discriminative faculties: (1) The recognition of spatial relations, in particular the recognition of passive movement of the affected part. Discrimination of two compass points and the topical localization of stimuli, which come within this faculty, may also be disturbed, but in lesser degree.

[1] Head: Brain, 1918, Pt. II.

(2) Appreciation of differences in intensity of pain, touch, heat and cold. To test capacity in estimating these differences is hard. "Appreciation of heat and cold, as one is forced to examine it clinically, depends comparatively little on cortical activity. For while it is easy to say whether an object is hot or cold, it requires an intelligent patient and unusually favorable conditions to test the relation of two warm stimuli to one another, or to determine the threshold for heat and cold with approximate accuracy. These difficulties are mul-

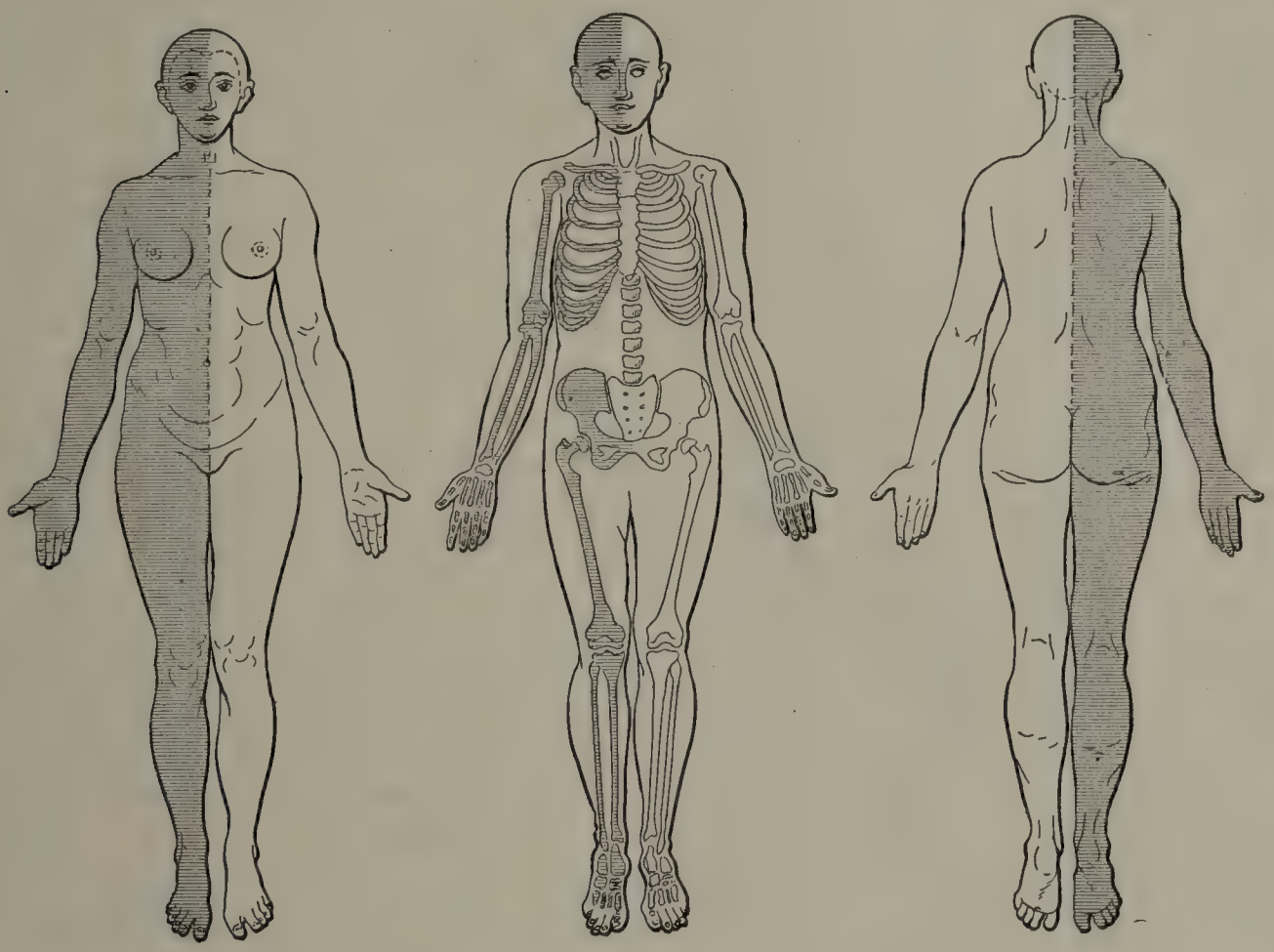


FIG. 366 FIG. 367 FIG. 368

FIGS. 366, 367 and 368.—Topography of the sensory disturbance, in a case of thalamic syndrome, right side, of six years' duration in a woman, aged fifty-five years. There is mild hemiplegia of the right side with intense choreo-athetoid movements of the hand and foot. There is a marked ataxia in all voluntary movements of the same side. Exaggeration of the tendon reflexes of the right side. No Babinski reflex.

There are very active and severe spontaneous pains over the entire right side; tactile pain and thermal sensibility is diminished, but not abolished, on the entire right side. Compass discrimination markedly affected. Complete astereognosis. Taste, smell and hearing are diminished on the right side. Vision is unimpaired and there is no hemianopsia. Deep sensibility is more altered than superficial. The sense of position is altered, bony sensibility is much diminished. (Thomas and Chiray.)

tipled a hundredfold in the case of pain." In the case of touch, however, useful results may be obtained by measurement with tactile hairs of graduated strength. (3) The recognition of similarity and difference, thus, the size, shape and weight of objects and the texture of stuffs, apart from their purely tactile qualities. These are the three faculties which fundamentally depend upon the activity of the sensory cortex.

How does the cortex handle and utilize the sensory material it receives? In doing this the observer must rely upon the study of

dissociated sensibility, of the consequences produced by interferences at various known points in the nervous system, where some impulses are intercepted, while others pass on to reach the highest receptive centers and form the underlying basis of an abnormal sensation. "The clue to the situation is given by the behavior of the optic thalamus. Until recently the part played by this organ was unknown. But it is now recognized that it is the seat of those physiological processes which underlie crude sensations of contact, pain, heat and cold, together with the feeling tone they evoke. The essential organ of the optic thalamus is the center for the affective aspect of sensation, while discrimination and spatial projection are the product of cortical activity." And it follows that if a lesion be subcortical a mixed disturbance will result, one having both thalamic and cortical characters, either dominating in accordance with propinquity to the respective levels. Touching the term "level," Head is careful to point out that, following Hughlings Jackson, he uses the term in its physiological rather than its anatomical sense. In addition to sensory changes, an uncomplicated lesion of the sensory cortex may produce hypotonia; this is a slight but unmistakable change, best seen in the hands, completely different from paralytic hypotonia and closely associated with the failure to recognize posture and passive movement. Concerning the anatomical localization of these cortical sensory functions, they may be placed in the pre- and postcentral convolutions, the anterior part of the superior parietal lobule and the angular gyrus. But it is the functions rather than the anatomical relations of any one part of the body that are represented in these centers. Hence, those portions, such as the hand, which are endowed with the highest powers of discriminative sensibility, are most exclusively represented. Next in order comes the sole of the foot, which constantly exerts a discriminative action in walking. Accordingly, a cortical lesion may disturb the sensibility of the hand and foot without of necessity affecting the elbow, shoulder or knee.

Apraxia.—This term was first used by Gogol in 1873, in a Breslau thesis on Aphasia. His patient ate his soap, urinated in his water pitcher, and was described as having lost his understanding for objects. Such defects had been noted before, and it is worthy of note that Hughlings Jackson, in 1866, called attention to a similar type of phenomenon, and attached much importance to it. Quaglino, in 1867, described a case; Finkelnburg, in 1870, another, in which recognition of things and people was lost, and created the term asymbolia. Wernicke, in 1874, expanded the term asymbolia; while Freud finally utilized the term agnosia, to cover all types of loss of sensory or motor object images, apraxia then being arranged as a form of loss of knowledge of objects, really a form of visual agnosia. The superficial observer calls these patients "demented."

From this early use of the term apraxia, there has been a distinct variation, brought prominently into the foreground by Liepmann in 1900. He defined the disturbance as a lack of knowledge of the

use of objects, although there was no true agnosia, or loss of recognition of what they were. Out of the later studies of Liepmann,[1] Pick, von Monakow, d'Hollander and others has come the following general description of apraxia.

It consists in an inability to perform certain subjectively purposeful movements, or movement combinations, the motor power, sensation, and coördination being intact. Such an inability will naturally depend upon at least three factors; one may be unable to recognize the object which is to be used, in which case there is a sensory apraxia, in the same sense as one speaks of a sensory aphasia, or a visual agnosia. Should the patient recognize the object, call it perhaps by name,

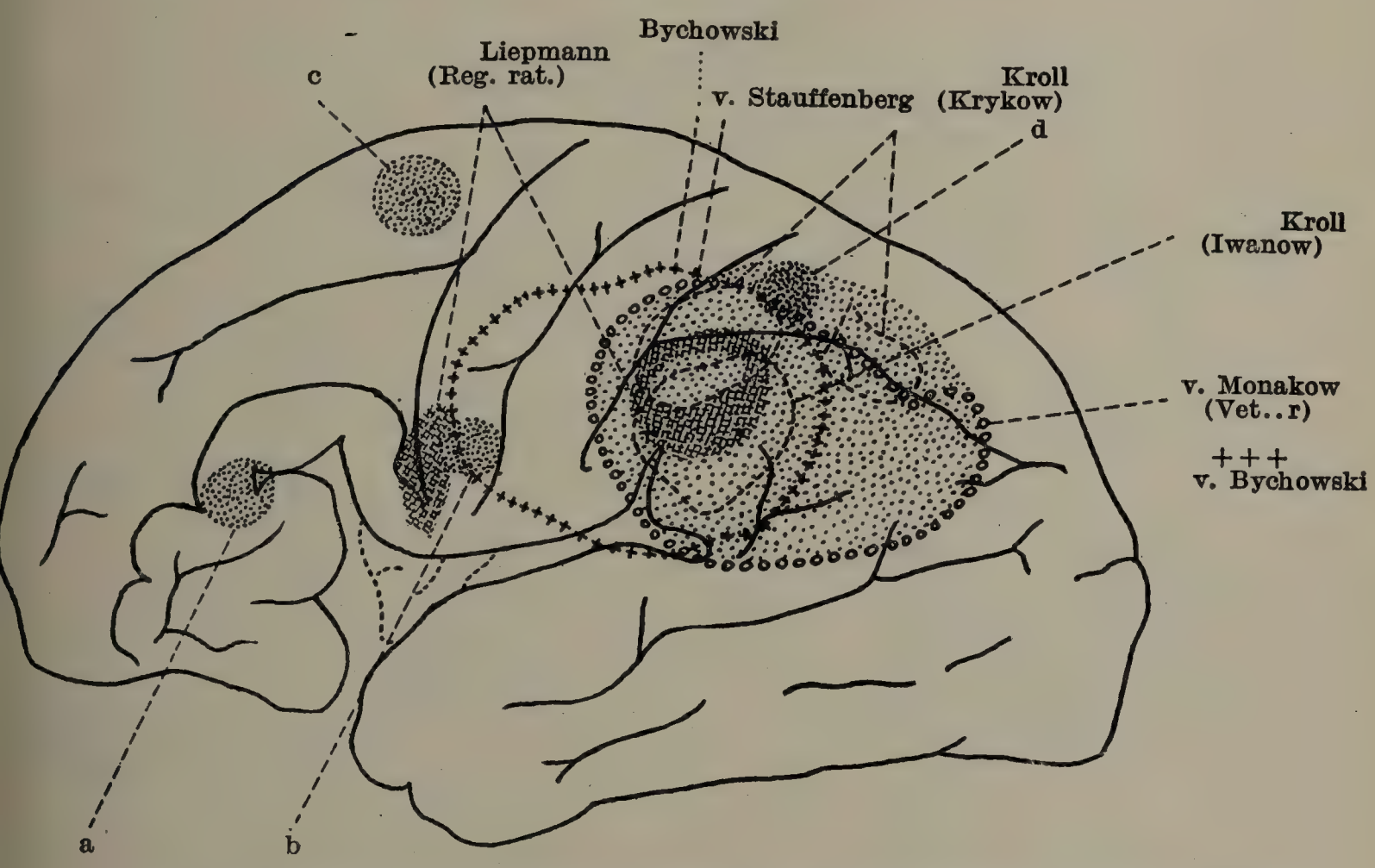


FIG. 369.—Localization of apraxia. Positive cases from lesion in the left supramarginal gyrus and (*a*, *b*, *c*, *d*) small metastatic tumors from one case of total bilateral apraxia. In this case foci were also found in both optic thalami. (von Monakow.)

state its use, and yet in attempting to use it totally fail in proper motor act, it is termed a motor apraxia; it being understood here that there is no necessary change in the motor arc, either on the incoming sensory or outgoing motor side. Thus one can speak, as Wilson has done, of a motor aphasic, as having an apraxia of his speech musculature. In grave intracerebral changes the knowledge of the proper kinetic images to carry out purposeful actions in the arms and legs may be complexly involved. This is termed an intrapsychic apraxia. Clinically it is usually overlaid in the general psychic loss, and is often included in the loose and unprecise term dementia.

[1] Liepmann, Kraus-Brugsch: Ergebnisse d. Med.

Three types of apraxia are distinguishable: (1) Innervatory apraxia, or motor apraxia, sensu strict. Here the motor activities are stiff and awkward and the sensori-motor acts, such as buttoning, are greatly disturbed. Lesions in the strio-pallidal system play a definite

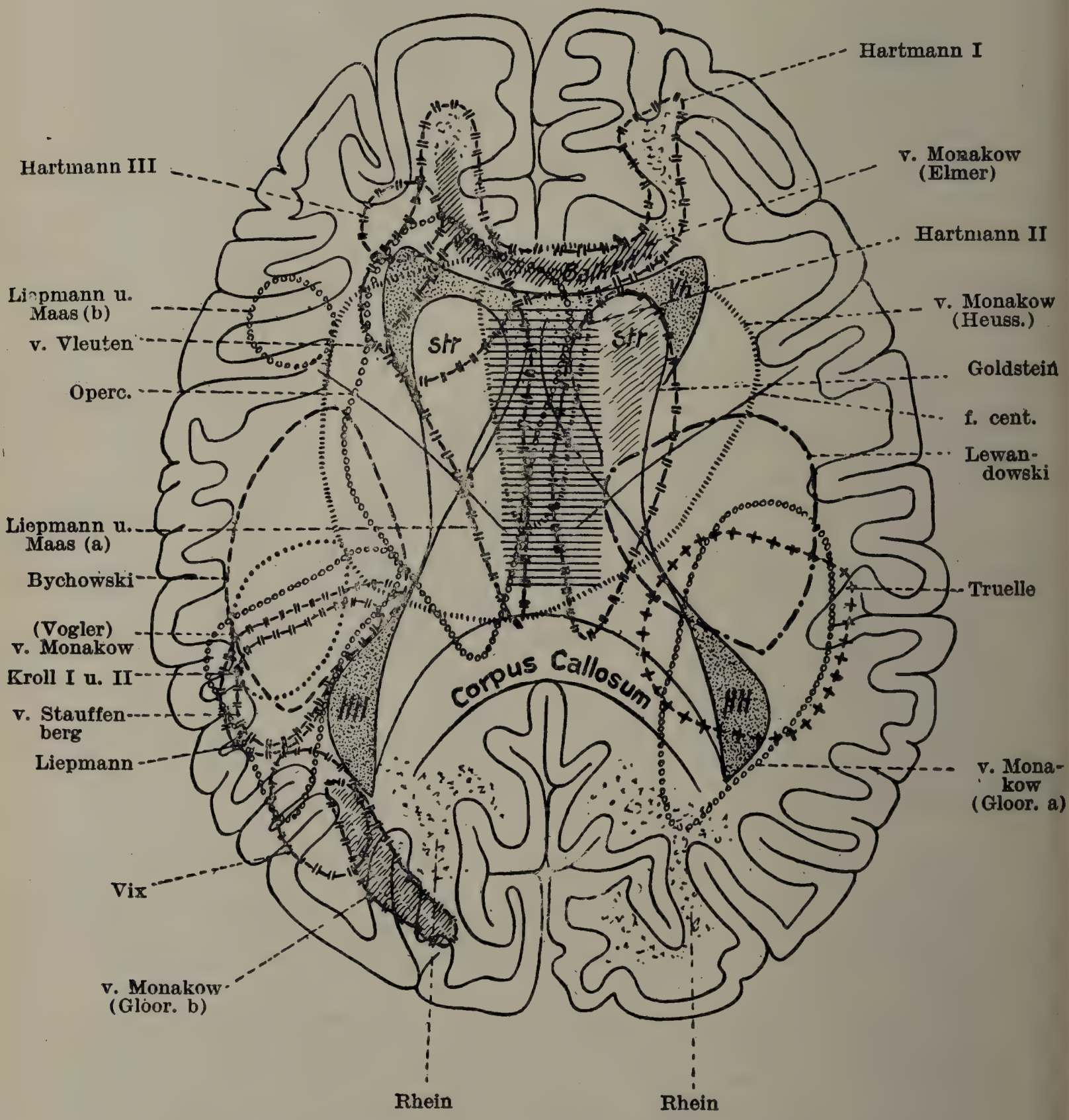


FIG. 370.—Site of the lesion in 20 cases of apraxia marked according to the author's localization. The shaded places in the illustration indicate the spreading of the lesion in certain cases. *Str*, corpora striata; *VH*, anterior horn of ventricle; *HH*, posterior horn. (von Monakow.)

role, probably, in this type.[1] (2) Ideomotor apraxia, the type of Liepmann's case. (3) Ideational type in whom the motor schemata of

[1] Kleist: Mon. f. P. v. N., 1922, **92**, 7.

things are disturbed. For (2) and (3) the supramarginal and angular gyri and other parts of the occipital and parietal lobes are of importance.

Liepmann's celebrated patient was able to do things with his left hand, but failed with his right. When told to brush the examiner's coat, he picked up a corner of it carefully in his left hand, then picked up the brush in his right hand, with which he made movements as if to brush his hair. Asked to pour water into a glass from a carafe, he grasped the carafe with his left hand, to pour water into the glass held in the right hand, after which the glass was brought to the mouth without any water in it. These patients fail to carry out the simple commands to blow a kiss, make a threatening fist, soldier's salute, etc.

In ideomotor apraxia the situation is more complicated. One patient given a tooth-brush recognized it, then began to brush his beard with it clumsily; another being given a pistol, which he named correctly, on being told to shoot it, grasped the barrel, blinked and put the muzzle into his left eye. Another patient, being given a cigar and a match-box opened the latter, stuck the cigar in it, and tried to shut the box as though it were a cigar cutter. Then taking the cigar out rubbed it on the side of the box as though it were a match. The entire order of procedure was badly devised.

Apraxia is usually an accompaniment of certain apoplectic attacks. It may also be present from tumors. The lesions center about the fibers of the corpus callosum and the medullary fiber areas from the second parietal region.

Hemianopsia is an infrequent sign occurring as a result of apoplexy. As a symptom of middle cerebral disease it may result from thalamic lesions (pulvinar, geniculates) and is usually a permanent residual. It is rarely a temporary condition but may show marked improvement. The type is a bilateral homonymous hemianopsia. Quadrant hemianopsias more often belong to the middle cerebral syndromes, although limited lesions of the cuneus may cause quadrant hemianopsias.[1]

The visual fields will vary greatly according to the tract involvement. In posterior cerebral syndromes occipital cortical types of hemianopsia are present. These show very irregular visual fields. Psychical blindness (loss of visual memories) may also result from posterior cerebral involvements. Here the portions of the occipital cortex about the calcarine fissure and cuneus are implicated. It is usually an accompanying symptom of the apoplectic attack but may be isolated and often combined with a hemianopsia.

Aphasia is a frequent result of the hemiplegic attack (right hemiplegia in right-handed persons; left-sided in left-handed persons).

The type of aphasia depends entirely upon the location of the lesion and the areas served. Transitory aphasias are extremely common, occurring in at least one-half of the attacks. Permanent aphasias are rarer, about one-half of the patients recovering. The

[1] Compare Wilbrand and Saenger: Die Neurologie des Auges, for complete discussion and bibliography; also Henschen, in Lewandowsky's Handbuch der Neurologie.

different forms of aphasia and the localization significance are discussed under Disorders of Speech.[1]

Papillary edema, even choked disk, may be a symptom of apoplexy, and is found on the side affected. It is most frequent with large lesions.[2]

Diagnosis.—In the matter of the middle cerebral syndromes chiefly, a differentiation of the apoplectic attack in terms of causation—hemorrhage, thrombosis or embolism—is highly desirable, although it is not always possible. The end-results may be identical, but the antecedent conditions vary. *Cerebral hemorrhage* is comparatively rare in the young, but it may be seen at all decades, and even in intrauterine life. When occurring in early youth it is usually a result of parental syphilis or of some acute illness. In cases of cerebral hemorrhage the patient is usually over forty, shows signs of arterial disease, frequently with hypertrophied heart, and often has nephritis. The attack is usually sudden, often preceded by emotional disturbance or sudden change in position that modifies the blood-pressure. The attack is more likely to be accompanied by unconsciousness (75 per cent.). Severe ventricular hemorrhages show blood on lumbar puncture, but this procedure is rarely called for save in supposed traumatic cases.

Thrombosis occurs also in older individuals, and in syphilitics particularly. Unconsciousness is less apt to occur, or develops in progressive stages as it were. The prodromata already mentioned are more apt to have been present. Markedly atheromatous arteries speak for thrombosis, and mild signs of deterioration—lacunar syndromes—speak for thrombotic types of disease.

Embolism is almost invariably associated with some acute disease—typhoid, acute septic infections (articular rheumatism, gonorrhea, scarlet fever, malaria, etc.). Acute endocarditis is often present and the individuals are apt to be young. Unconsciousness is less apt to occur; when occurring it is likely to develop very suddenly, and is more often present in basilar and carotid occlusions than when other arteries are blocked.

Hemorrhages, especially when small, frequently show the maximum symptoms early, with gradual betterment, while thromboses usually show the reverse, the symptoms having a tendency to spread or to deepen.

Ophthalmoscopic examination is always imperative. Dilatation of the pupil, usually present, can be obtained by cocaine in a few minutes. Diffuse retinitis, so-called retrobulbar neuritis, is highly indicative of basal syphilis or syphilitic endarteritis. In hemorrhage the vessels of the disk are apt to be engorged. This is less so in embolism nor in thrombosis. Choked disk points to a neoplasm.

The blood-pressure affords diagnostic criteria. It is apt to be high

[1] Comp. v. Monakow's Die Lokalization im Grosshirn, 1914, for comprehensive discussion of the complicated aphasia question. Later studies of Kleist, Golsternand Henschen.

[2] Uhthoff: Neurol. Centralbl., 1909.

in hemorrhage and in embolism, but low in thrombosis, also in neoplasms, syphilitic endarteritis and cerebral abscess.

Other disease processes to be distinguished are hysterical hemiplegias, syncope, multiple sclerosis, epileptic attacks, general paresis, uremic, alcoholic or encephalitic coma.

Hysterical hemiplegias are rarely accompanied by unconsciousness. Hysterical delirium may complicate the picture. Later the signs of pyramidal tract involvement, such as increased reflexes, Babinski, Chaddock, loss of abdominal reflexes, Grasset and Hoover phenomena are not present in hysterical hemiplegia. In certain mild thalamic cases the sensory changes may be thought of as of psychogenic origin, but careful summing up of the results of the tests of Head will show the somatic signs of thalamic involvement. Personal consultation experience has shown that many thalamic cases have been diagnosed as psychogenic.

Certain lacunar syndromes, especially in the anterior cerebral distribution, which are mild and which present momentary confusion (worse at night), emotional instability, irritability, tendency to weeping, etc., are frequently mistaken for hysteria. Suggestive treatment of such a case, as by a metronome as personally observed, is not likely to succeed.

Lacunar syndromes with softenings in the frontal areas are also mistaken for *manic* attacks. There is confusion, some disorientation, excitement, and after a short time the whole thing may clear up, leaving only slight defect, easily overlooked, unless careful intelligence tests are applied (presbyophrenic excitements, presenile excitements —see Senile and Presenile Psychoses.)

Uremic coma is usually of more gradual onset. There are preceding signs of heaviness and toxemia; convulsive movements are not infrequent. But signs of inequality of respiration of the two sides of the chest, possibly pupillary inequalities, possible variations in response to sensory stimuli on the two sides, involuntary reflex motor responses to joint squeezing and finger squeezing (Marie-Foix) are usually absent in uremic coma. The general odor of the patient is often characteristic in uremia. Some uremic patients develop apoplectic attacks as well.

Diabetic coma shows similar difficulties. Acetone odor, large amounts of sugar-containing urine, usually slow onset and the previous history must be relied upon.

Alcoholic coma is frequently diagnosed by the police as apoplexy, since many alcoholics do have apoplectic attacks. Some severe intoxications resemble apoplectic coma very closely, but, as a rule, the coma is less profound in alcoholism; the reflexes are often preserved, in a measure, and the uniformity in the bilaterality of the relaxation is evident (absence of Marie-Foix signs). One should be on one's guard, however, and look for all of the little signs of hemiplegia.[1]

[1] Dejerine: Semiologie, 1914, 2d ed. Jelliffe: Little Signs of Hemiplegia, Postgraduate, 1912. See Menninger, W.: Pupils in Coma, *loc. cit.*

Epileptic and syncopal attacks rarely present great difficulties. The history of a previous attack, the scarred body or head and tongue of the epileptic is often evident, while in syncope the coma is usually shallow and the feeble respirations and superficial heart action point to the difficulty.

An apoplectiform attack may be the first sign of *paresis*. Such attacks are usually due to cerebral edema, and may be at first indistinguishable from an apoplexy (non-paretic). The later history and examination will usually establish a diagnosis, although the pseudo-paresis of arteriosclerotic softening is often only distinguishable from paresis by the cytobiological reactions. (See chapter on Syphilitic Diseases of the Nervous System.)

Prognosis.—Recovery from the attack and amelioration of the residual symptoms are separate problems.

Cerebral hemorrhage is usually more immediately fatal than either thrombosis or embolism. Deepening coma, Cheyne-Stokes' respiration, irritative phenomena, jerking, convulsions, blood-pressure (very high, over 230 mm. or very low, under 90 mm.), marked rise in temperature are the usual lethal signs. General convulsions, retinal hemorrhage, blood on puncture, bilateral paralysis are unfavorable signs. After recovery from coma, continued temperature, advancing symptoms, restlessness, delirium, loss of sphincter control, trophic disturbances, indicate a grave prognosis and probable death in from two to three weeks.

Early attacks of lacunar softening are rarely fatal, but indicate that a fatal termination from a more severe type of attack will be probable in from one to three years. Inasmuch as this is a form which is frequently mistakenly diagnosed as hysterical in the early mild attacks, sometimes as a mild manic attack (excitement and confusion), one should be on one's guard in this not infrequent syndrome.

The recovery from the residual symptoms will vary greatly upon the nature, localization and extent of the lesion. A careful plotting of the entire symptomatology will determine the area involved in the destructive process, and the effects of diaschisis must first be separated out from those of actual tissue destruction, since the former are more apt to disappear.

Aphasias are usually recovered from, almost invariably in left-sided hemiplegias in the right-handed. In right-handed hemiplegias aphasias are recovered from in about one-half of the patients, especially in the intelligent who will make an effort to reëducate themselves. If the lesion lies directly within the aphasic area the chances for recovery are less.

Hemianopsias are not infrequently diaschitic. When so they pass within a few weeks. In thalamic lesions they are apt to be permanent, as are cortical hemianopsias persisting over a few weeks.

Hemiplegia, if total, is apt to persist in some degree at least, although most patients are able to get about in three months or more. Helplessness persisting over six months or a year has been partially recovered

from. Facial palsies usually recover. Leg palsy is rarely as persistent as arm palsy, but both react favorably to appropriate treatment. Early contracture usually means bad therapy.

Thalamic involvement is a bad prognostic feature—the pain and irregular movements (choreo-athetoid), hemianesthesia, etc., usually persist.

Mental defects may clear up almost entirely, especially when slight, but careful intelligence tests (see Chapter on Mental Examination) should be utilized in all cases, especially to decide medico-legal problems which may arise—testamentary or contract capacity, responsibility, etc. A sensory or motor aphasia alone is not necessarily a sign of an intelligence defect; many aphasics are very intelligent. They simply cannot utilize, in speech, the knowledge they have. (See Chapter on Senile and Presenile Mental States.) A motor aphasic who cannot get his symbols over, as it were, may not be any more of a dement than an Englishman trying to make a Chinaman understand what he is saying.[1]

Treatment.—Prophylaxis applies particularly to those over fifty years with arteriosclerosis, and sustained high blood-pressure. The tendency to hemorrhage here is great. Such patients should slow down somewhat in their work, if strenuous and calling for intense and sustained effort, and especially if emotional calls are frequent. A moderate amount of work and freedom is desirable. Invaliding a prospective hemorrhagic case is inadvisable. A partial psychoanalysis in intelligent patients who have not crystallized all their ideas, to show faulty handling of the unconscious emotional reactions may be highly beneficial, especially in hypertensive states.

Dietary faddism is to be avoided. Excessive eating is to be stopped. Protein-free diet—vegetable proteins are the same as animal—may keep down the blood-pressure in some cases. Alcohol is not contraindicated if used with common sense.

Careful regulation of the gastro-intestinal tract is called for, the kidney functions should be scrutinized, and the liver metabolism regulated.

It is doubtful if drug therapy is of any service in prophylaxis. The iodides have been used widely, but their utility is still undecided.

Treatment of the attack, even if there is no unconsciousness, requires immediate rest in bed if possible. If striken away from home the patient should be moved as little as possible. With high tension, hot foot-packs will help to reduce it (not hot enough to burn the unconscious patient). In cases in which the blood-pressure rises steadily, keeps above 250 mm. and with very deep coma, blood-letting (10 to 12 oz.) is advisable.

Hypodermic medication by blood-pressure reducing drugs in hemorrhage is alone advisable in coma, and only very small quantities of water should be used, as water raises blood-pressure. Hydrochlorate

[1] See interesting autobiographic account by Forel, A.: Progrés Méd., 1927, **42**, 126.

of gelsemine in doses of $\frac{1}{15}$ grain is fairly active and reliable. The nitrites are not available.

If the patient can swallow, tincture of aconite in ♏v doses may be given, watching the blood-pressure. The dose may be repeated in an hour. Pressure should be kept below 200 mm. if possible. Tincture of veratrum viride in ♏v–xv, every two hours, or the fl. ext. of gelsemium in same doses at similar intervals.

Pressure-reducing drugs should be used with caution. The high pressure following hemorrhage is usually compensatory and for the purpose of keeping up an effective vascular irrigation of the medullary nuclei following a rupture in the arterial pipe-line. These drugs should be used only when it is known that a high pressure preceded the attack.

Early purgation is desirable; 2 gtt. of croton oil is useful in states of deep coma. This may be placed with butter on the back of the tongue.

If there is marked excitement, chloral, gr. v–xv, or paraldehyde, ʒj–ij, may be given by mouth or by rectum. Tepid sponge baths help restlessness. Veronal and trional are useful in the restlessness of lacunar softenings.

Care of the mouth, drooling, moving the patient, removal of all obstructions to breathing, should be attended to at once.

Nourishment may be omitted in the early stages. A purin-free diet, mostly milk and gruels, to which sugar and eggs may be added later, should constitute the standard diet. In the presence of difficulty in swallowing, milk enemas are to be given.

In cardiac atonic cases, with low blood-pressure—chiefly thromboses—vasodilators, camphor and adrenalin may be necessary.

Surgical treatment may be advisable for meningeal or subcortical hemorrhages. Certain nephritic cases are helped by lumbar puncture, and the withdrawal of 20 to 50 cc. of fluid. The patients should be kept quiet several weeks, attention being paid to giving ease and comfort by cushions, props, supports, and frequent changes of position.

Late Treatment of Hemiplegia.—The recent investigations on the functions of the pyramidal tracts (von Monakow, Sherrington, Franz) make the outlook for the reëducational treatment of hemiplegia much more hopeful. It would seem, as a result of this work, that the pyramidal tracts are not the exclusive carriers of volitional motor control. Destruction of the motor cortex of the cat, dog or monkey causes only about two-thirds of the pyramidal tract to degenerate. The indications are that there is a large extrarolandic motor area, probably in the frontal and parietal lobes, particularly for the face and upper extremities. In addition to these facts are the further ones brought out by stimulation of the motor cortex, viz., the so-called motor centers do not correspond in extent in different animals of the same species; they are not of the same extent on the two sides in the same animal; repeated stimulation does not always produce the same movements, sometimes there are added movements and sometimes the movements are the exact opposite as extension where there was formerly flexion.

All this indicates that the motor cortex is by no means as fixed in the relation of its cells to a definite muscular function as has heretofore been supposed. On the contrary, it would seem that it had retained a considerable plasticity, a large capacity for adjustment so that the movement resulting from stimulation of any particular part was dependent upon the functional set of the associated neurons at the moment of stimulation. If this is true it can be seen how systematic motor reëducation may serve to facilitate discharge through paths not heretofore consciously used both by bringing into use associated cortical motor cells and pathways nearby and also by drawing upon the extrarolandic regions.

The application of the principles elucidated have in fact been productive of most excellent results in the treatment of hemiplegias. A most detailed scheme of movements calculated to overcome the motor defects should be mapped out and persisted in daily. This plan should be begun a few days after the injury, practically as soon as the patient's mental state will permit of coöperation. It should, at first, be limited to a few minutes and later prolonged with care to avoid excessive fatigue. The nurse can finally learn what is to be done and exercise the patient perhaps an hour daily, adding diversions to the daily routine that include a utilization of the newly acquired motions.

Such exercises must get away from the hackneyed and stupid calisthenics, etc., which have prevailed in the past. The psyche of the patient must be put into his work and the exercises worked out along proper kinetic lines (Mensendieck exercises and the like) employed.

In certain cerebral hemorrhage diplegias the contractures may be helped by the Foerster operation or some modification of it. (See Little's disease.)

Electricity is of doubtful service. Galvanism aids some of the pains, the anode should be over the painful area.

The general care of the invalid will depend largely upon his economic status. Travel is helpful in supplying a mental stimulus and the warmer climates and more interesting foreign resorts are enjoyable, and thus of direct therapeutic value. The proper exercises must be kept up, however. Travel alone has no particular benefit. Varied occupations suited to the individual's temperament and habits should be sought for, wherein resourcefulness is a great asset.

Speech training for aphasia may accomplish much. Its details, as well as many others, cannot be entered into here.[1]

SINUS THROMBOSIS.

There are three main varieties of sinus thrombosis, the so-called marasmic variety, occurring in debilitated individuals, and as a result of cardiac weakness, cachexia, chlorosis, etc. Thrombosis of a sinus

[1] Consult White and Jelliffe: Modern Treatment of Nervous and Mental Disease, vol. **2**, for detailed discussion of all of the features of treatment in chapters by F. Tilney and Kinnier Wilson.

may also be dependent on injury as a result of fracture of the skull or injury to the skull during difficult labor. The most important form of sinus thrombosis, however, is that dependent upon infection, and the most frequent varieties are sigmoid thrombosis, from mastoid

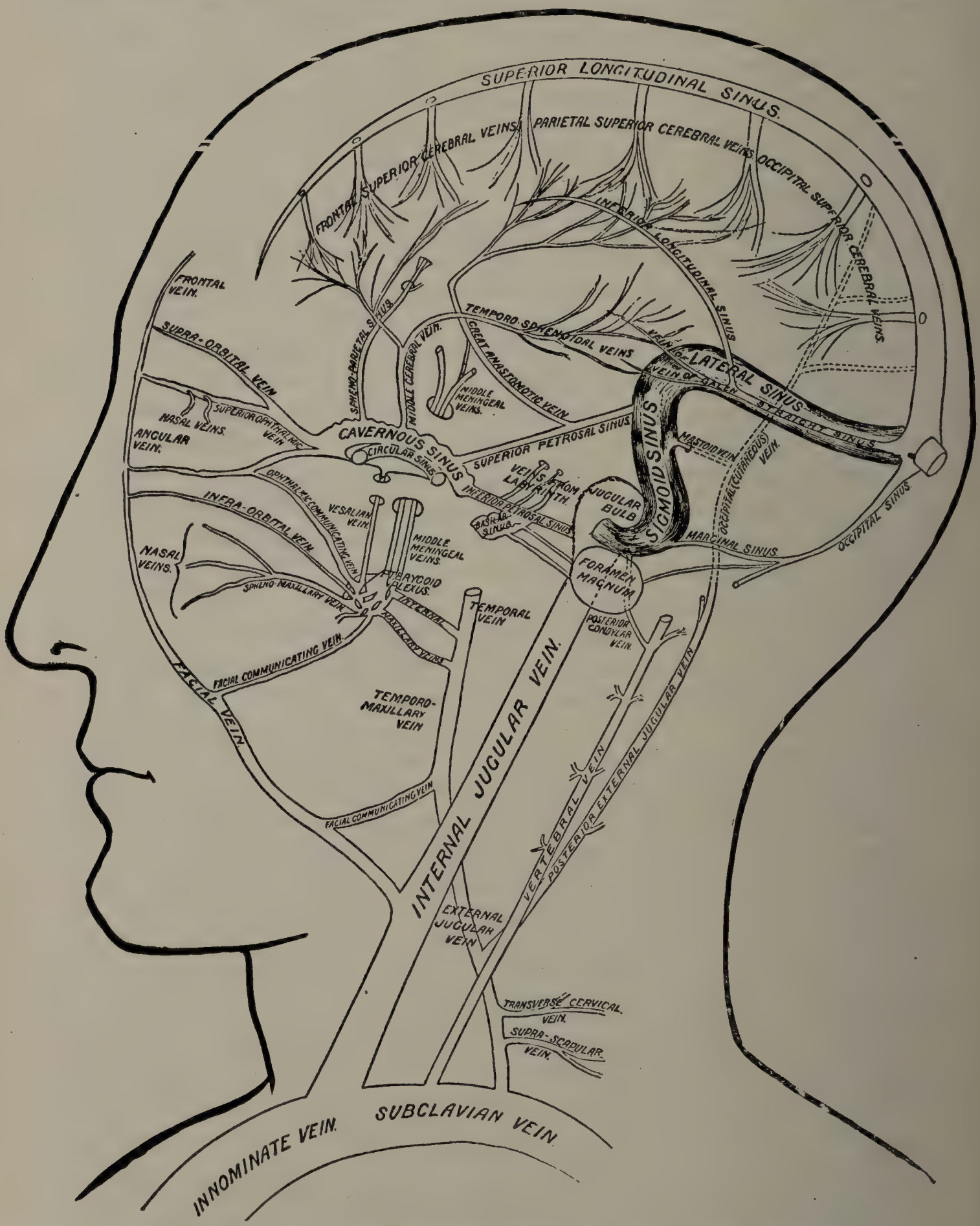


FIG. 371.—Schema showing venous sinuses of the head. (After Macewen.)

disease, and lateral thrombosis. Primary thrombosis of the jugular bulb sometimes follows middle ear infections.[1]

[1] Braun, A.: Diagnosis of Sinus Thrombosis, New York Med. Jour., December 21, 1918.

Symptoms.—The general symptoms of sinus thrombosis are, in accordance with the above, those of infection, namely, chills and fever, and with the breaking down and liquefication of the thrombus and its distribution in the general circulation there may be pyemia, with abscess formation. These abscesses may occur in the brain, cerebrum and cerebellum, or the infection may extend directly from the sinus and produce a local or a general meningitis.

The local symptoms vary with the sinus involved. In thrombosis of the *lateral sinus*, dependent upon middle-ear disease, there is usually a venous congestion and edema over the mastoid process, and the thrombus may extend into the jugular vein and be palpable.

The chief symptoms in lateral sinus disease are: (1) Pain, at times merged in the mastoid pain. In certain cases a marked euphoria, from temperature, masks the pain and may even be diagnostic of a thrombosis. (2) Optic hyperemia is not infrequent. (3) Sixth nerve paralysis occasionally. (4) Perijugulitis, with pain on moving the head or in swallowing. (5) Glossopharyngeal implication with loss of power in swallowing from palatal paresis. (6) Vagus implication rarely as seen in hoarseness and bradycardia. (7) Spinal accessory irritation or paralysis. These are the chief results of the inflammatory process in the sinus wall. The chief signs of interference with the venous circulation are: (1) Griesinger's sign, painful swelling at the middle of the posterior margin of the mastoid; (2) Painful swelling in the posterior triangle of the neck; (3) Gerhardt's sign, an unequal fulness of the jugular veins, not very constant.

Cavernous sinus thrombosis, owing to the drainage of the ophthalmic vein backward into this sinus, produces quite characteristically marked exophthalmos, venous congestion and edema of the lids, chemosis; these are often mistaken for orbital disease and are fairly constant. To these may be added choked disk, infrequently, together with retinal hemorrhages, oculomotor palsies, dilated immovable pupils, involvement of the fifth nerve, with hyperesthesia in its area of distribution. Involvement of this sinus rarely remains unilateral but soon becomes bilateral by spread of the affection by way of the circular sinus.

In *longitudinal* sinus thrombosis, distended fontanelles (in children), nosebleed, caput medusæ, and swelling of the frontal and parietal veins are the chief local symptoms.

Treatment.—The treatment of these conditions is essentially surgical. Septic involvement of the cavernous sinus is generally fatal. The condition is usually inaccessible to surgical interference.

Thrombosis of the lateral sinus is not an infrequent complication of the middle-ear disease, and if seen early, is susceptible of successful operation. The sinus may be opened and the clot cleared out.

The other sinuses of the dura may of course become involved, but generally secondarily, and do not present such characteristic clinical pictures.[1]

[1] Eagleton: Sinus Thrombosis, 1926.

CHAPTER XIII.

TUMORS OF THE BRAIN.

Tumors of the brain are relatively infrequent. They occur at all ages, are found in every conceivable location within the cranial cavity, and are of a greatly varied pathology. Extensive monographs have been published, and no feature of disease of the nervous system has attracted more attention, not only by reason of its practical importance, but also because of the contribution to cerebral localization and function. The chief literature to 1919 may be found in Starr,[1] von Monakow,[2] Oppenheim,[3] Tooth[4] and Redlich.[5] Later studies by Christiansen, (1924), Cushing[6] (1922), Cushing and Bailey (1925) Puusepp (1927), Purves-Stewart (1927) should be consulted and in the French *Traité de medecine*, Roger, Widal and Teissier *et al.*

In 18,000 cases of nervous disease occurring in ten years at the Vanderbilt Clinic there were 48 brain tumors. This is a minimum computation.[7] Bruns states it as high as 2 per cent., Cushing as 0.75 per cent., Redlich, from 4000 cases of nervous disease, the same.

Etiology.—The causes for certain tumors, such as tuberculoma, syphiloma, actinomycoses, are well known. For both syphilitic and tuberculous tumors, and possibly other tumors, traumatism may be an additional element for their special localization. Metastatic tumors follow from their primary sources. Certain teratomata, dermoids, angiomata are congenital conditions, while cholesteatomata, chordoma, chondroma, lipoma and myxoma are also developmental anomalies.

The traumatic genesis of tumors in general, or of any one type in particular, apart from aneurisms, is highly problematical, yet if the trauma and symptom development are related in specific and very definite ways the causative role may be debated. Thus, if the injury is sufficiently intense to definitely injure the skull, the time interval between the accident and the development of the symptoms not too great, and the localization of the probable tumor near to the site of the injury, the relationship may justifiably be open to serious consideration.

[1] Text-book of Nervous Diseases, 4th ed., Brain Surgery.
[2] Gehirnpathologie, 2d ed., Die Lokalisation im Grosshirn, 1914.
[3] Die Geschwülste des Gehirns, 2d ed. [4] Brain, 1912, **35**, 61.
[5] Handbuch der Neurologie, Lewandowsky, 1912, vol. iii. Also consult Current Literature in Journal of Nervous and Mental Disease.
[6] Cushing: Intracranial Tumors of Preadolescence, Am. Jour. Dis. Child., 1927, **33**, 551.
[7] See Report of Clinic of Prof. M. Allen Starr, 1900–1909, by Jelliffe and Brill. Christiansen: Les Tumeur du Cerveau, Paris, 1921. See Förtig, H.: Ueber Hirntumoren, Monat. f. Neur. u. Psych., 1921, **49**, 89.

Varieties.—The chief forms met with may be classified as: (1) True tumors, (2) infectious tumors, (3) parasitic cysts, (4) aneurisms, (5) vascular cysts.

1. **True Tumors.**—Of these *gliomata* are the most frequent. They preponderate over any other class in adults. In Tooth's summary of 500 cases, 49 per cent. were gliomata. Children rarely come to the National Hospital in London, hence Tooth's low percentage of tuberculous tumors. Gliomata occur throughout the brain as circumscribed or as diffuse tumors, and are extra- as well as intracerebral. The general tendency is toward diffuseness, and degeneration with hemorrhage and fatty and cystic formations take place principally in intracerebral growths. Thus the symptoms are apt to develop slowly and intermittently in cortex or basal ganglia tumors, occasionally from those of the ependyma. The tumors may invade an entire hemisphere.



FIG. 372.—Infiltrating glioma of basal ganglia.

Sarcomata are less frequent, restricting the term sarcoma more strictly than is usual, and rejecting the compromise gliosarcoma. Nearly all gliomata show elements indistinguishable from sarcoma. They usually develop slowly in the brain.

Chloromata are leukemic in origin and are rare. They involve the periosteum or the base and thus cause compression phenomena which often persist for some time.

Fibromata (neurofibromata) are comparatively common brain tumors (10 per cent. in Tooth's collection, including fibroglioma). They develop chiefly about the cerebellopontine angle (acoustic) but may develop along other cranial nerves. Occasionally they are multiple. They develop slowly.[1]

[1] Cushing: Tumors of the Nervus Acusticus, 1918.

Endotheliomata are comparatively frequent (14 per cent. in Tooth's collection). They seem to confine themselves chiefly to the anterior fossæ. They are usually small and multiple, develop slowly and chiefly in the falx region.[1]

Chordomata are infrequent, and only rarely reach a considerable size.[2]

Carcinomata (5.8 per cent.—Tooth), closely related to the endotheliomata are usually secondary (metastatic), rarely primary.[3]

Psammomata,[4] lipomata, enchondromata, angiomata, osteomata, adenomata, cysts,[5] cholesteomata, hypophysial tumors, teratomata (pineal),[6] and dermoids are among the rarities.

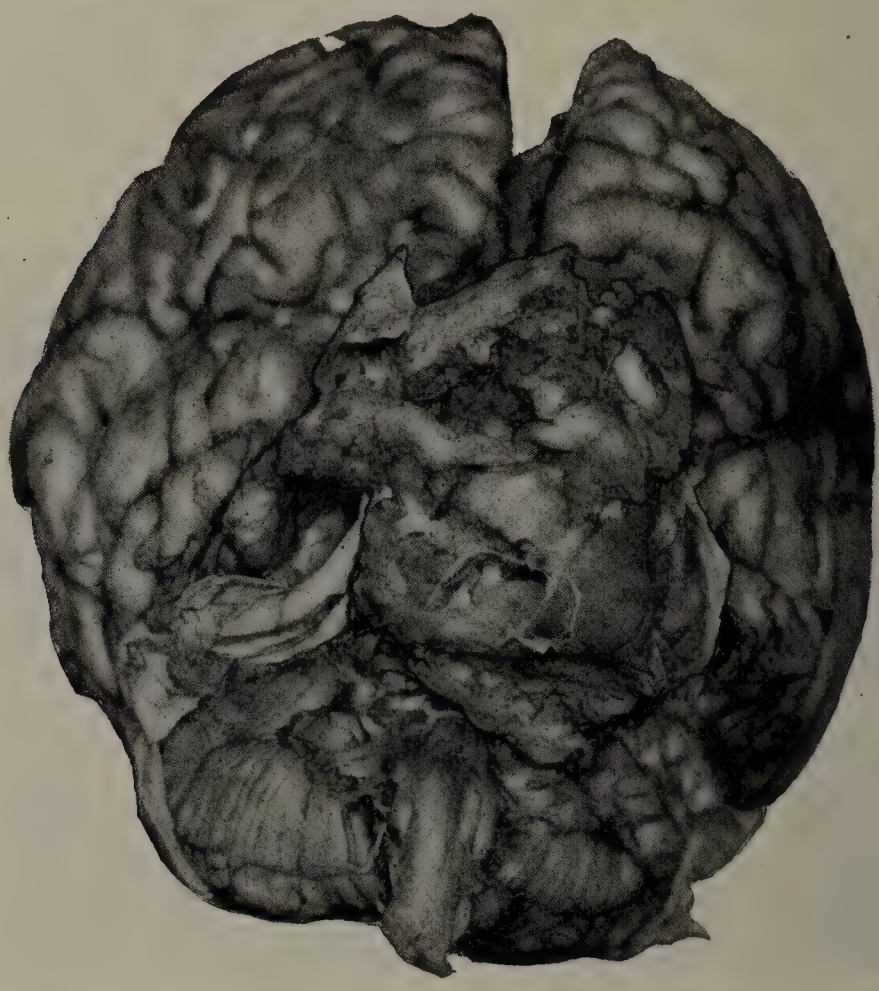


FIG. 373.—Chordoma of base. (Jelliffe and Larkin.)

2. **Infectious Tumors.**—These are tuberculous, syphilitic, actinomycotic.

Tuberculomata are possibly the commonest of all tumors, certainly in children (Starr, 193 in 600 tumors recorded). They are extremely rare after forty years. They are frequently conglomerate or multiple in type, hence giving rise to mixed syndromes. There may be a few very small miliary tubercles or a large broken-down tubercle mass, with every conceivable intermediary stage. They develop chiefly in the cerebellum, peduncle, basal ganglia, pons and cortex. In Zappert's

[1] Haeger: Berlin, 1911.
[2] Jelliffe and Larkin: Jour. Nerv. and Ment. Dis., January, 1912.
[3] Levin: Metastasis of Cancer in the Central Nervous System, Jour. Nerv. and Ment. Dis., June, 1917.
[4] Roussy: Arch. gén. de Méd., ii, 82.
[5] Meyer: Berlin, 1913.
[6] Bailey and Jelliffe: Tumors of the Pineal Body, Arch. Int. Med., December, 1912. Pineal Syndromes. Crowell's Loose Leaf Medicine, 1928, for tumors 1921–1927.

group of 89, 37 were in the cerebellum, 29 in the cerebral cortex, 13 in the basal ganglia, 5 in the pons. They have a bad prognosis.

Syphilomata.—Gummata are not infrequent.[1] They are practically limited to adults and are rarely recorded from congenital syphilis. Patients with congenital syphilis may develop symptoms of tumor pressure. They may appear from one year to thirty years after infection. They occur chiefly as flat, infiltrating, irregular masses—less often as definite nodular masses, chiefly at the base of the brain. They are discussed more fully in the chapter on Syphilis of the Nervous System (*q. v.*).

Actinomycosis of the brain is a rarity.

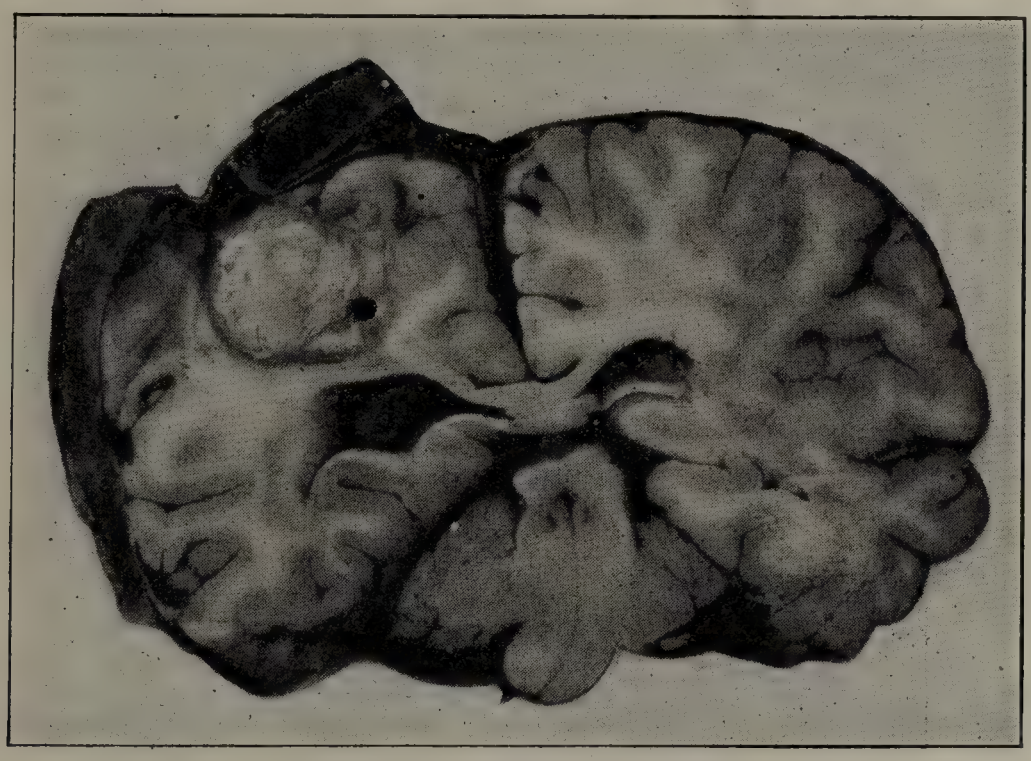


FIG. 374.—Gumma of brain.

3. **Parasitic Cystic Tumors.**—*Cysticercus of the brain* is a rarity and is secondary.[2]

4. **Aneurismal Tumors.**—Aneurisms are very frequent in cerebral vessels. They are mostly small, but large aneurisms occur at times and give symptoms of pressure. They occur in patients usually from forty to seventy years old, and are mostly of the basilar. They cause pressure symptoms at times, with obstructive symptoms—basilar syndrome—or they rupture and produce symptoms of cerebral hemorrhage.[3]

Symptoms.—Brain tumors, even of a large size, may be found at autopsy, and yet not have given rise to any recognized symptoms. Tuberculomata are thus frequently found in children. With more precise investigation such latent tumors are becoming rarer, especially

[1] Tooth: Gumma Cerebri, Tr. Royal Soc., April 17, 1913.

[2] Lewy: Beitrag zur Kenntnis der zerebrospinal Zystizerkose, Breslau, 1913. Lloyd: Philadelphia Med. Jour., March 19, 1898.

[3] Beadles: Brain, 1907, p. 285. Reinhardt: Ueber Hirnarterienaneurysmen und ihre Folgen, Mitt. a. d. Grenzg., d. Med. u. Ch., 1913, vol. **26.** Booth: Jour. Nerv. and Ment. Dis., September, 1909. Also consult Locke: Arch. of Surg., 1921, **3,** 560, for a review of 255 tumors seen in 1920–1921 in Cushing's clinic. Ann. Clin. Med., 1927, **5,** 1097. Brouwer: Ned. Tid. f. Gen., 1927, I, 619.

since the importance of mental symptoms—psychoses, so-called hysterias, etc.—unaccompanied by sensorimotor syndromes, is becoming recognized. Many small tumors, especially osteomata, psammomata, slowly developing and circumscribed gliomata, cholesteatomata may cause very few symptoms. Occasionally a tumor will show monosymptomatically, as by epileptic convulsions, mild speech disturbances, mild sensory defects, optic, olfactory, auditory hallucinations or hemianopsia, without being recognized.

The symptoms are best considered as (1) general and (2) local or focal.

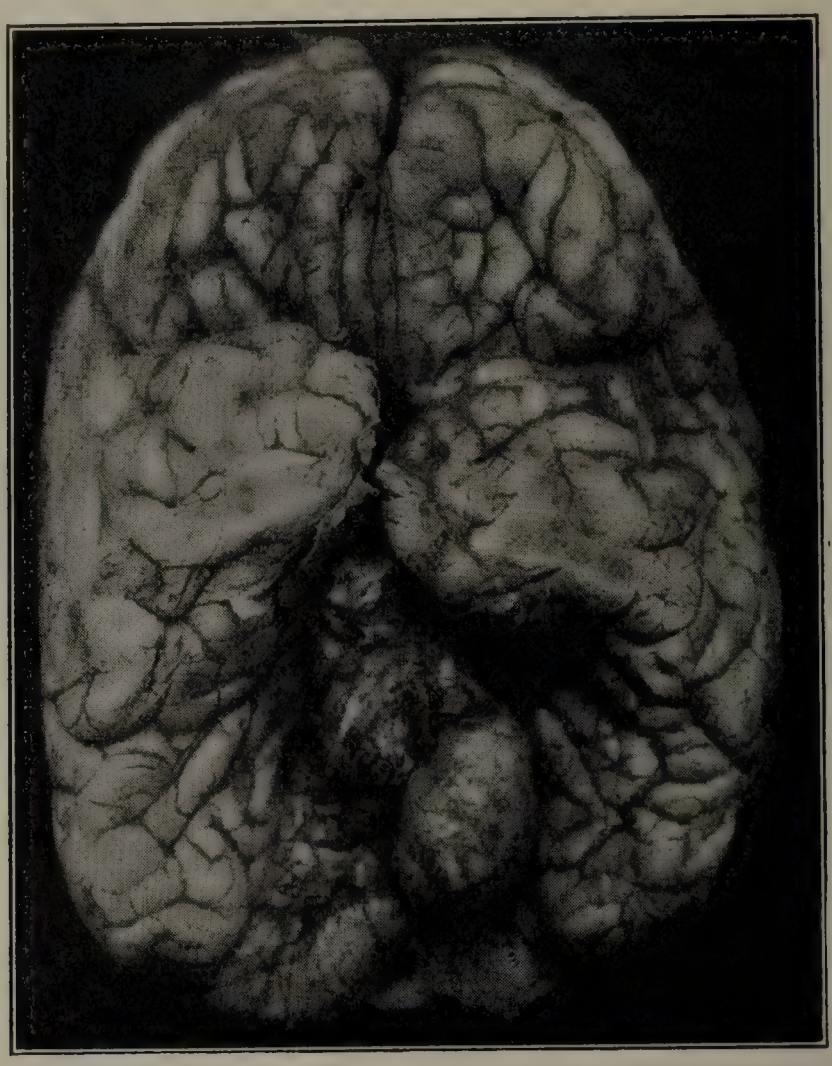


FIG. 375.—Aneurism of basilar artery. (Larkin, Jelliffe.)

The *general symptoms* are indicative of the effects of the tumor as a whole, irrespective of its special nature or localization. They are due in general to the effects of increased intracranial pressure, which in certain tumors, notably of the posterior fossa and cerebellum, appears early and is usually marked even with small tumors, whereas tumors elsewhere often may show little of such pressure symptoms. Sometimes the focal symptoms appear before the general ones. General symptoms rarely have any localizing diagnostic value. Some of the general symptoms of pressure, cranial nerve palsies for example, may even tend to mislead one as to a localizing diagnosis. Furthermore, in cerebral tumors, acute swellings, not identical with but related to edemas, often give rise to very anomalous symptoms and tend to obscure the diagnosis and render it uncertain.[1]

[1] Spiller: Jour. Nerv. and Ment. Dis., 1914, **41**, 184. Müller, E.: D. Z. f. N., **47**, 48. Marburg: Wien. med. Wchnschr., 1911, No. 40.

The chief symptoms of general value are headache, nausea, vomiting, dizziness, respiratory and cardiac disturbances, metabolic changes, mental signs of sleeplessness, sometimes drowsiness, optic nerve changes, and convulsive phenomena. These general symptoms have a tendency to be progressive, but may vary considerably in their intensity from time to time, especially in syphilomata, tuberculomata, and gliomata. At times they remain stationary, again they may regress and disappear.

Headache.—This is frequent. Most patients will have headache, especially if the tumor is of protracted growth. Headache is an early sign. Practically every patient with a headache should be questioned and examined for the possibility of its being caused by a cerebral tumor. The headache usually starts more or less irregularly, is frequently intermittent in the early stages and then becomes persistent, being dull or severe; if dull, with periods of excruciating exacerbation. Diurnal variations may be noted, and emotional excitement or general causes for increasing intracranial pressure increase it, often with additional symptoms, such as vertigo, vomiting or even agitated or comatose confusions. Aneurismal tumors may reveal pulsation by auscultation. Migraine-like headaches are very frequent in early states, particularly in basal cases, pituitary, basal gummata, neurofibromata, or there may be migraine-like exacerbations on a dull, heavy, gray background of pain. Children usually respond to such variations by attacks of screaming, pulling the hair, or beating the head.

Later, somewhat mentally dulled patients may even deny any headache, or even forget having had a period of great distress.

The headache of cerebral tumor is mostly diffuse, but it may be localized, in which case it may serve to indicate the general site of the tumor. Such is rendered more probable if further substantiated by percussion tenderness, a highly important procedure, percussion dulness, and *x*-ray shadow. The site of a headache is a very uncertain guide for localization purposes, however. Frontal tumors often give rise to occipital headaches and *vice versa;* right-sided tumors to left-sided pains and *vice versa.* The *general* drift for localized pain is, however, in favor of a similarly located tumor. Among head symptoms may be mentioned the occasionally found auscultation notes of aneurismal tumors; the presence of enlarged head (hydrocephalus), and the overfilling and increased tortuosity of the veins on the forehead, face, conjunctiva, etc., of the affected side.

Trigeminal neuralgic attacks may be general or at times a focal (cerebellopontine angle) sign.

Nausea, vomiting, and dizziness are frequent in late stages of a cerebral tumor, and more often found in children and in those patients with rapidly increasing signs of intracranial pressure—posterior fossa tumors particularly. Such vomiting may occur spontaneously—projectile in type—or as an accompaniment of the headache crises, especially when migrainous in type. Vomiting often is absent entirely even with large tumors. When present it more often occurs in the

morning, and at times it is so persistent as to lead to inanition, exhaustion, and death.

In cerebellar, peduncle, pontine, and medullary tumors, and those causing pressure on the superior cerebellar peduncle, or its incoming pathways, the nausea and vomiting may be accompanied by unilateral vertigoes or with tendencies to turn or to fall in one direction. Here general and focal symptoms coincide. Frontal tumors may occasionally cause such one-sided vertigoes from implication of the frontal extensions of the cerebellar pathways. Vertigoes from ocular palsies are localizing symptoms (quadrigeminal syndromes—occasionally cortical).

Cardiac and Respiratory Signs.—Slowness of the pulse, at times marked—30 to 40—is a general sign of intracranial pressure, and more especially in marked grades. Hence it is apt to be a late rather than an early symptom, unless one of direct irritation of the vagus (medullary pressure). The bradycardia, at times arrhythmia, may appear periodically during headache exacerbations, or at times independent of the same (acute swelling reaction).

Medullary tumors cause respiratory changes, at times slowness, again irregularity, and Cheyne-Stokes—with acute pressure symptoms (hydrocephalus internus). Hiccough, yawning, and related respiratory signs are occasionally present.

Metabolic Disturbances.—These are irregular in their development and evolution. Fever is infrequent save as a complication of the late stages.

Cachexia and marasmus are present with certain carcinomata. Marked adiposity, ovarian and in particular testicular aplasias, are frequent in certain hypophysial (pituitary) tumors or those causing internal hydrocephalus by possible implication of the infundibular region through general pressure (pineal and corpora quadrigemina tumors). Acromegaly is a special case, as are also the dystrophia adiposogenitalis syndromes which are discussed in the chapter on the Disorders of the Endocrinous Glands.[1]

Mental Signs.—These are of great value, both general and localizing, in from 60 to 85 per cent. of the cases.[2] They vary considerably, and are particularly prominent late in the disease, although here masked under the general symptoms of apathy, confusion or coma. Tumors of any region, large and small, and independently of their pathological nature, may cause psychical changes. Certain localities cause special psychical alterations to be discussed under focal and localizing symptoms.

In the early stages, slight impairment of attention, with slowness and difficulty in grasp, retardation in motor response, and a confusion

[1] Nobécourt et Lebée: Bull. Soc. de Ped. de Paris, 1927, **25**, 70.

[2] Gianelli, Schuster and others: See Bibliography in Redlich, Hirntumor, Lewandowsky's Handbuch der Neurologie. See Förtig for a penetrating discussion of the psychical symptoms of brain tumors, Monat. f. Neur. u. Psych., 1921, **49**, 89. Barûk: L'Encéphale, 1926, **21**, 760; Gordon: Rev. Neur., 1927, i, 599; Souques et Baruk: Rev. Neur., 1927, I, 87.

or bewilderment may be present. Ready forgetting, slight esthetic lapses, and moral breaks—with the telling of shady stories, showing of bad taste, exhibitionistic fancies, even gross lapses—such as open masturbation, etc.—occur. These are the precursors of a more marked grade of retardation of mental function, leading to apathy, listlessness, lack of initiative, at times with confusional episodes—getting lost—fugues, fussiness, emotionalism, etc. In marked states of confusion and disorientation a typical Korsakoff's syndrome (*q. v.*), without polyneuritis, may be present.

Later stages show typical pictures, not distinguishable from those of arteriosclerotic deterioration, or paresis—*i. e.*, so far as the purely mental picture is concerned.

Certain less general symptoms often present themselves and are of a certain localizing value. Hallucinations of smell speak for olfactory lobe or olfactory tract involvement. Those of sight for occipital cortex localization, those of sound for temporal lobe trouble.

The tendency to joke, be facetious, show manic idea associations, even flight of ideas with euphoria, is infrequently present. This is more often found in prefrontal tumors, especially left-sided, but may occur in tumors of other regions, usually, however, all reaching to and involving the cortex. These symptoms are possibly thalamic over-responses from thalamo-cortical interference at cortical levels.

Certain patients show definite depressed states—with hypochondriasis or even melancholic suicidal ideas. Others show manic pictures with wild flight or marked maniacal delirium. Certain patients develop delirium during certain of their headache paroxysms. Paranoid trends also manifest themselves in a few instances. So-called hysterical symptoms are frequently encountered. Careful analysis shows, however, no psychical conversions. Emotionalism and delirium are incorrectly termed hysterical because of a loose application of the term hysteria. The type of response at the symbolic level is probably conditioned by a relation between the psychical and the physical in which it seems not impossible that unconscious factors may bring about organic variations which in turn reinforce the psychical components. Patients develop manic, depressed, paranoid tendencies or what not, dependent upon their characteristic personality trends as is being shown in a number of organic conditions.[1]

Optic Nerve Changes.—These are of the greatest importance in diagnosis of brain tumor. The general features are discussed in the chapter on Cranial Nerves. From 60 to 80 per cent. of all patients show optic nerve changes, which vary largely, depending upon the grade of intracranial pressure and the size and location of the tumor. Those tumors causing intracranial pressure (posterior fossa particularly) force the cerebrospinal fluid into the space between the optic nerve and its vaginal sheath. This causes a papilledema, or choked disk, with little change in vision, and may be later if organization of the fluid takes

[1] Osnato: Jour. Nerv. and Ment. Dis., May, June, 1919, vol. **50,** Kempf, loc. cit. Lewis: Megalocolon, Jour. Nerv. and Ment. Dis., October, 1922.

place, a true neuritis. Optic nerve changes may be absent even with large tumors, and small tumors of the pons, medulla, motor area, basal ganglia, corpus callosum, and pituitary may give rise to no optic nerve changes. The optic nerve changes develop gradually. They are unilateral, later bilateral or develop bilaterally synchronously. As a rule tumors of one side show beginning nerve changes on the same side earlier and more markedly than on the opposite side. The reverse can also be true and the localizing value must not be overestimated. The general trend, however, is as stated (Gunn).

The visual power may not be lost even with marked grade of swelling but there is later a gradual loss of vision—often seen in early signs by the irregularity of the color field loss (interlacing phenomena, scotomata, etc.). The hemianopic changes which are not infrequent in chiasm, posterior tract, pulvinar, and posterior parts of the optic pathways are discussed in Chapter V.[1]

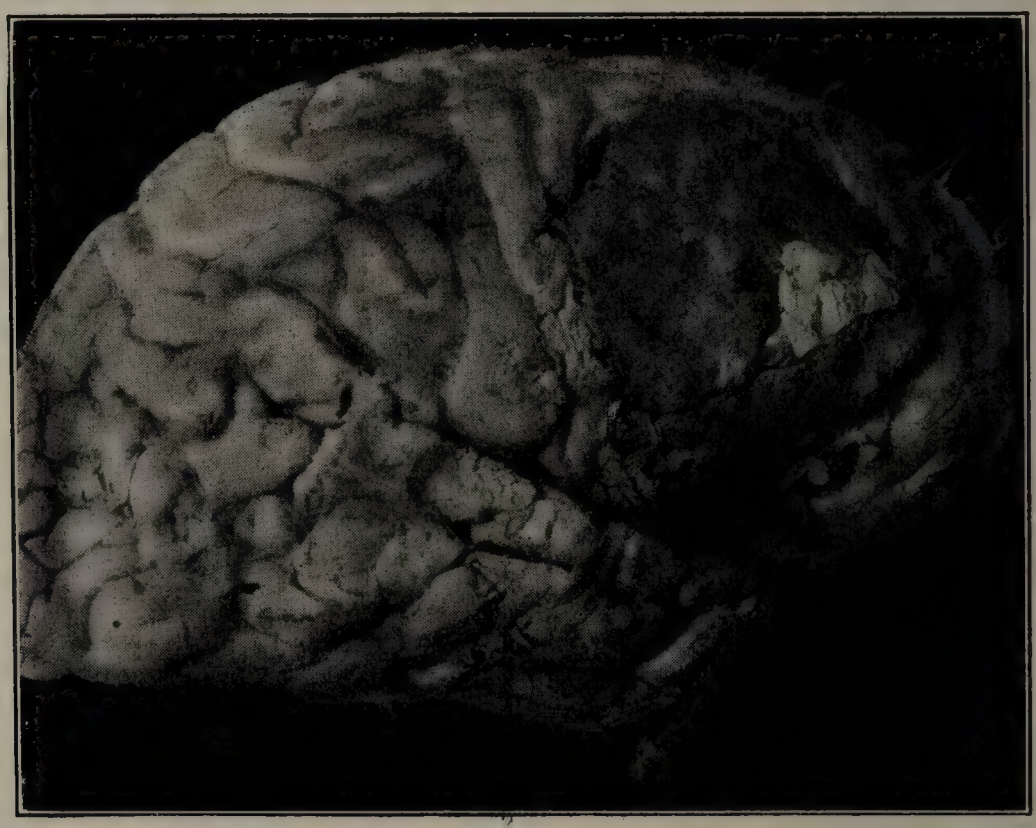


FIG. 376.—Frontal lobe tumor. (Larkin, Jelliffe.)

Motor Phenomena.—These are local or general. Epileptiform attacks are frequent—particularly in children. When limited Jacksonian attacks are present, the localizing value in the motor cortex is evident, save for the few exceptions of pontine, cerebellar, and peduncular Jacksonian attacks. Petit mal attacks are also not infrequent. Some patients die in the convulsive seizures.

Focal or Local Symptoms.—These may be among the first of the symptoms encountered, especially in circumscribed cortical tumors, but, as a rule, the focal symptoms develop after the general ones; again they advance together. They are best considered with reference to

[1] See study on hemianopia in brain tumors by Bourguet: Ann. de Med. de l'Oreille, 1927, **46**, 1.

the areas involved, such as the symptoms of the frontal localizations, central convolutions, parietal lobes, temporal etc.

Frontal Lobe Tumors are those located ahead of the precentral sulci. Those of the frontal poles, foot of third frontals, are frequently termed prefrontal tumors.[1] The functions of the frontal areas are chiefly those of coördination of psychical, chiefly intellectual processes.[2] They contain also motor areas for the innervation of the muscles of the neck, throat and abdominal muscles and the third frontal convolution—Brocas' convolution—is a motor aphasia area. The olfactory and optic pathways may be involved by direct or indirect injury by tumors of the base of the brain, and pressure posteriorly causes pyramidal tract symptoms. Cerebellar pathway projections also lie in the frontal poles.

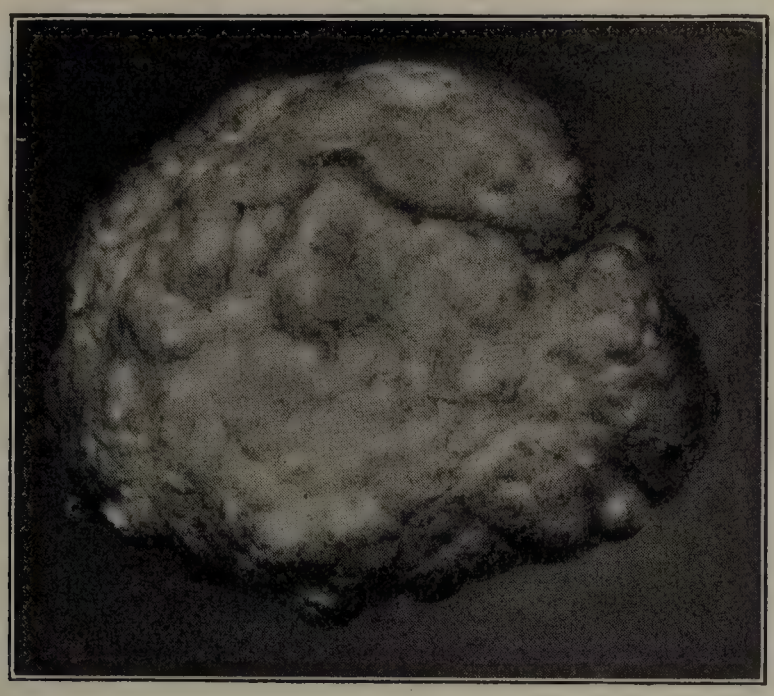


FIG. 377.—Frontal lobe tumor removed. This patient had loss of smell on the tumor side. Witzelsucht, staggering gait, and loss of control of feces at times, with otherwise unimpaired intelligence. Natural size. (Jelliffe)

Thus the symptoms of tumors lying within the frontal lobes may show considerable variation according to their site and their size, many of the symptoms being due not to the local involvement, but to pressure upon surrounding structures (neighborhood symptoms). Many small tumors located deep in the cortex are apparently symptomless from the neurological point of view, but careful psychiatric observation may develop certain very definite symptoms. Paranoid states, occasionally resulting from such tumors, are readily overlooked, also mild depressed states which are called neurasthenia.[3] A symptom of much interest, even if not common, is a tendency to make jokes

[1] For early literature see Grimm: Tumoren an den vorderen Hirnlappen, Greifswald Diss., 1868. Newmark: Prefrontal Tumors, California State Jour. Med., November, 1913.

[2] Franz: Functions of the Frontal Lobes, Archives of Psychology, 1907. Bianchi: The Mechanism of the Brain and the Function of the Frontal Lobes, 1922. Souques et Baruk, *loc. cit.* Pisani: Rev. oto-neur. oftal., 1926, **30**, 289. Nonne: Med. Klin., 1927, **23**, 1.

[3] Dynan: New York Med. Jour., November 4, 1922.

(Witzelsucht), or a tendency to talk or answer beside the point—at times an apparently intentional effort to mislead. One does not necessarily locate a tumor in the frontal lobes by reason of this newly developed tendency to joking alone. At times the behavior is infantile and churlish, and is diagnosed hysteria. Again patients are irritable, excitable, childish, even have furious outbreaks of wrath and are violent, capricious, or the picture of gradually advancing stupidity, with inability to grasp, loss of initiative, slowness of power of application, is seen. Schilder[1] emphasizes this loss of interest as an initial frontal sign. In the war wounds of the frontal lobes reported on by Schob,[2] Müller[3] and Bostrom[4] this is confirmed. This akinesis from frontal impairment is to be carefully separated from a similar akinesis from striatum lesions as seen in the Parkinsonian syndromes of encephalitis in that conscious initiation seems more involved. One would expect to find, particularly from the researches of Franz, (loc. cit.) that destruction or involvement of the left prefrontal lobes, expressors in right hand would produce more mental disturbances. The older researches of Phelps point in this same direction. There are not wanting observations apparently to the contrary, however, Schuster,[5] Pfeiffer,[6] Müller.[7] [8]

The experimental researches of Franz and of Bianchi[9] upon the frontal lobes have developed a group of symptoms in dogs, foxes and monkeys, which careful observation in man are confirming more and more. The mental changes emphasized by Bianchi which should be looked for in suspected frontal lobe involvement may be briefly summarized as: (*a*) Defect in the perception of objects in the external world. Recognition of objects is impaired and particularly some loss of relationship between objects. Primitive analogies, such as of color, similar forms, etc., are sufficient to bring about erroneous conclusions as to identity. This perceptive impairment is closely related to the loss of interest just mentioned as having been frequently observed in war neuropsychiatry. (*b*) The memory is apt to be unreliable. It becomes impaired for both old and new impressions This often brings about a repetition or perseveration type of conduct. As Bianchi expresses it, with relation to the memory of harmful activities, the animal is impaired in his biophylactic capacities. (*c*) Associative capacity, as shown in judgment, is impaired. Animals often develop a "dignified indifference" as a protective device against their mistakes. So does the frontally involved human being; (*d*) emotional variations are introduced. The simpler desires, as for food and eroticism, are not interfered with, but the more complex symbolic substitutes are

[1] Dimitz and Schilder: Mediz. Klinik., February 26, 1922; also see Souques et Baruk, *loc. cit.* Bravetta: Boll. Soc. Med. di Pavia, 1926, **1**, 1217 (Paretic syndrome).
[2] Allg. Zeit. f. Psych., 1912.
[3] Arch. f. Psychiatrie, 1921, **64.**
[4] Deutsch. Ztschr. f. Nervenh., 1921.
[5] Thèsis, Stuttgart, 1902.
[6] Arch. f. Psychiat., vol. **47.**
[7] Deutsch. Ztschr. f. Nervenh., vols. **21, 22, 23.**
[8] Blackburn: Intracranial Tumors among the Insane.
[9] See Bianchi, Function of Frontal Lobes, 1922 for complete discussion (Ceni).

swept away. Sublimation therefore is interfered with. In man the tendency to indecency, to filthiness, etc., in frontal lobe tumors is related to this loss of sublimation capacity. (*e*) A certain incoherency in conduct is a necessary result of these changes noted, plus a motor component itself. Hence the stereotypies, tics, perseverations, repetitions, monotonies, so frequently observed in these patients.

Orientation for the external world, time and space, is apt to be involved more than personal orientation. Complete disorientation, as in Korsakoff's syndrome, is occasionally found.

At times hallucinations of smell appear from pressure on the olfactory pathways, or hallucinations of sight, photomata, from similar pressure on optical pathways at the base.

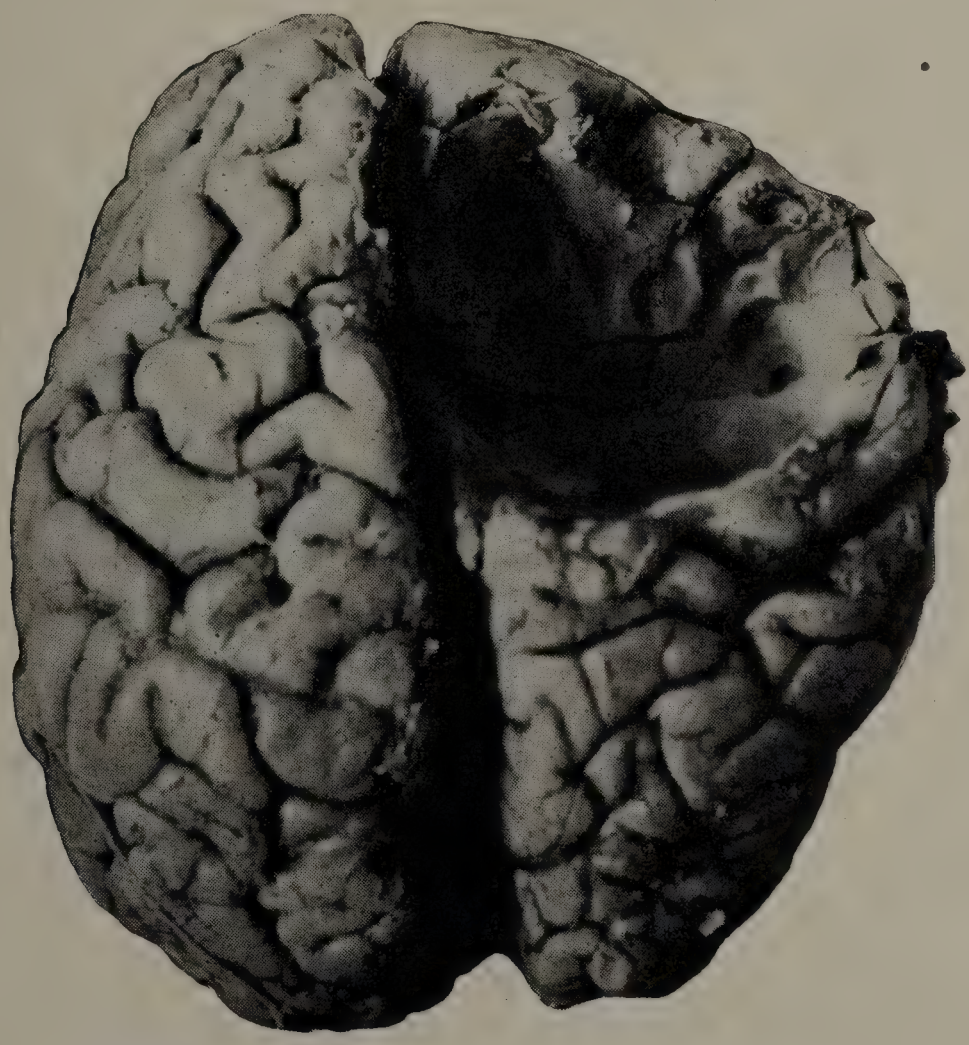


FIG. 378.—Depression in brain after removal of a frontal tumor. (Goodhart.)

Vertigo, with a drunken gait or related disturbance of equilibrium (first described by Bruns), may be ascribed probably to the higher association of space perceptions. These were thought to point to frontal involvement of cerebellar components, the gait being closely related to that of cerebellar or striatum syndromes—the patient staggers to the tumor side. Here Dimitz and Schilder, are inclined to implicate the psychical component.[1] Further these patients show a slight tendency to hypertonicity of the extrapyramidal type. Whether this is to be regarded as a frontal sign or due to possible pressure on the striatum remains for future investigation.[2] Adiadokokinesia and asynergia are usually absent here, however. (See Cerebellar Syndromes.)

[1] Fremel and Schilder: Wien. klin. Wchnschr., 1920, No. 49.
[2] Wrexberg: Zeit. f. d. ges. Neur. u. Psych., 1921, **71.**

Involuntary defecation or urination occurs at times; most frequently with somnolent patients. The patient whose tumor is here figured had a marked cerebellar gait, was keen and active, jocular and happy, with occasional loss of bladder and rectal function and hallucinations of smell. The tumor occupied the left frontal pole (Fig. 377).

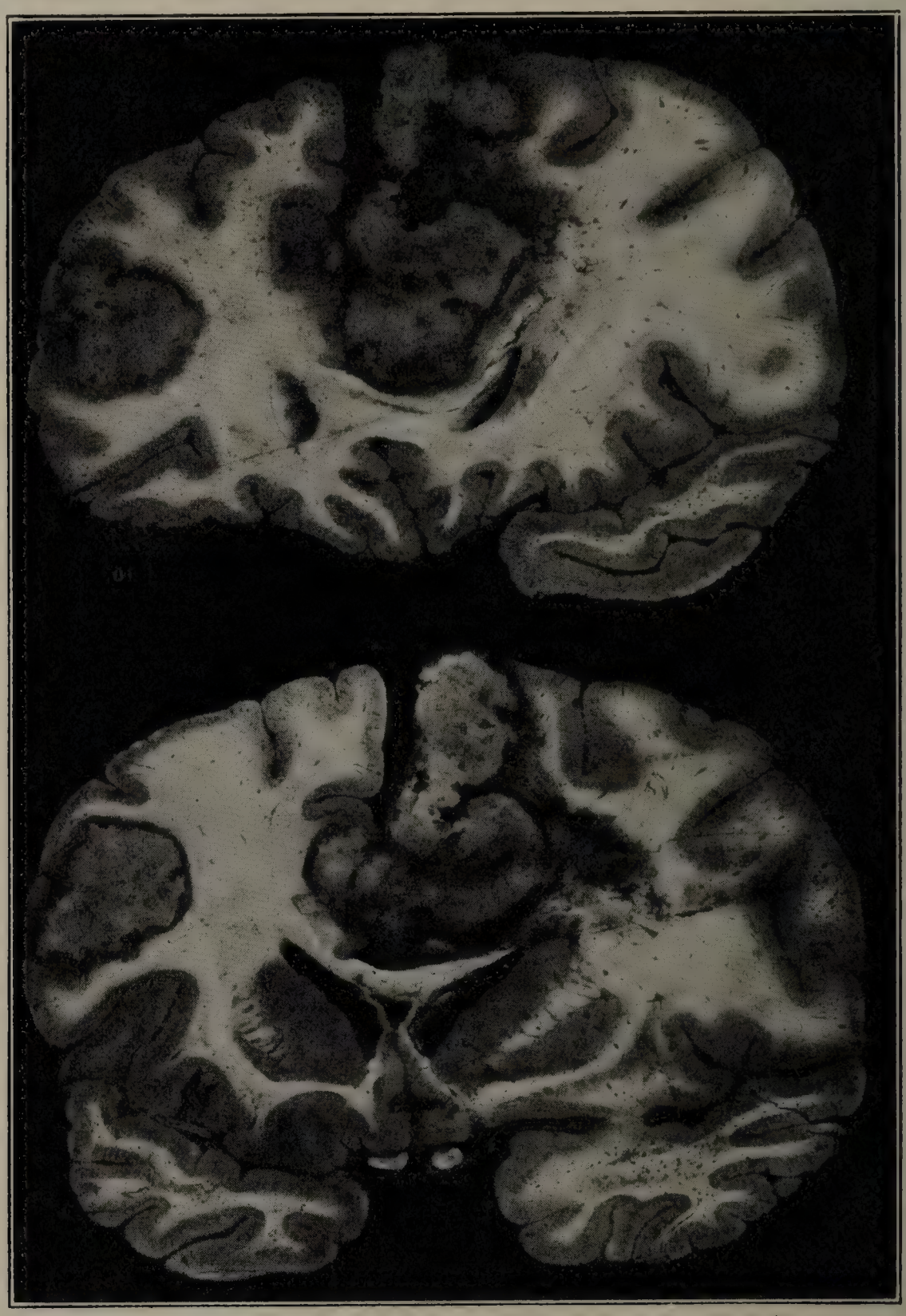


FIG. 379.—Multiple sarcoma of brain. (Larkin, Jelliffe.)

Apraxic disturbances are occasionally met with in frontal tumors, and those involving or pressing upon Brocas' convolution, left side, cause mild (paraphasia), or severe motor aphasias in individuals using the right hand for expression, usually of gradual onset and often remittent in character.

Other motor signs are stiffness in the neck with forward and back-

ward fixations of the hand and tendencies to tremor of the hand on the side of the tumor. In a third of the cases, epileptiform attacks, often Jacksonian, occur, from pressure on the motor area.

In certain patients a diagnosis of a slowly advancing frontal tumor may be made on the general signs of tumor, some loss of smell on the tumor side, and a true retrobulbar neuritis with central scotoma, ipsilateral to the lesion, and a papilledema of the opposite eye. Here the phylogenetically younger and more susceptible macular fibers degenerate on the side of the tumor. Bianchi regards the hemianopic disturbances, in experimental lesions of the prefrontals, to be due to diaschisis.

Dandy's intraventricular air injections are of some localizing service in some frontal tumors.[1]

Central Convolutions.—The functions chiefly involved are those of the voluntary muscular activity, hence paresis, paralysis, spasms. Tumors of this region are practically never latent. Irritative phenomena, spasms, convulsions, speak for cortical location; paralysis for deeper-seated lesions involving the pyramidal paths from the motor areas. Small tumors, cortically located, cause isolated Jacksonian attacks; the more extended the tumor the more widespread the muscular involvement; even small tumors, however, may cause widespread Jacksonian or grand mal symptoms. Often the first observed motion accompanied by tingling affords a clue to the more definite localization of the tumor; again an orderly and uniform progression in the development of a Jacksonian attack is valuable in localization.

Monoplegiæ[2] and monopareses are not infrequent from small lesions, and in the beginning of the tumor growth. The slow extension of the paresis or paralysis is of diagnostic moment. Advancing hemiplegia is often accompanied bv the vasomotor phenomena seen in hemorrhage—especially in deep-seated lesions. The usual signs of an organic paralysis (*q. v.*) are present.

Psychical symptoms of general nature are not infrequent. Occasionally large tumors will cause a Korsakoff syndrome. Katatonic symptoms may also appear. Sensory phenomena are frequent in postcentral convolution tumors. The phenomena have been extensively described in the chapter dealing with sensory changes due to cortical and subcortical lesions.

Central convolution tumors are among those more readily localizable, and usually more accessible to operative relief.

Parietal Lobes.—When the tumors press forward toward the posterior central lobes sensory signs are produced similar to those already mentioned. Left-sided tumors, in right-handed persons, especially of the inferior parietal lobes, cause cortical sensory aphasia of Wernicke of various grades. Alexia and agraphia may also be found in left-sided lesions.

[1] Jüngling: Ergeb. d. med. Strahlenf., 1926, **2**, 1—a very complete summary upon ventriculography.

[2] **Bergmark: Monoplegia, Brain, 1910.**

Epileptic attacks with hallucinatory auras of taste occur; also a general Korsakoff syndrome may develop. Other pathways from the sensory areas may be cut off by tumors in this region; hence either hemianopsias, optic agnosias or optical aphasias (gyrus angularis). Apraxic disturbances are often of value in localizing left-sided tumors. Katatonic syndromes, confusion, apathy, or general loss of orientation may be encountered but are equally present in right-sided and left-sided cases.

Occasionally parietal tumors give rise to ptosis, paresis of the lateral movements of the head and the conjugate motion of the eyes to the opposite side. (Cephalorotary and oculorotary paralysis, see Plate X.)

Deep-seated lesions may impinge upon or involve the motor pathways.

Temporal Lobes.—Tumors of the temporal lobes are comparatively common as brain tumors are counted. Fifty-nine of 276 of Cushing's[1] series of supratentorial tumors were located in the temporal lobes. Even a cursory review of the more recent studies shows that the accepted diagnostic criteria laid down by Bramwell[2] in 1899 and later by Kennedy[3] (1911) must be radically modified, especially as to the emphasis laid upon Jackson's[4] uncinate fits, important though they may be; present in 14 of Cushing's 59 patients only. Bramwell early recognized the necessity for separating the discussion as to left and right temporal localizing features and Knapp[5] in his series of studies on temporal lobe tumors has emphasized the importance of this distinction.

Much as seems desirable to give a complete resumé of recent studies, only the barest outlines can be presented in a text-book in which Knapp's conclusions are given emphasis and Cushing's valuable contribution to the perimetric opportunities for early diagnosis incorporated.

Knapp summarizes his important studies as follows:

The most important and characteristic localizing symptoms, unfortunately only for left-sided temporal tumors, are sensory aphasia and paraphasia and its accompanying symptoms, amnesic aphasia, apraxia and agnosia as the most common symptoms found in left-sided temporal tumors.[6]

The most frequent symptoms of tumors of both temporal lobes are a late appearing, transitory or recrudescent partial oculomotor paralysis, especially a cotemporaneous, seldom contralateral or double ptosis and a frequent mydriasis, without pupillary deformation.

[1] Brain, 1921, **44**, 341.
[2] Ibid., 1899, **22**, 37.
[3] Arch. Int. Med., 1911, **8**, 317.
[4] Brain, 1889, **12**, 358.
[5] Die Tumoren des Schalfenlappens, Zeit. f. d. ges. Neur. u. Psych., 1918, **42**, 226. Die Geschwülste d. rechten u. linken Schläfenlappens, 1905. München. med. Wchnschr., 1908. Deut. med. Wchnschr., June 27, 1918.
[6] Marcus: Acta med. Scand., 1921, **55**, 486. Also consult van der Horst: Zetschr. f. d. g. Neur. u. Psych., 1927, **108**, 255.

Next in importance are paretic phenomena of monoplegic or hemiplegic character, due to pressure upon the brain stem giving rise to a predominantly contralateral, rarely a homolateral or bilateral, hemiplegia alternans superior.

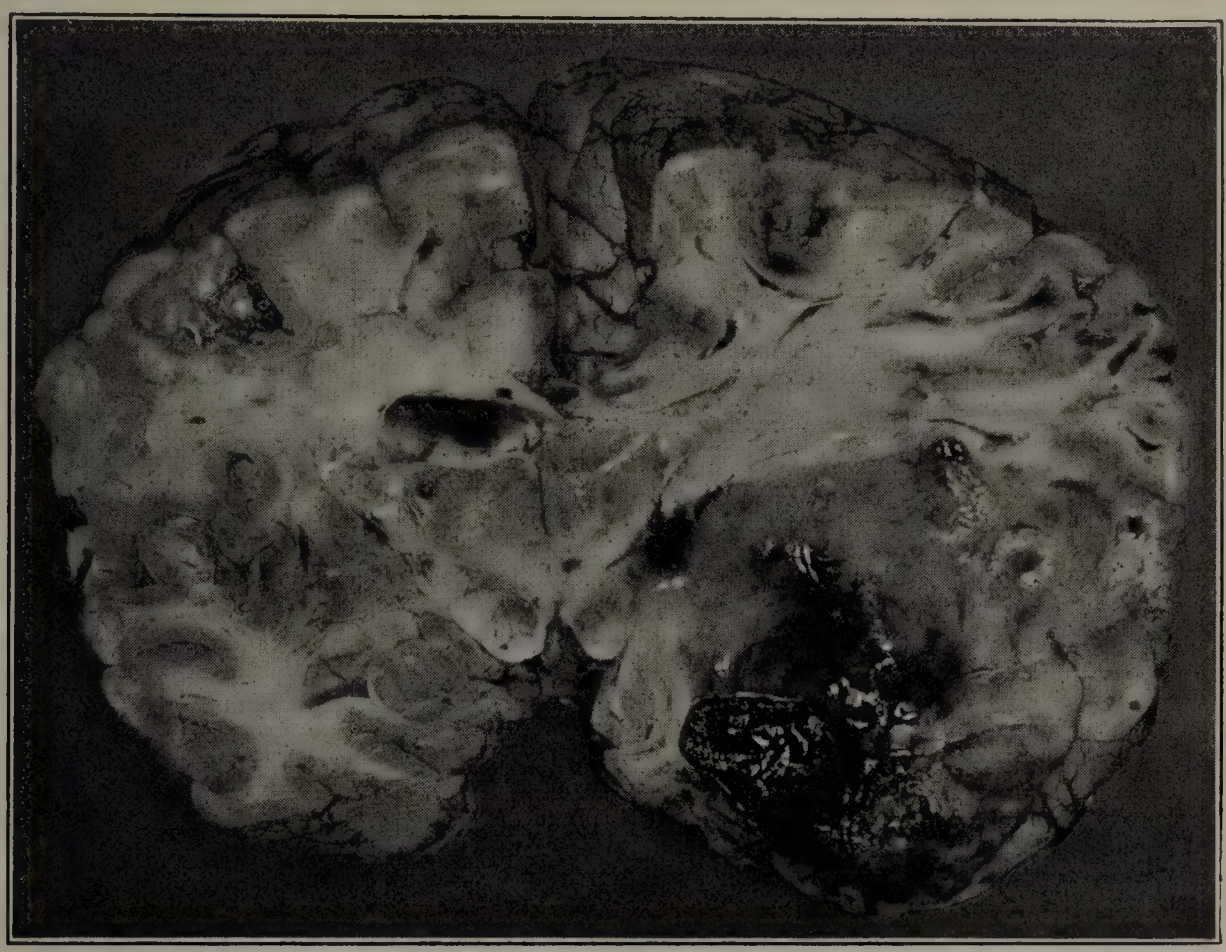


FIG. 380.—Glioma of temporal lobes. (Jelliffe.)

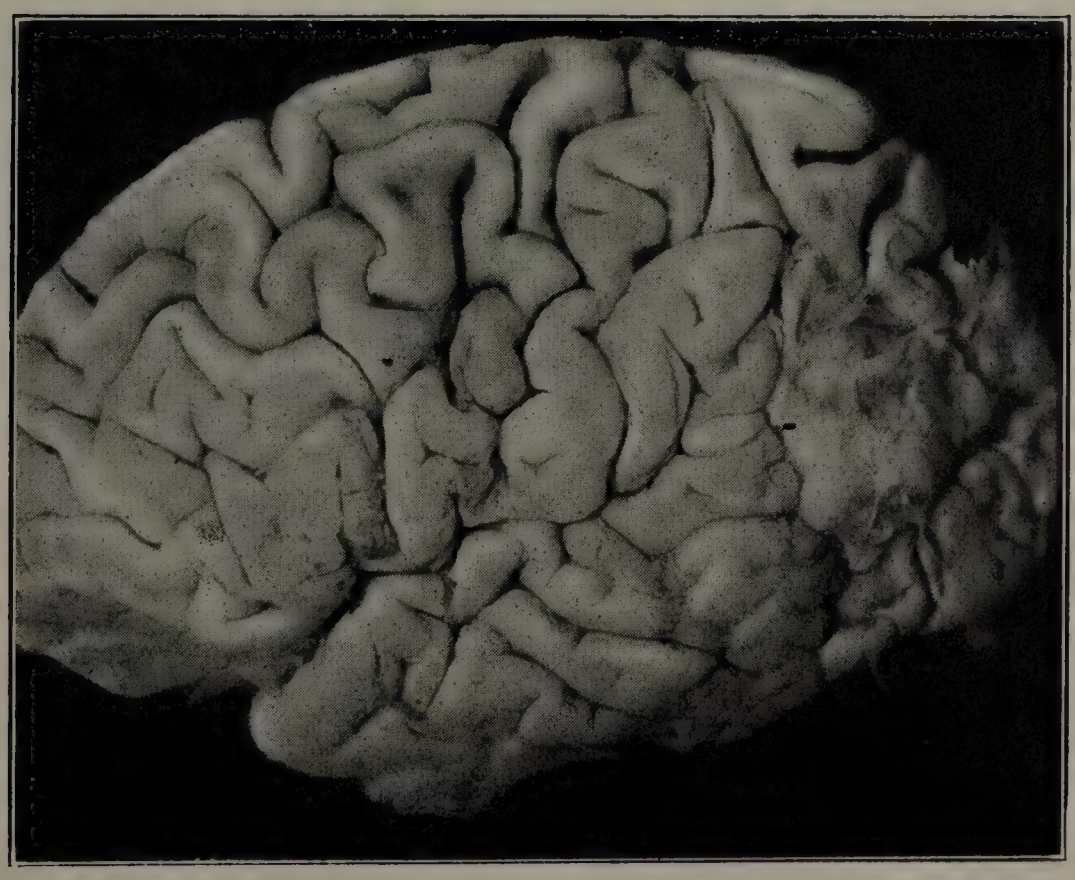


FIG. 381.—Tumor of temporo-occipital lobes. (Larkin and Jelliffe.)

Thirdly, a disturbance of equilibrium, which is separated with difficulty from a cerebellar ataxia, and to be designated as a pseudo-

cerebellar ataxia. Pain in the back of the neck, stiffness of the neck muscles, grinding of the teeth, areflexia of the cornea, nystagmus, and abducens paralysis as symptoms of posterior fossa involvement are important for a diagnosis of temporal lobe tumor.

Characteristic of temporal lobe involvement, but not frequent, are smell and taste disturbances at first ipsilateral and later contralateral.

Of less value are the following but not infrequent signs.

Neuritic symptoms of posterior root involvement, pains, sensitiveness to deep pressure of nerves and muscles, involvement of the tendon reflexes and muscle tonus.

I'sychical disturbances chiefly Korsakoff's confusional syndrome, delirious states, hallucinations, personality changes.

Epileptiform or apoplectiform convulsions (uncinate fits), thalamus symptoms, hemianopsia, hemianesthesia and anesthesiæ.

This tablet compressed formula of Knapp, however, generalizes the situation out of all proportion to the clinical data and Cushing's[1] discussion offers a much more satisfactory series of observations. His findings are therefore offered in brief recapitulation:

Convulsions: These occurred in 29 of his 59 cases. They were few in number. A few of the patient had two or three, and twelve was the greatest number. Of the historically interesting Jacksonian uncinate fits, 24 only had such attacks of 59 cases. Only 14 of these had the classically described olfactory or gustatory impressions. Postimpressionistic descriptions of these were characteristic, but the perimetric observations of Cushing were more valuable in the diagnosis than these uncinate fits, even though when these occurred they were very striking. Thus the perimetric changes were verifiable in at least 18 in which no history suggestive of petit mal or dreamy states could be secured.

Visual Hallucinations.—These occurred in 13 of Cushing's 59 cases. They are usually related to the seizures. They are probably related to an implication of the geniculo-calcarine radiations. In Jackson's case they appeared as seeing "a little black woman engaged in cooking," a type emphasized by other observers since. In one of Cushing's cases "as in a dream, he saw, always to his left side his father and some other man in an unfamiliar room engaged in a game of cards which ultimately led to an altercation. The scene would fade away and leave him with a bad taste and smell of something indescribably horrid." While in ordinary conversation "a vague look would come over his face, he would turn his head and stare to the left and shortly after would begin to make smacking and tasting movements with his lips. This was followed by an expression of disgust and the attack would be over. He had an homonymous upper quadrantal defect produced by a temporal lobe glioma."[2]

Auditory Symptoms.—Cushing speaks of these as being uncommon (in early seen cases), and thinks aphasia to be a neighborhood rather a true temporal lobe phenomenon.

[1] Brain, 1921, **44**, 341.

[2] See reports from Cushing's Clinic in Bailey and Cushing (loc. cit.).

Pseudocerebellar Signs.—Nystagmus was present in 15 of the 59 cases; dizziness and vertigo fairly common. Ataxia and static instability also are infrequent.

The perimetric findings emphasized by Cushing are due to involvement of the temporal loop fibers of the optic radiation—geniculocalcarine fibers (Meyer)—which cause homonymous field defects in the upper and lower quadrants.

Occipital Lobe.—The end-projections of the optic tracts are located here particularly in and about the calcarine fissure. Homonymous hemianopsia in the contralateral field is a striking localizing symptom of

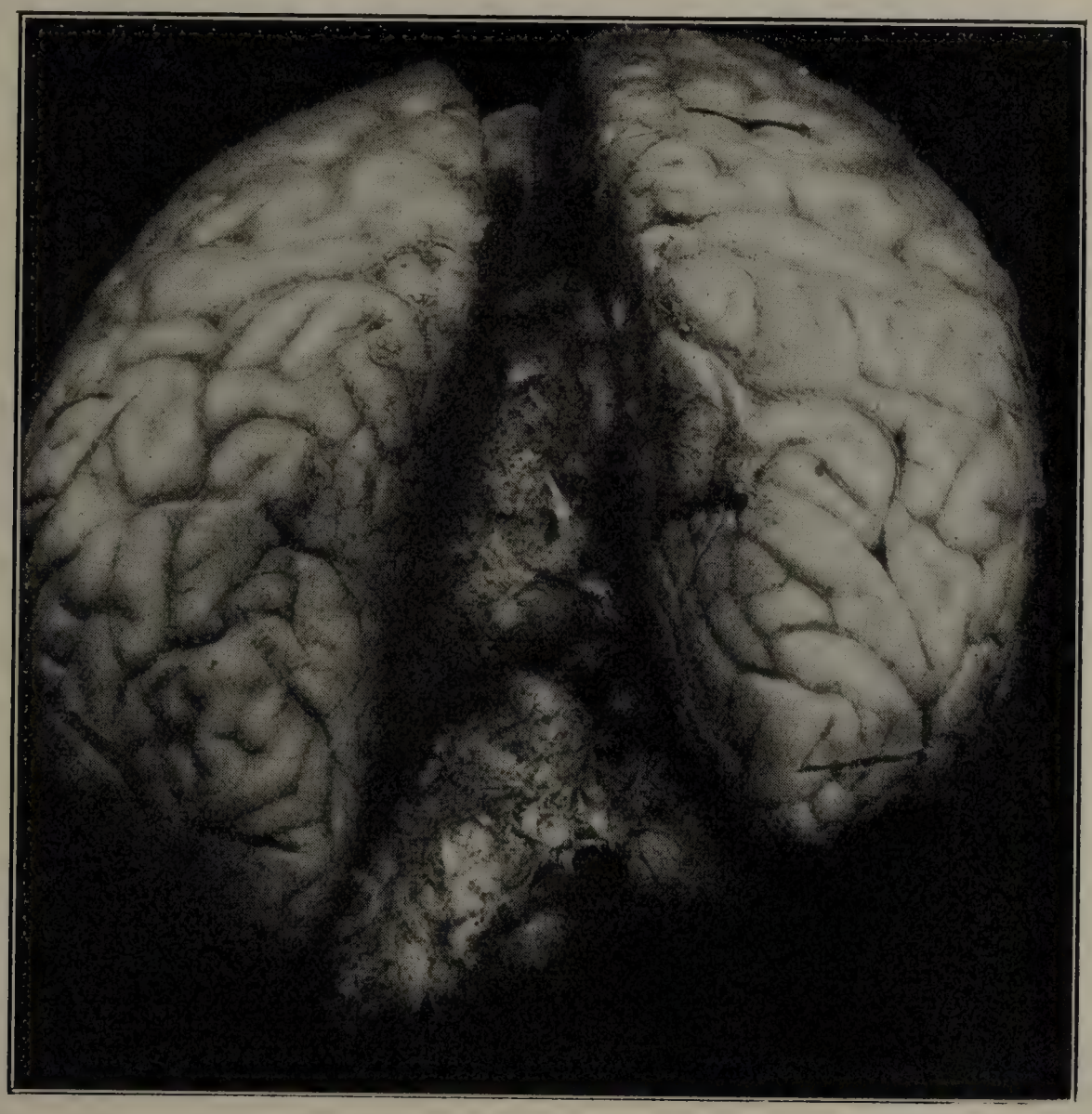


Fig. 382.—Tumor arising from the meninges and pressing down to the corpus callosum. (M. P. J.). (Baldwin.[1])

tumors of this area. This hemianopsia usually spares the papillomacular bundles and is often unperceived by the patient. Quadrant hemianopsia is also found.[2] Tumors may exist and hemianopsia be absent. It is most often present with tumors of the median aspect of the occipital, also with those lying on the convex surface, and hence the more readily removable. Various stages of blindness may also result, and there may exist a mind-blindness from left-sided tumors, also alexia, agraphia and sensory aphasia.

[1] Jour. Nerv. and Ment. Dis., 1901.

[2] Bramwell: Lesions of Occipital Lobe and Affections of Vision, Clinical Studies. Holmes and Lister, loc. cit. See Chapter on Cranial Nerves, p. 307. Bourguet, loc. cit.

Other optic signs, such as phonemes, various scintillating scotomata, optical hallucinations and illusions occasionally result from occipital tumors, and these may exist as auræ preceding general epileptic convulsions.

Tumors on the inferior surface, by compression of the cerebellum, will cause cerebellar signs,[1] and occasionally palsies result from tumors lying on the external surface of the occipital lobe.

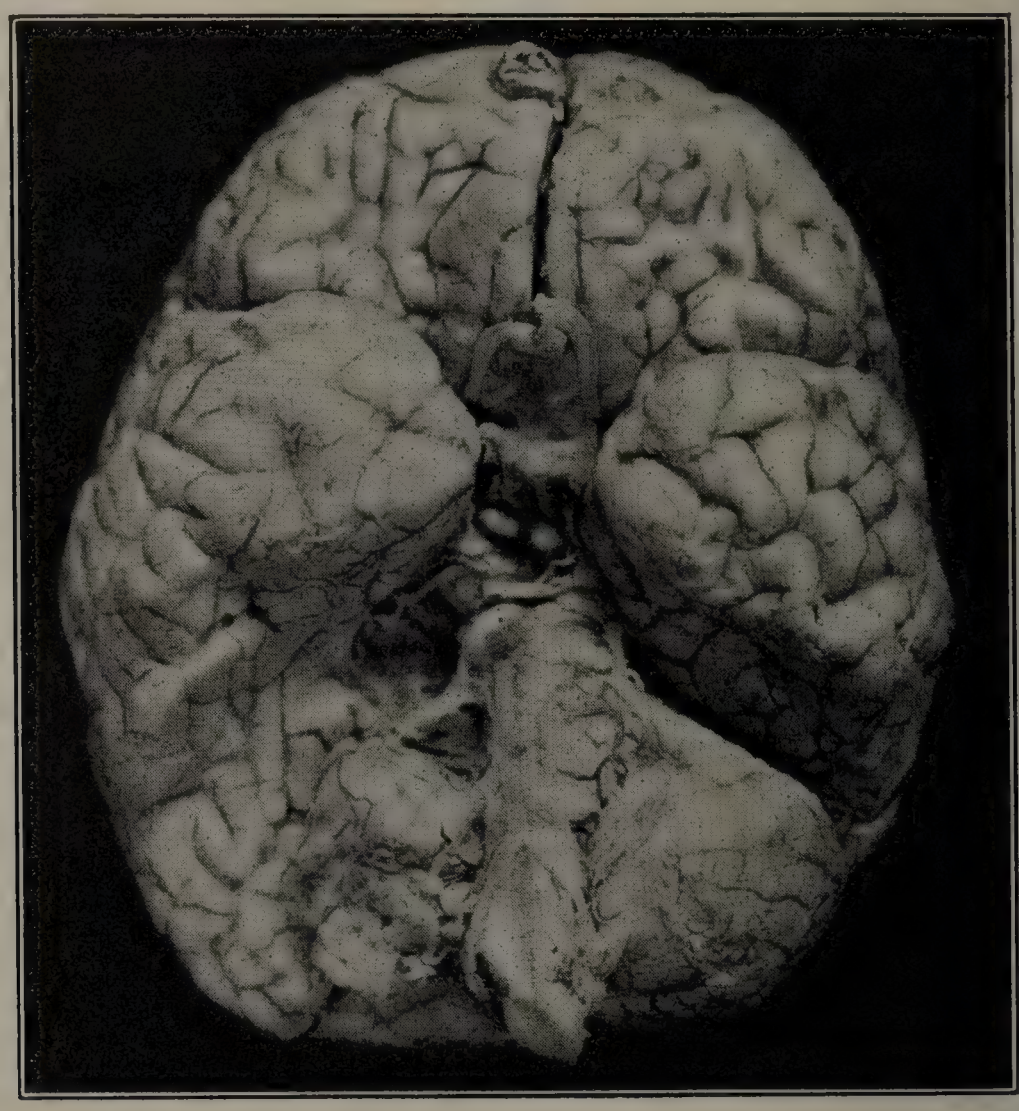


FIG. 383.—Tumor with atrophy of the cerebellum. (Jelliffe.)

Corpus Callosum Tumors.—These are comparatively rare. Ayala[2] collected 112 cases up to 1919. Bristowe was one of the first to report upon them. Schüpfer in 1899 collected 25 observations since which time to the appearance of Mingazzini's exhaustive monograph,[3] excellent descriptions have been given by Duret, Schuster (1902), Lippmann (1907), Panegrossi (1908), Lévy-Valensi (1910), Milani (1914) and Ayala (1919).

Sarcomata seems the most frequent. Hemorrhage and softening are to be looked for. The location of the tumor in or involving the callosum varies also. In about 25 per cent the entire callosum has been implicated. In the others the knee, the trunk, the anterior and posterior thirds, sometimes the entire callosum has been involved.

[1] Gordon: New York Med. Jour., 1921, **113**, 540.
[2] Riv. d. Pat. Nerv. and Ment., 1915, p. 449.
[3] Mingazzini: Der Balken, Springer, Berlin, 1922, for literature to 1922.

In only a few of the cases reported has the tumor been restricted to the corpus callosum, 15 in the 112 cases reported. (Mingazzini.).

The symptoms vary greatly. Some cases of congenital absence are known with but few symptoms, whereas Cameron[1] recently reported a case with wide spread defect in the corpus callosum with manifest mental disturbances. Imbecility being prominent. Lévy-Valensi emphasized first the negative character of the situation. The more positive signs are: (1) Psychical. The type has been given the name of Raymond's syndrome. (2) Motor-praxic signs. Apraxia and dyspraxia. (3) Later occurring unilateral or bilateral irritative or paretic signs.

The general symptoms of brain tumor are absent in but few instances. At least 78 per cent of the cases show them. Headache is the most frequent of these, at least 50 per cent. It may be unilateral, but that does not necessarily indicate the main seat of the tumor growth. Papilledema was noted in 68 of the 112 cases of Ayala's collection. Vomiting is less often observed. Dizziness and epileptiform attacks are still less frequent.

The psychical symptoms are the most frequent. Practically all patients show them in some degree. Gradually developing sleepiness, forgetfulness, apathy, and depression often of a very insidious onset are among the earlier signs. Raymond described a psychical syndrome, which has been frequently observed but is not universal. It begins with disturbances of association of ideas, memory defects, especially of recent events, peculiarities of conduct, changes in character and in consistencies of mood. As the disturbance increases the psychical syndrome becomes more diffuse and less characteristic. Mental confusion, hallucinations and delusions develop, maybe the sleepy stupor is broken by attacks of agitation or excitement, but there is a tendency towards increasing depression, torpor, and coma.

Finer localizations of the tumor involvement on the basis of the psychical picture are often possible, but the student is referred to the studies quoted for the many details which cannot be included here. Stern[2] has studied this problem in detail. Mingazzini has given a résumé of the entire literature.

Motor disturbances are frequent and gradually become more and more pronounced. At first weakness in the extremities, of the side opposite to the greatest extension of the tumor, is observed. Unequal bilateral hemiparesis or hemiplegia has been present in about 50 per cent of Ayala's collected cases. The lower extremities rarely show a paraparesis, and only a few real paraplegics are reported.

A number of isolated motor phenomena, tremors, choreatic, choreo-athetoid, choreo-atheto-cerebellar movements, Jacksonian attacks, etc., have been observed in isolated cases. In a few the cranial nerves have been implicated. Bruns laid great stress on this absence of cranial nerve signs as of diagnostic importance, but their appearance, later in the history is by no means uncommon.

Motor apraxia is a symptom of specific importance. The number of

[1] Canadian Med. Jour., June, 1921.

[2] Arch. f. Psychiatrie, 1914.

carefully analyzed cases is small. It is of ideokinetic type; is chiefly observed in the left side, or when bilateral the left side shows more striking involvement. Lesion of the anterior half of the corpus seems to be the chief producing cause of this apraxia.

Disturbances in equilibrium, ataxic gait, and a small museum of sensory, motor and reflex changes may be collected in the histories of the reported cases. These must be consulted in the monographs cited. They are rarer and isolated happenings, chiefly due to extension of the tumor to other than callosum pathways.

Tumors of the Optic Thalamus.—The symptoms are discussed under the section on the Thalamic Syndrome (*q. v.*).

Basal Ganglia Tumors.—Involvement of the lenticular nucleus (see Wilson's disease), tumors of the midbrain, pons and medulla have been discussed.

Those of the cerebellum and cerebello-pontine angle will be found under Cerebellar Syndromes.

Diagnosis.—Multiple sclerosis, paresis, arteriosclerotic disease, all forms of headache, tuberculous meningitis, chronic hydrocephalus, and hysteria are the chief conditions causing difficulty.

Multiple sclerosis, if the patches are solely cerebral, may cause confusion, especially in the acute cases, as described by Marburg and others. The bitemporal pallor of the disks in this disorder differs from the usual pressure changes in the disk. Nystagmus is not a frequent brain-tumor sign (temporal, cerebellar). Other signs of pyramidal tract implication may be identical. Headaches are usually absent, also nausea and vomiting.

Paresis with marked focalization of the pathological process may for a time closely simulate the picture of tumor.

Pseudoparetic and arteriosclerotic psychical syndromes (Korsakoff's psychosis) are frequently confused with cerebral tumor, *i. e.*, the cause of the mental picture is overlooked. Thus a tumor, which might have been removed, has been missed under the psychotic disguise. Eye-ground changes are usually positive in these cases, yet may be absent. Perimeter examination is imperative.

Headache should always be scrutinized carefully. A postinfluenzal occipital headache which is very frequent and extremely severe and persistent, is frequently highly suggestive of brain tumor. The headaches from lead poisoning, anemia, and nephritis are also to be excluded.

Brain abscess and tuberculous meningitis must be excluded on the ground of their difference in development of symptoms. Symptomatically speaking, they may be considered as tumors. This is also true of chronic hydrocephalus.

Cerebral puncture, withdrawing a small plug of brain tissue through a trephine opening and cannula, is often of great aid in diagnosing very puzzling cases.

Prognosis.—No definite prognosis can be laid down. All depends on the site and nature of the tumor. In general, apart from surgical relief and from medicinal treatment of syphilomata, the outlook is pessimistic. Sudden death is not infrequent and lumbar puncture is

an extremely dangerous procedure with brain tumor, often leading to sudden collapse and death, especially with tumors of the posterior fossa.

Syphilomata and gummata of the brain have a fair prognosis. Better results are obtained with mercury by inunction and by iodides than by salvarsan in the beginning treatment. Salvarsan may be used later to attempt to kill off all the spirochetes, but with well-advanced syphilomata, salvarsan is apt to set up a dangerous reaction. Round, hard gummata do not absorb, as a rule, and are best considered surgically.

Treatment.—Medicinal treatment, excepting for syphilomata, is useless, and involves a waste of valuable time. The chief objects to be attained are *early diagnosis*, immediate exclusion of syphilis by serological tests, eye-ground examination with particular study of the perimetric fields, *exact localization* as soon as possible, and *surgical removal* or *palliation* (decompression) to save the eyesight or to gain time for a more exact localization.

The details for applying these principles have already been noted. The results to be expected in any particular case are problematical, yet from 10 to 20 per cent. of all brain tumors (seen in the large) have been removable, with at least in 10 per cent. practical recovery. Even with such chances against him the patient should have the benefit of the doubt if a competent surgeon is available. Surgical skill is a very large factor in the results; a good abdominal surgeon is not necessarily a good brain surgeon. The brain is semifluid and an intricate switchboard of highly important structures; there are *no unimportant areas* in the brain. To speak of *latent* areas implies latent intelligence on the part of the speaker. Many surgeons have treated the brain in the past as though it were an abdominal viscus; the results have been disastrous.

Most brain operations are best done in two stages. Decompression, usually subtemporal, alone is often the only possible procedure. It often relieves a recently acquired blindness.

The situation referable to brain surgery for cerebral tumors is rapidly advancing, and better and better results are being obtained and regions hitherto impossible to reach (pituitary, etc.) have been approached with results which a decade ago would have been impossible.[1] Notwithstanding all this, the general attitude should be one of extreme caution. With increasingly refined methods of clinical examination, neurologic, psychiatric, roentgenologic, metabolic, etc., the prognosis is improving.

It is too early to generalize upon the use of the roentgen ray or of radium in brain tumor therapy but at times the results obtained have been most satisfactory.

[1] See Starr: Brain Surgery. Halsted: Treatment of Brain Tumor. White and Jelliffe: Modern Treatment of Nervous and Mental Diseases. Sachs, E.: Present Day Status of Surgery of Brain Tumors, South. Med. Jour., 1927, **20**, 171. Roentgen Diagnosis of Brain Tumors: See monographs by Schüller, Hirsch, Sicard: Rev. neurol., 1926, **2**, 421. Mayer: Fortsch. d. Roentgenet., 1926, **35**, 187. Olivercrona: Acta rad., 1926, **7**, 259. Eiselsberg: Arch. klin. Chir., 1926, **142**, 203. Jüngling: Ergebn. d. med. Strahl., 1926, **2**, 1.

CHAPTER XIV.

NEUROSYPHILIS.

NEUROSYPHILIS[1] is so kaleidoscopic in its clinical manifestations, that the significance of the *Treponema pallidum* infection as a primary etiological factor in the production of nervous disorders is frequently overlooked. The physician with his eye riveted upon a clinical picture, be it an amyotrophic lateral sclerosis, a failing memory, a persistent nervous weakness, an isolated cranial nerve palsy, a progressive muscular atrophy of the arm muscles, a protracted neuralgia, a failing eyesight, a progressive deafness or a profound metabolic disorder, may readily overlook the fact that syphilis is the unique cause for these different syndromes.

There is no field in medicine wherein similar disease pictures may arise from so many differing causes as in the domain of the nervous system. Nor, on the other hand, where a single etiological factor may give rise to so many dissimilar clinical pictures. Hence the complexity of the whole subject, and the need for iterating and reiterating the advice that in nearly all of the clinical pictures which have been given descriptive terms in nervous or mental disease, the possibility of the syphilitic infection as a direct etiological factor, as a complication, or as causing confusion, should be borne in mind. Hence the necessity for detailed and minute inquiries into all of the possible symptomatology of hereditary or acquired syphilis, which in all questionable cases should be supplemented by complete serological and cytological examinations of the blood and cerebrospinal fluid.

The essentially distinct dividing lines between the lesions of hereditary syphilis, acquired syphilis, and so-called metasyphilis which have been orthodox teachings are slowly being obliterated, and it now seems strange that it could have been taught that spinal and cerebral syphilis should have been considered a different process from that which caused tabes and general paresis.

Since it has been demonstrated that both tabes and general paresis are syphilitic, the variety of syphilitic disorders showing fairly definite clinical pictures has been enlarged. Further, with the recognition that many acute and subacute psychoses are due to syphilitic processes in the brain tissues, the psychiatric syndromes have been further extended.

In the present chapter, then, the discussion will concern itself chiefly

[1] Consult recent monographs by Nonne, 1925; Gennerich, 1920; Tony-Schmidt Kraepelin, 1920. Abstracts, Am. Jour. Syph. Citations, Quartery Cumulative Index.

with clinical forms, without any extended attempt being made to differentiate types, which in reality are so kaleidoscopic that they defy description. At the same time it needs to be emphasized that intermediary forms are every-present realities. Classical pictures of a disease are largely literary efforts. They are the product mainly of the descriptive art and are constantly undergoing revision by reason of enlarging experience and newer points of view. In the conflict going on in the human organism against the infectious agent, the resulting manifestations do not show classical types, but rather a multiplicity of variations, with here and there the statistical predominance of this or that trend in the reaction, which the classification tendency of man calls a *disease* and gives it a *label, i. e.,* a *diagnosis.*

History.—Leoncino as early as 1497 has been said to have described paralysis as syphilitic.[1]

Joseph Grünbeck (1503), Emser (1511), Ulrich von Hutten (1519), all lay writers, mention paralyses of the limbs, as due to the infection. Emser speaks of a patient, a syphilitic paralytic, and with a psychosis, as having made a remarkable recovery, under treatment by Bruno, by making a vow. Paracelsus (1530), confusing gonorrhea with syphilis, as is still being done through the senseless use of the term "venereal" disease, recorded descriptions recognizable as a syphilitic meningitis. He spoke of the syphilitic virus as affecting all of the organs of the body. Nowhere in the writings of Fracastorius (1521), who first originated the name *syphilis,* are direct references to involvement of the nervous system to be found. Nicolaus Massa (1556) gave an early description of syphilitic neuralgias. Borgarutius (1567) described neuralgic pains due to syphilitic disease of the meninges. Amatus Lusitanus (1561) described headaches due to intracranial osteitis of syphilitic origin. Botalli (1536) made an observation that blindness might be due to syphilitic disease of the brain. Ferrus (1537) Morgagni (1600) noted the arthropathies of syphilitic origin.

During the following century many references may be found in the writings of medical men showing the recognition of the relationship of syphilis to nervous disease. Only a few can be mentioned here. Thus, Guarinoni (1610) described epileptic attacks from syphilis of the brain. Vidus Vidius (1611) described epilepsies as due to syphilitic cranial caries. Thiery de Hery (1634) and Zechius (1650) called attention to syphilitic spasms as well as to syphilitic epilepsies. Zacutus Lusitanus (1644) described blindness due to gumma of the brain, quoting Botalli a century ahead of him. In 1696 a special treatise on syphilitic pains was written by Blagney.

It is of historical interest to call attention to the writings of Scholtzius (1610) and to those of Willis (1672) *a propos* of the subject of general paresis. Willis gave the first description in which one could possibly recognize *general paresis.*

[1] See Sudhoff's volume by Garrison on History of Syphilis (Bloch and Nonne).

[2] Streeter, of Boston, a most capable student of medical history, doubts this reference after a careful study of Leoncino's "de morbo Gallico," 1497.

By the end of this century (seventeenth) a fairly broad view of syphilitic nervous disease had been obtained. Syphilitic headaches were described by Felix Plater (1641). Rhodius (1657) described gummata of the dura and syphilitic hemiplegias; Ballen (1663) spinal syphilitic disease and spasms in the facial region; Cummius (1684), diplopias and eye palsies. Astruc has reviewed these writings completely.

During the eighteenth century the outlines of the picture expanded rapidly. Intercostal neuralgias (1762), deafness, loss of smell, caries of base of skull (1762), sciatica (1745), psychoses. Syphilitic mania was described by Sanché in 1777, amaurosis (1748), facial palsy (1758), leptomeningitis (1766), syphilitic arterial disease (1766), paraplegias (myelitis) (1771), and a number of other conditions were described, and may be seen in Lagneau's interesting monograph in which 234 case histories are collected. The works of Astruc, Bonet, and Morgagni offer much for the student interested in the historical side of this large subject.

It is not without special interest that John Hunter (1787) stated that he had never observed the effects of syphilis in the internal organs, including the brain. The weight of his authority retarded progress for many years, especially in England. As a matter of fact, it was not until Ricord's sound observations were published that the results of Hunter's stupidity were fully remedied.

Virchow's studies (1847) on phlebitis and arteritis laid the foundation for the modern knowledge of bloodvessel syphilis, although it may be recalled that Morgagni (1766) and Horne (1782) both made extremely important studies on vascular syphilis. These have been fully developed by Huebner (1874) and by Alzheimer (1904).

The studies of Virchow on the formation of gummatous granulomata and related syphilitic phenomena, practically established the modern era of study of the pathology of this aspect of the disease reaction.

The succeeding years have filled in the picture with a number of important details, the chief additions having been those of Nissl and Alzheimer, who established the highest criteria for the pathology of this disease so far as the nervous system is concerned.

The latest chapter in this interesting history is that dealing with the discovery of the inciting agent, and the final clearing up of the entire subject of etiology and modes of infection. Schaudinn[1] (March 3, 1905) found the parasite which he first called *Spirochæta pallida*. Its synonymy has varied to accord with principles of botanical and zoological nomenclature. *Treponema pallidum* has the best sanction. Doele (1892) is said to have first seen the parasite, but Schaudinn, then Epaschen, Fischer, Metchnikoff, and Roux established its identity and showed its affiliations with the protozoa.[2]

[1] Arbeiten aus dem Kaiserlichen Gesundheitsamte, April 23, 1905.

[2] See Noguchi: "Spirochetes," Jour. Lab. and Clin. Med., March, 1917. Also consult Stein, J. B.: Med. Record, April 13, 1912, for an interesting history of the details of the discovery of Treponema pallidum, illustrated by photographs of the chief investigators of the problem to that date.

The parasite has been found in patients with the signs of congenital syphilis. It has also been seen in syphilitic gumma of the brain and spinal cord, in syphilitie meningitis, even in the cerebrospinal fluid, both of congenital and acquired syphilitics. Moore (1913), Noguchi, Nichols, and Hough found it in the paretic brain, and it has been found in the spinal meninges of tabetics.[1]

The earlier studies of Neisser, Metchnikoff and Roux and later those of Plaut, Jahnel and others have laid bare the entire story of the inoculability of the disease and its transmission from animal to animal, while utilizing the knowledge gained by Bordet and Gengou, Wassermann and his pupils elaborated a serobiological technic which critically applied and carefully interpreted has made one partly independent of clinical or anamnesic data relative to a knowledge on the part of the patient of the infection. In more recent years Sachs, Georgi, Kahn, Guillain, Leriche and others have elaborated other serobiological tests of great importance.

Thus, in two decades of the twentieth century a flood of light has been thrown upon the infection and its relation to other protozoan-caused infections, notably trypanosomiasis,[2] which has illuminated and made clear the entire path which has been so busily travelled since Columbus, as claimed by some (Bloch) and denied by others (Sudhoff), brought this most portentious exotic to the old world.

Frequency. — An enormous literature is available to show that neurosyphilis is very widespread and frequent. It has been found in greatly varying percentage variations all over the world. Probably it is the most frequent of all types of disorders of the nervous system. Great variations exist in different countries, parts of countries and in different decades. A widespread source of infection was the World War, which from many factors induced great lowering of the erotic morale. The statistics regarding consequent neurosyphilis are not yet available. Whether there will result a great rise in the neurosyphilis aftermaths remains to be determined by strict statistical methods. Increased knowledge of precautionary measures has and will undoubtedly diminish the incidence of neurosyphilis.

Diagnosis.—The diagnosis of neurosyphilis presents certain difficulties which rapidly increasing perfections in clinical neuropsychiatric examinations, laboratory technic are resolving with considerable success. These combined technics permit an almost certain diagnosis either as congenital, acquired, or as so-called para- or metasyphilis.

The chief factors in such a technic are: (1) Search for the organisms; (2) serological investigation of the blood and cerebrospinal fluid; (3) cytological examination of the cerebrospinal fluid; (4) chemical

[1] Strassmann: Meningitis, Ziegler's Beitr., p. 49. Moore: Jour. Nerv. and Ment. Dis., 1913, **40**, 172. Noguchi and Moore: Jour. Exp. Med., February 1, 1913.

[2] Spielmeyer: Paralyse, Tabes, Schlafkrankheit, Ergebn. d. Neurol. u. Psychiat., 1911. Also consult numerous recent papers by Jahnel. Bloch attached much importance to the "Lama" South American theory. Nonne (loc. cit.), who visited South America in 1922, says the diagnosis is not certain (5th ed., 1924, p. 21).

examination of the cerebrospinal fluid; (5) clinical examination of the pupillary reflexes; (6) general neuropsychiatric signs.

1. *Search for Organisms.*—The parasite has been found in the cerebrospinal fluid, but as yet in but few instances. It has been cultivated from the cerebrospinal fluid (Nichols and Hough). More recent work by injecting the fluid into the testicle of rabbits has added considerable information.[1] The results thus obtained added to those previously on record show positive findings in primary and secondary syphilis, without nervous manifestations, in a total of 2 cases (none before the eighth week); in secondary syphilis, from the third to the twelfth month, in 7 cases; never in tertiary syphilis. In the cases with objective nervous symptoms: in early syphilitic meningitis; once; neurorecurrence (eight months), once; apoplexy and hemiplegia (seven months), once; (one year), once; syphilitic meningitis, once; spinal syphilis (ten years), once; tabes, 2 cases, and progressive paralysis, 5 cases. In inherited syphilis, soon after birth, 2 cases; syphilitic leptomeningitis later in life, once, and juvenile paralysis, once. This compilation includes Frühwald and Zaloziecki's 23 syphilitic patients tested in this way. Spirochetes were found in only 4 of the total 23; once in recent secondary syphilis, once in older secondary syphilis (without nervous symptoms), once in early meningitis, and once in progressive paralysis. They add that large numbers of others with tabes, paralysis and brain syphilis were also tested, with constantly negative results. The inoculation lesion in the rabbit testicle was so slight that only the microscope disclosed its positive nature. Fildes[2] found the organism once in 15 fluids investigated. The pleocytosis was 1000 cells per c.mm. in one patient only twenty weeks after infection. The data presented show that the cerebrospinal fluid only rarely contains the spirochete. The fluid otherwise may or may not be normal, and there may or may not be a positive seroreaction and symptoms on the part of the nervous system. Thus there is no regularity in the cerebrospinal fluid findings. Newer methods (ultramicroscopic and others) are showing more and more positive results.

2. *Biological or Serological Tests of Blood and Cerebrospinal Fluid.*—(*a*) *Blood.*—Practically all forms of early syphilis of the nervous system should show a positive Bordet-Wassermann reaction in the blood. There are exceptions, but neurosyphilis, like syphilis elsewhere, gives a positive reaction in early cases.

Whereas, the number of positive findings should be as high as 100 per cent. in cases of early neurosyphilis, in its secondary and tertiary stages the number of positive results may fall to even 70 per cent., and in the latent period may sink to 50 per cent. Whether these reduced percentages speak for the good results of treatment or are dependent upon other as yet uncertain factors is yet to be decided.

[1] Frühwald, Zaloziecki: Berl. klin. Wchnschr., January 3, 1916. See Nonne.
[2] American Arch. Neur. and Psych., 1919, 1, 231.

The alcohols interfere with the reaction. Hence the results of a Wassermann test are apt to be unreliable if the patient has taken alcohol within forty-eight hours of the time of making the test.

(*b*) *Cerebrospinal Fluid.*—The behavior of the cerebrospinal fluid to the Wassermann reaction is of special significance in the diagnosis and treatment of neurosyphilis. It is almost uniformly positive in general paresis, even when small quantities (0.02 c.c.) of the serum are employed. By the use of small quantities Hauptmann and Hössli attempted to show that paresis might be separated from other types of neurosyphilis, all of which will cause a positive result, with larger quantities of cerebrospinal fluid (0.4 to 0.8 c.c.). Later studies tend

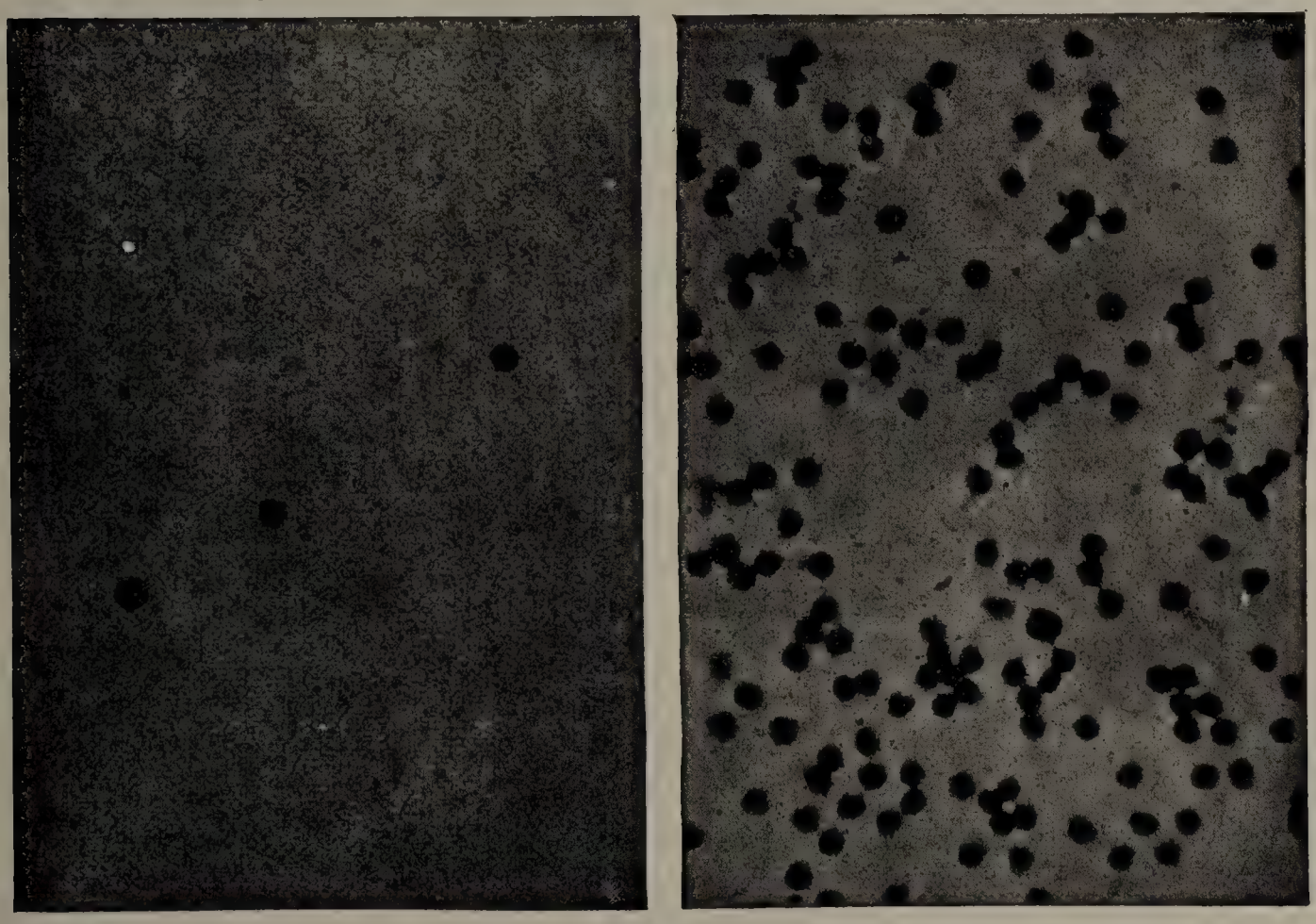


FIG. 384.—Lymphocytes in normal (left) and paretic (right) cerebrospinal fluid. (Kraepelin.)

to show that whereas this may be a useful differential at times, it is not reliable, for the intensity of the serological and cytological reactions seem to depend more upon the localization and intensity of the process than upon the kind. Thus meningeal involvements of the cord and brain tend to show marked positive reactions, while intracerebral vascular lesions tend to run low in respect to both cellular and sera criteria. Central parenchymatous types almost always show positive serological and cytological changes.

Syphilis without nervous involvement may give a negative reaction, even when large quantities of the fluid are used but many cases of undoubted neurosyphilis will show both pleocytosis and positive

Bordet-Wassermann in the fluid, long before evident neurological or psychiatric symptoms.[1] [2]

It must constantly be borne in mind in the diagnosis of syphilis of the nervous system that the reaction of the cerebrospinal fluid in the Wassermann test is purely monosymptomatic. The positive or negative results must always be interpreted in association with other laboratory and clinical tests. As Nonne has well said, the Wassermann reaction is only a symptom. Like other symptoms in a syndrome it may or may not be present without affecting the validity of the syndrome from its diagnostic aspects.

3. *Cytological Examination of the Cerebrospinal Fluid.*—The technic of puncture cannot be entered into here, but one point should be borne in mind: the procedure is not always without danger. It should be done with care, the fluid being withdrawn very slowly, dropwise in some cases, and the patient should rest in bed several hours, preferably both before and after the operation. One of the functions of the cerebrospinal fluid is to maintain an equilibrium in the intracerebral pressure, and any sudden alteration, such as is produced by the withdrawal of 5, 10 or 15 c.c. of the fluid, is apt to disturb such equilibrium. Headache, nausea, vomiting, dizziness, are among the unpleasant effects in those who react badly. Such are few, but they exist, and care is imperative. Some are helped by lying quiet, with foot of bed elevated and with aspirin, 10 grains, repeated if necessary. In patients in whom brain tumor is suspected special care should be taken, as here a few cases of sudden death have been reported following lumbar puncture with withdrawal of fluid.

In pathological cerebrospinal fluid an increase in lymphocytes is the most striking feature. Over 5 to 10 lymphocytes to the cubic millimeter indicates pathological fluid. Other authors maintain even lower averages to be pathological (Petrén *et al.*). Other cells may also be found, such as polymorphonuclear leukocytes, plasma cells and occasionally eosinophile and endothelial cells. Red blood cells usually come from the wound of the puncture.

The fluid is best fixed and imbedded, and then stained by the Alzheimer method. This is the most complete and satisfactory method thus far devised, as it permits of the counting and study of all the cells. The Fuchs-Rosenthal counting chamber, however, is that used in the greater number of instances. Its results are more quickly obtained, although they lack the finality of the Alzheimer method.

A normal cerebrospinal fluid is a clear liquid. It has a specific gravity of 1006, a slightly alkaline reaction, and is almost free from cellular constituents—1 to 5 lymphocytes per cubic millimeter, as estimated by the Fuchs-Rosenthal method, may be considered normal.[3]

[1] See Kaplan: Serology in Nervous Syphilis, Philadelphia, 1915. Kraepelin: Lehrbuch der Psychiatrie, 9th ed., 1927, vol. **2**.

[2] Compare study of Fildes *et al.*, loc. cit. Cornaz: Jour. Nerv. and Ment. Dis., 1919. Head and Fearnsides: Brain, vol. **36**. Also consult Highman: Arch. Dermat. and Syphilol., February, 1920, p. 194.

[3] See Thomsen, Hill, Halliburton (Proc. Roy. Soc., vol. **64**) for normal fluid. See volume of Research Association of Nervous and Mental Disease, Cerebrospinal Fluid, 1924. Schottmüller, Plaut and Rehm. Kafka. See literature in Nonne, 5th ed.

Changes in Cerebrospinal Fluid.—In cerebrospinal syphilis, in tabes, and in general paresis there is usually an increase in the number of lymphocytes. They may run up into the hundreds, especially in some cases of acute meningomyelitis; also in some fulminating cases of paresis. In fact, most meningitic processes are accompanied by increase in cellular content. Polynuclear cells are frequent in the acute types, sometimes tuberculous meningitis excepted, and as the process tends to chronicity there is a tendency to lymphocytosis and loss of other cell types. The pleocytosis of syphilis has usually been attributed to a meningitic process. It has been suggested as due to a periarteritis as well (Szecsi).

Certain general variations may be recorded. The earlier students were more didactic in their statements concerning specific differences in the number of cells as distinguishing cerebrospinal syphilis, tabes, and paresis. Further extension of the studies shows them to have been in part unwarranted. Thus it has been said that the lower numbers point to cerebrospinal syphilis, the higher number to tabes, and the highest to paresis. This may at times be so, but it is not an absolute rule by any means. The number of cells seems to bear a more definite relation to the *activity* of the underlying irritation or inflammatory process than to its *kind*. Thus a stationary tabes may show few cells, also a paresis in remission, whereas an acute cerebrospinal syphilis or an acute meningomyelitis may show many cells.

A fluid rich in polymorphous cells is indicative of a very active process, syphilitic or otherwise.

In neurosyphilis in general, in tabes, and paresis in particular, it is of importance to note that a pleocytosis, oftentimes of very marked grade, may antedate all neurological symptoms of the after-coming disorder. This has been shown repeatedly by Sicard, Ravaut, Fildes, Cornaz and many others. This preparetic period has been diagnosticated as long as two years before the onset of the disease. This point is well to bear in mind when one's advice is asked as to the advisability of marriage of syphilitics in the forties. It is not at all an infrequent experience to find the outbreak of paresis occurring in men of from forty to forty-five, who have been from one to three years married, and many have waited this long, feeling that because of an early syphilis it were wiser to defer marriage until a safe period. A return will be made to the prophylactic features later in the more detailed discussion of the therapy.

How soon after infection by syphilis may lymphocytosis appear in the cerebrospinal fluid? Varying answers are available, but Ravaut has reported its presence at least two months after infection. In one patient of Fildes a positive Wassermann, 1000 cells and the spirochete were found in the cerebrospinal fluid twenty weeks after infection with no other symptoms of neurosyphilis. This author reports a series of 624 unselected cases of syphilis in all stages of the disease.

Eighteen per cent. of these had 10 cells or over per c.mm. and 12 per cent. had from 5 to 9 cells per c.mm. In 80 per cent. of individuals

who had abnormal cerebrospinal fluid there was no clinical sign nor symptom of nervous disease, although 29 patients had over 100 cells per c.mm., 9 had over 300 cells per c.mm., and 3 had 1000 cells or over per c.mm. Many of the other 20 per cent. had symptoms so slight that they were of uncertain value in pointing one to neurosyphilis.

An interesting study by Cornaz[1] is worth quoting at length in this connection. The figures are based on an investigation of about 354 cases. Cornaz accepts 9 lymphocytes per c.mm. as the upper limit of the normal number.

1. In *primary syphilis,* hard chancres with no secondary manifestations, there were 76, all punctured before beginning treatment. In 24 patients the puncture was made at a time when the Wassermann reaction in the blood was still negative. The average lymphocytosis in these cases was only 2.4; it was increased only twice. Of the 52 punctures made when the blood Wassermann was already positive, the average lymphocytosis was 6.1 it was increased in 35 per cent. of the cases. Pleocytosis rising to 34 was found once.

Wassermann reaction in the fluid was tested for 59 times; 58 times it was negative and only once positive with a patient whose blood was equally positive and who presented a lymphocytosis of 9.

The albumin, precipitated by nitric acid and measured approximately by means of the scale of Bloch, remained generally within normal limits, 0.2 or 0.3 per thousand. It was plainly increased (0.7 per cent.) in only two instances. In one of these cases the lymphocytosis was 1 and in the other 8; there was therefore no parallelism between content of albumin in the fluid and the number of lymphocytes. Nonne's reaction, disclosing the presence of globulins, is, so to speak, always negative at the beginning of the luetic infection.

2. *Roseola* (with or without persistance of chancre).—84 cases. These were also patients punctured before treatment. The Wassermann was positive in the blood every time. The average lymphocytosis was 8.2 per c.mm.; it was increased in 40 per cent. of the cases. The Wassermann reaction was tested for in the fluid 71 times; 10 times it was positive. For these 10 cases the average lymphocytosis was 17.1. In the 61 fluids in which the Wassermann reaction was negative the lymphocytosis had an average of only 5.6. There exists a parallelism between the two phenomena in the sense that, when the reaction of fixation of complement is positive in a fluid, that fluid presents most often a lymphocytosis clearly augmented. The greatest pleocytosis was 67, observed in a fluid in which the Wassermann was positive. The albumin was increased in 45 per cent. of the cases. As a rule, if the albumin is abundant there is also augmentation in the number of lymphocytes. The Wassermann in the fluid may be positive with a normal quantity of albumin.

3. *Mucous plaques* (without exanthematous lesions).—Twenty-five patients punctured. They all presented only mucous plaques and not

[1] Cornaz, G.: Study of Cerebrospinal Fluid in Different Periods of Syphilis, Jour. Nerv. and Ment. Dis., April, 1919.

large condylomata nor remains of roseola. The blood Wassermann was positive 24 times (96 per cent.). The lymphocytosis was increased 8 times (32 per cent. of the fluids); this was an average of 5, therefore inferior to the degree attained in the roseola. The fluid Wassermann was positive only once (4 per cent.); in this case the lymphocytosis was 4. Increase of albumin was observed in 30 per cent.

4. *Large Condylomata.*—Thirty patients presented neither roseola nor mucous plaques. Blood Wassermann was positive in 100 per cent. of the cases. Lymphocytosis was increased in 27 per cent. of the cases with an average of 5.1. Fluid Wassermann was positive 4 times (13 per cent.). In 1 instance a negative Wassermann had become positive when a second puncture was made after a complete treatment with neosalvarsan. The albumin content averaged 0.5 per cent., increased in 30 per cent. of the cases.

5. *Latent Syphilis.*—This treats of long-standing syphilis, 65 cases, already treated previously or of patients in whom the lues was incidentally discovered without their presenting other signs of neurosyphilis. In those of them in whom the blood Wassermann was positive (70 per cent.), the lymphocytosis, increased in 40 per cent. of the cases, showed an average of 8; for the patients in whom the blood Wassermann was negative the average was only 4.7 (increase in 30 per cent. of the cases). Fluid Wassermann was positive 5 times in 64 examinations (7.8 per cent.). In 1 instance we found the Wassermann negative in the blood and positive in the fluid. This was in a longstanding syphilitic treated on several occasions. The albumin, increased in 35 per cent. of the cases showed an average of 0.6 per cent.

6. *Hereditary Lues.*—Only 6 observations. One small patient presented hydrocephalus with idiocy. His lymphocytosis was 67. If this special case be excluded there was for the 5 others a normal lymphocytosis averaging 3.9. The blood Wassermann was positive in 100 per cent. of the cases, the fluid negative 6 times at the first puncture. In one instance it had become positive at a second puncture made after treatment the lymphocytosis remaining normal.

7. *Tertiary Syphilis* (gummata and ulcerations).—Forty-five patients were punctured. The blood Wassermann was positive for 77 per cent. of the cases. Lymphocytosis, increased in 37 per cent. of the cases, averaged 7.1. It was evidently the same whether the blood Wassermann was positive or negative. The fluid Wassermann was positive 3 times (6.6 per cent.). Here also he found in 1 instance the Wassermann reaction positive in the fluid and negative in the blood. Increase in albumin was noted in 40 per cent. of the cases; it is most often parallel with the pleocytosis.

8. *Cerebral Syphilis and Meningomyelitis.*—Six cases. Blood Wassermann was positive 4 times (66 per cent.). There was marked pleocytosis in 100 per cent. of the cases. This averaged 51 lymphocytes per c.mm., 1 case presenting an exceptional pleocytosis of 235, the 5 others giving an average of 15 lymphocytes. The fluid Wassermann was positive every time, even in the 2 cases in which

the blood reaction was negative. The quantity of albumin surpassed 60. per cent. only in the patient noted above who presented a lymphocytosis of 235; it reached in this case 1.5 per cent.

9. *Tabes.*—Punctured patients 13. The blood Wassermann was positive 9 times (69 per cent.). The average lymphocytosis, which was 30, was increased in 11 instances (85 per cent.). It was still normal with the two patients presenting clear symptoms of tabes at the outset. Fluid Wassermann was positive 9 times at the beginning, but after treatment by neosalvarsan or novarsenobenzol it became positive in the 13 patients (100 per cent.). Once, in a case of demonstrated tabes, the fluid Wassermann had become positive after treatment while remaining completely negative in the blood. The average quantity of albumin is 0.6 per cent. Here its increase is generally proportional to the lymphocytosis.

10. *General Paresis.*—Only 4 cases are reported.

The Wassermann reaction was positive all 4 times in the blood and the fluid (100 per cent.). The average lymphocytosis was 28, it was increased in every case. The quantity of albumin, 0.8 per cent. on the average was in each instance equally augmented. It attained a maximum of 1.5 per cent. in a case where the lymphocytosis was 12. On the other hand in another patient it was only 0.8 per cent. for a lymphocytosis of 51, which shows that the parallelism between the quantity of albumin and the number of lymphocytes is not by any means constant.

The results may be tabularized as follows:

	Average Lymphocytosis.	Lymphocytosis increased in, per cent.	Wassermann fluid. positive, per cent.	Blood Wassermann, positive, per cent
Hard chancre (Wa.+)	6.1	35	1.6	68
Hard chancre (Wa.−)	2.4	8	0.0	
Roseola	8.2	40	14.0	100
Mucous plaques	5.0	32	4.0	96
Large condylomata	5.1	27	13.0	100
Latent lues (Wa.+)	8.0	40	8.8	70
Latent lues (Wa.−)	4.7	30	5.0	
Hereditary lues	3.9	0	5.0 16.6 after treatment	100
Tertiary lues	7.1	37	6.6	77
Meningeal lues	51.0	100	100.0	66
Tabes	30.1	85	69 after neo. 100.0	69
General paresis	28.0	100	100.0	100

His observations permit him to confirm fully that which Dind[1] pointed out in 1914, that is, how frequent and early is the extension of the syphilitic infection into the meninges, since in 35 per cent. of the cases of hard chancre the number of leukocytes was already increased at a time when the cutaneous or mucous manifestations

[1] Revue d'Urologie, 1914.

were still totally absent or at least could not be demonstrated by the means of investigation. It is the lymphocytosis which at first betrays the modifications in the fluid, when the quantity of albumin is still normal and when the generalization of the infection has not yet been demonstrated by the Wassermann reaction. In the exanthematous stage 14 per cent. of the fluids gave a complement fixation. This figure is met with again much the same (13 per cent.) in the cases of large condylomata when, among the patients showing mucous plaques, the fluid Wassermann was positive only once in 25 cases. This curious difference is not easily explained. Are there anatomico-histological conditions favoring the passage in the fluid of bodies capable of becoming fixed in the antigen especially among the carriers of the condylomata or indeed do the condylomata result in a more abundant formation of these bodies? The modifications of the fluid seem at least to be in accord with the reactions of the skin and it may be asked if the phenomenon of allergie is not present. This conclusion would support the idea which Dind has been one of the first to defend, that allergie is an essentially cutaneous phenomenon. The condylomata, cutaneous lesions, would provoke a violent reaction of the organism when the lesions of the mucous membrane alone would not excite the individual to defense in the same degree.

As soon as the syphilis localizes itself in the central nervous system, the cerebrospinal fluid constantly presents important modifications and it is of great value in investigation for fixing the diagnosis or for controlling the course of treatment. The Wassermann reaction should be specially made in these cases for it is relatively frequent that it is positive in the fluid and negative in the blood. The presence of lymphocytes almost invariably speaks for the beginning of neurosyphilis.

With active therapy the pleocytosis is apt to disappear. Are those patients who retain their lymphocytosis on the way to tabes or paresis? This is an important problem which has been answered yes and no. Its definite answer is yet to come, as the necessary time for determining the full significance of the results of cytological study of the fluid has not yet elapsed.

From the therapeutic point of view then the whole subject of pleocytosis of the cerebrospinal fluid is full of significance, and in need of constant attention. In this connection it should again be emphasized that lymphocytosis alone does not mean syphilis alone. It can be said that absence of lymphocytes at least negatives tabes and paresis—to this generalization there are but few recorded negative observations.[1]

Lymphocytosis is not limited to neurosyphilis, however. It is marked in sleeping sickness, an allied disease (Spielmeyer, Mott). It is often also high in tuberculous meningitis, but is here usually complicated by the presence of polymorphonuclear leukocytes. In a few cases of multiple sclerosis lymphocytes have been found. In the

[1] Klieneberger: Arch. f. Psychiat., 1911. Foster, Lewandowsky: Handbuch der Neurologie.

acute stages of poliomyelitis lymphocytes may be found, in epidemic encephalitis, also in epidemic cerebrospinal meningitis and in herpes zoster.[1]

4. *Chemical Examination.*—Here the presence of a reducing agent (Fehling's) and of globulin is to be estimated. Most fluids contain the former. When present in large quantities it may point to a tuberculous meningitic process.

Increased globulin content is a characteristic feature of paresis. In tabes increased globulin is the rule, as is also the case in cerebrospinal syphilis, but to a less extent. Markedly increased globulin content is not infrequent in spinal cord tumors, gummatous or non-syphilitic. Increased globulin is also a feature of the acute stage of thrombotic softening of arteriosclerosis when the softened area touches the meningeal sac. The globulin reaction is apt to run along with the lymphocytosis. It has no apparent alliances with the findings of the Wassermann test.

Summary of Laboratory Findings.—Four Reactions.[2]—Before passing to the diagnostic significance of other methods of examination in neurosyphilis a word should be said relative to the value of the "four reactions," as Nonne has called them. It has been said that taken alone they may mean nothing positive, so far as a differential of the different trends in neurosyphilis is concerned, but when read together they afford important guides to diagnosis, to localization and to treatment.

Expressed schematically, which schemes, as he well remarks, are not products of Nature but of man, the following results of the four reactions are to be expected. (I) Blood and (II) fluid.

I. Blood examination.

Wassermann reaction.

(*a*) Positive. Is characteristic of syphilis with few exceptions (already noted). A positive Wassermann of the blood serum says nothing further than that the individual has come in some manner in contact with syphilis, either through heredity or by infection. It does not say that the disease from which he suffers is due to syphilis.

(*b*) Negative. Is differentially diagnostic, against paresis, since it is only very rarely that the blood in paresis gives a negative reaction.

II. Study of cerebrospinal fluid.

(*a*) Normal fluid. Pressure 90 to 130 mm. water. Phase I, reaction negative, Weichbrodt reaction negative and mastic reaction negative. Globulin reaction negative—not over 3 or 5 cells to c.mm. (Fuchs-Rosenthal.) Wassermann (0.2 cc. fluid) negative.

[1] See study by Schaller (loc. cit.) on Herpes Zoster and the Cerebrospinal Fluid.

[2] See Nonne: Syphilis und Nervensystem, 5th ed., Berlin, Karger, 1924.

(*b*) Pathological fluids.
1. Increased pressure—over 150 c.mm. water.
2. Positive Phase I. Globulin reaction.
3. Increased cell count. (These three symptoms, in coördination or alone, indicate the presence of an organic nervous disorder, syphilitic or non-syphilitic.)
4. Positive Weichbrodt.
5. Positive mastic.

(*c*) If the disease of the nervous system is syphilitic, then the Wassermann test of the cerebrospinal fluid will show. If the Wassermann reaction (original method—0.2 cc. of the fluid) is positive, there is great probability that the patient is a paretic, or a taboparetic, much less often a cerebrospinal syphilitic, or a pure tabetic. In nearly all cases of cerebrospinal syphilis and of tabes the Wassermann reaction becomes positive by using 0.4 to 1 cc. of fluid.

Nonne's typical findings are as follows:

I. Paresis or taboparesis.
1. Wassermann reaction in blood positive (80 to 100 per cent.). Pressure increased.
2. Phase I, globulin reaction positive (95 to 100 per cent.).
3. Weichbrodt +.
4. Mastic. Paresis curve.
5. Lymphocytosis (95 per cent.).
6. Wassermann in cerebrospinal fluid.
(*a*) Positive—about 85 to 90 per cent. with original method and 0.2 cc. cerebrospinal fluid.
(*b*) Positive in 100 per cent., with larger quantities of cerebrospinal fluid.

II. Tabes without paresis.
1. Wassermann reaction in blood positive (60 to 70 per cent.). Pressure usually increased.
2. Phase I, reaction globulin and positive (90 to 95 per cent.).
3. Lymphocytosis positive (90 per cent.).
4. Weichbrodt +.
5. Mastic. Tabes curve.
6. Wassermann in fluid.
(*a*) Original method, 0.2 cc. positive (20 per cent.).
(*b*) Larger quantities (90+ per cent.).

III. Cerebrospinal syphilis.
1. Wassermann reaction in blood positive (70 to 80 per cent.). Pressure frequently increased.
2. Phase I, reaction usually positive, exceptionally negative.
3. Lymphocytosis nearly always positive.
4. Weichbrodt +. Greater than Phase I in meningeal predominance.

5. Mastic. Syphilitic (meningeal) curve.
6. Wassermann in fluid.
 (*a*) Original methods (0.2 cc.) positive in about 20 to 30 per cent.
 (*b*) Larger quantities nearly always positive (of value in diagnosis of multiple sclerosis, cerebral and spinal tumor).

These results of Nonne's summarize fairly accurately the present-day attitude on the value of the four reactions. The full significance of the findings can be gained only by reference to the originals. This field of work is rapidly expanding, and that which now appears on the frontiers of our knowledge will undoubtedly be much modified by the rapidly advancing army of investigators.[1] As Nonne well remarks, Nature does not schematize, man does that, hence all schemata are to be regarded as suggestive only. All laboratory tests, again it must be emphasized, are but a small part of the clinical picture and must not get out of proportion in the complete analysis of the clinical details.

Additional Tests.—These additional tests are valuable because syphilis may be present with a negative Wassermann. These cases of so-called latent syphilis with negative Wassermann occur in about 35 per cent. of cases with no active signs of the disease but with a clear history of infection.

The Luetin Test.—This is a cutaneous test with a suspension of killed *spirochete* cultures as prepared by Noguchi, known as luetin. A positive reaction appears in the form of a red papule with indurated areola in five or six days. This test is especially valuable in tertiary and latent syphilis, conditions in which the Wassermann reaction is sometimes negative. It is valuable also in differentiating from other conditions which might give a positive Wassermann, for this test appears to be a specific for syphilis.[2]

Provocative Wassermann.—This test, like the luetin test, is valuable where syphilis is suspected, but the Wassermann is negative. It is dependent upon the fact that a negative Wassermann may be changed to positive after an injection of salvarsan. This change may occur quite promptly or only after several days. Nichols recommends making the Wassermann twenty-four and forty-eight hours after an injection (0.4 gram salvarsan or 0.6 gram neosalvarsan) and again after seven and fourteen days.

Lange's Colloidal-gold Test.—This test is dependent upon a color reaction which makes it very delicate. It has an added value because a single test, depending upon the degree of discoloration, tends to show whether one is dealing with a frank luetic or a metaluetic process. For this reason it is to some extent replacing the Wassermann test. A typical reaction in the luetic zone could be 3321122200, a typical reaction in the paretic (metaluetic) zone would be 5555432000. For a description of the technic the student is referred to special works.

[1] See Plaut's outstanding monograph on the Wassermann Reaction in Neurosyphilis.
[2] See later studies by Fleischmann (1921), Kaffka, Nonne (loc. cit.).

The Sachs-Georgi reaction parallels the Wassermann findings, at times being more sensitive. It is a valuable addition. Of the many modifications, that of Dreyer[1] is valuable in that it has quantitative possibilities according to its discoverer.

Guillain[2] has developed a number of useful serobiological technics with the use of mastic (Emanuel, 1916), benzoin and related substances. They have certain very practical points of advantage and have proved to be as reliable as the older methods.

Kahn's precipitation test, Kolmer's improved Wassermann test, Meinicke's precipitation test, Ruediger-Wassermann tests, Harris test, Boltz paresis tests—these are among the newer developments which must be consulted in the original and in the copious literature.[3]

5. *The Eye Reflexes.*—In the diagnosis of syphilis of the nervous system the neurological examination of the eye reflexes is of paramount value. Here irregularity in the size of the pupils, irregularity in the pupillary margins, the impairment of the consensual light reflex, the slowing in reaction to light, fatigability of the light reflex, alterations in response to accommodation efforts, the full development of the Argyll-Robertson syndrome, are all to be considered. These, one or all, constitute extremely delicate and valuable criteria for the clinical appraisement of neurosyphilis.

A fully developed Argyll-Robertson syndrome—loss of direct pupillary light reflex, with free and ample response to accommodation reflexes in one or both eyes—is indicative of neurosyphilis. There are many who believe that the finding of this syndrome affords positive proof of nervous syphilis. This is not so. Clinically the Argyll-Robertson syndrome has been observed following direct injury of the midbrain structures (pistol shot—Guillain and a group of cases seen in war practice), it has been observed in poisonings other than those of syphilis, alcohol (in Korsakoff, Wernicke's polioencephalitis superior). It may result from pressures (tumors of third ventricle, pineal), from poliomyelitis, from epidemic encephalitis, multiple sclerosis, diphtheria[4], diabetes from trypanosomiasis, from orbital trauma (Velter, Ohm), and from a few other rare disorders.

As a practical issue, however, the complicated anatomical combinations which make an Argyll-Robertson syndrome possible are chiefly present in but one type of poisoning, *i. e.*, syphilitic. Hence it is safer to assume syphilis on finding the Argyll-Robertson pupil and then check it up later, than to assume something else.

Testing for the Argyll-Robertson syndrome, however, is not as simple as it is usually supposed. The ordinary devices of having a patient face the window, and then cover and uncover the opened eyes with

[1] British Med. Jour., 1921, i, 859.

[2] Laroche and Lechelle: La reaction du benjoin colloidal, Masson, 1922. See Nonne (loc. cit.) as to value of mastic reaction also in his study for the Unna Festschrift (Dermat. Studien, No. 22). Kahn: Serum Diagnosis by Precipitation, Baltimore, 1925. Sachs: Osler and McCrae: Modern Medicine, 1927.

[3] See Jour. Syphilis for full discussion of all these tests.

[4] Kellner, F.: Deutsch. f. Ztschr. Nervenh., vol. **74.**

the hand; focussing the eye upon a distant object, and then upon the finger in close proximity to the nose; these tests for the most part are entirely too crude to permit one to judge with certainty concerning the Argyll-Robertson syndrome. Such methods may suffice for the majority of instances, but in no field of neurology is it more desirable to utilize the most accurate methods than in dealing with the vexed question of syphilis of the nervous system.

Few clinicians can follow out the intricacies of Weiler's complicated methods, but they may be necessary in certain doubtful cases. Repeated examinations, under carefully regulated supervision, in the daylight, and in the dark-room, are therefore desirable when testing for anomalies in the pupillary reflexes. Careful checking of the results obtained by the small pocket electric lamps is imperative, as occasionally they give anomalous results and may lead to serious error (Oppenheim).

The presence of a fully developed Argyll-Robertson syndrome may be said to be a highly probable positive proof of neurosyphilis, particularly of the cerebral and midbrain neurons. Its absence, however, by no means negative syphilis of the nervous system, since cerebral gummas, cerebral syphilis, paresis, tabes, syphilitic meningitis, meningomyelitis, cord gummas, syphilitic radiculitis, syphilitic neuritis, all may be present without any anomalies in the pupillary reflexes.

It has already been pointed out that the serobiological changes of paresis and of tabes may antedate the clinical neuropsychiatric signs by at least a couple of years, and, moreover, it has been emphasized that pathological alterations in these pupillary reflexes are after all only chance happenings, when certain reflex arcs in certain combinations are caught in the mesh of the infiltrative, syphilitic alterations. The chance is a large one, it is true, but still it is purely a statistical matter of what has happened. One word may be added: in many cases of cerebrospinal syphilis one can gauge the progressive amelioration of the patient's condition by the gradual return of the anomalous pupillary reflexes to a more average condition. Thus, an absolute Argyll-Robertson syndrome may become a relative one. A unilateral Argyll-Robertson may be lost; slowly reacting pupils may show prompt reactions; irregularities in size may disappear; rapid fatigability may let up; a consensual light loss, often the first anomaly to appear in cerebrospinal syphilis will clear up; irregularities in the pupillary outlines will make way to regular outlines, etc.

An inability to modify pathological pupillary reactions by ample syphilitic therapy argues in part for the chronicity of the process, or the inefficacy of treatment. This is not an absolute rule, however. It may be possible for a syphilitic process to permanently destroy portions of the pupillary reflex paths, and then be completely and permanently arrested. The pupillary reflexes, however, remain impaired.

In relation to this question of the pupillary reflexes and anti-syphilitic treatment the problem arises—What is the probable outcome of a syphilitic process which comparatively early in its course has destroyed

the pupillary reflex paths? Can it be decided, say after two, five, or ten years, during which time there has existed an Argyll-Robertson syndrome and little else, that the disease has been completely and permanently arrested?

Since the rapid extension of knowledge concerning the cerebrospinal fluid this question can be answered better at the present time than ever before. If the signs of a meningitis—acute, subacute, or chronic, *i. e.*, increase in cellular elements, increase in globulin, and positive fluid Wassermann, remain absent, then it can be taken as highly probable that the disease process has been arrested. Judging by clinical methods alone a longstanding and unchangeable Argyll-Robertson pupil may be the only lesion in a practically cured syphilis of the nervous system. It is, however, to be realized that such a pupillary anomaly may exist as long as from twelve to sixteen years (alone) and then the patient may develop a paresis or tabes, and occasionally it is found as an apparent personal peculiarity without other significance. Of about 50 personal observations, now followed twenty years, only 5 patients with long-standing Argyll-Robertson pupil have not developed further signs of brain syphilis. Certain deep-seated, chronic, syphilitic arterial processes, which may lead to focalized lesions, hemiplegia, aphasia, etc., may, however, go on for some time without distinct signs of meningeal irritation with the characteristic cellular reactions.

Clinical Trends.—It has already been stated that the so-called classical forms of neurosyphilis are largely abstractions. The pathological processes are predominantly either meningeal, arterial or infiltrative in character, meningo-vascular syphilis, or parenchymatous alone or in combinations, and the clinical manifestations are extremely variable, complex and confusing, depending upon the interactions of the pathological trends and the localization of the anatomical paths interfered with.[1]

Fortunately for the therapy, the clinical type is of secondary consideration, yet there are certain therapeutic variables that render it desirable that a fuller analysis of clinical forms should be made than would at first sight seem advantageous. For instance, it may be recalled that certain patients with meningeal infiltrations of the base, with or without gummatous nodules, either of the base or of the convexity, at times may be indistinguishable clinically from those suffering from a parenchymatous or central type of neurosyphilis. A pessimistic therapeutic attitude relative to the latter process would therefore work greatly to the disadvantage of a patient with the former. This is one of the gravest reflections brought against a laissez faire handling of the so-called "hopeless paretic" in institutional or in private work. Many curable cases are grossly neglected because they are diagnosed as "incurable." Neurosyphilis meningo-vascularis is usually very amenable to treatment. Often its clinical picture is that of a paresis.

[1] Spielmeyer: Histopathologie des Nervensystems, 1922, vol. 1. Nonne: Loc. cit., 5th ed., 1924. Kraepelin: Loc. cit., 9th ed., 1927.

The following clinical trends will be discussed here.

1. Syphilis of the cranial bones.
2. Syphilis of the basal meninges.
3. Syphilis of the convexity; neurosyphilitic epilepsies.
4. Cerebral syphilis—arterial types.
5. Cerebral syphilis—parenchymatous or central types (general paresis, taboparesis).
6. Syphilitic psychoses, acute and subacute.
7. Tabes.
8. Syphilitic spinal meningitis; meningomyelitis; myelitis; syphilitic radiculoneuritis, and related neurosyphilitic syndromes.
9. Hereditary neurosyphilis.

For a fuller consideration of the protean variations the monographs of Rümpf, Nonne, Mott, Plaut, Oppenheim, Forster, Schaffer, Fournier Southard and Solomon, Gennerich, Tony Schmidt Kraepelin, should be consulted.[1]

1. **Syphilis of the Cranial Bones Causing Nervous Symptoms.**[2]—These were recognized as early as the end of the sixteenth century. Syphilis of the cranial bones shows itself practically in the form of gummata. Caries of the cranial bones alone, while known, rarely gives rise to nervous symptoms, headache excepted. These gummata may be circumscribed, in which case, if large enough, they give rise to the symptoms of a tumor of the brain, which signs are largely determined by the precise location of the gummata. These circumscribed gummata may attain enormous proportions. A personal observation (J.) recalls a gumma of the left frontal region (the size of a tennis ball) which originated in the bone dura and protruded into the right frontal lobe. Similar gummata are not infrequent.

Cranial bone gummata—usually involving the dura as well—are more frequently flattened and spreading. Here the symptoms of brain tumor are usually present. Headache, nausea, vomiting, sleeplessness, are among the general symptoms, while localizing signs in great variety, depending upon the situation of the gummatous masses are present. Epileptiform convulsions, lasting for years, may be the sole signs of such gummatous formations, occupying or due to pressure upon the motor areas. Such patients are often mistakenly treated as epileptics, and the monographs of Mott, Oppenheim, Rümpf, Nonne, in recent years, are replete with autopsy records of such cases. Monoplegias of various sorts result from such, as also aphasias, word-blindness, and various cranial nerve palsies.

In addition various forms of periostitis and of osteitis occur. This latter may lead to exostoses or to osteoporosis. In connection with syphilis of the vertebræ[3] it should be remembered that the vertebral

[1] The periodical literature is available in the chief neurological journals, Jour. Nervous and Mental Disease, New York; Revue Neurologique, Paris; Rivista di Patologia Nervose e mentale, Florence; Zeit. f. d. ges. Neurol. u. Psychiatrie; Referat Antheil Berlin. Am. Archives of Neurology and Psychiatry, Chicago.

[2] Karpas and Gregory: Jour. Nerv. and Ment. Dis., 1913, **40**, 651.

[3] Hunt, J. R.: Syphilis of the Vertebral Column, Am. Jour. Med. Sci., August, 1914.

processes are more often involved whereas tuberculosis more frequently involves the vertebral body. Syphilis more often affects the cervical, while tuberculosis more often the dorsal vertebræ.

Rarer cases of cranial bone caries of the base (sphenoid) complicated often by caries of the upper vertebræ, are also known. Petrén has studied these in detail, and has shown the value of roentgen-ray examinations in their diagnosis.[1]

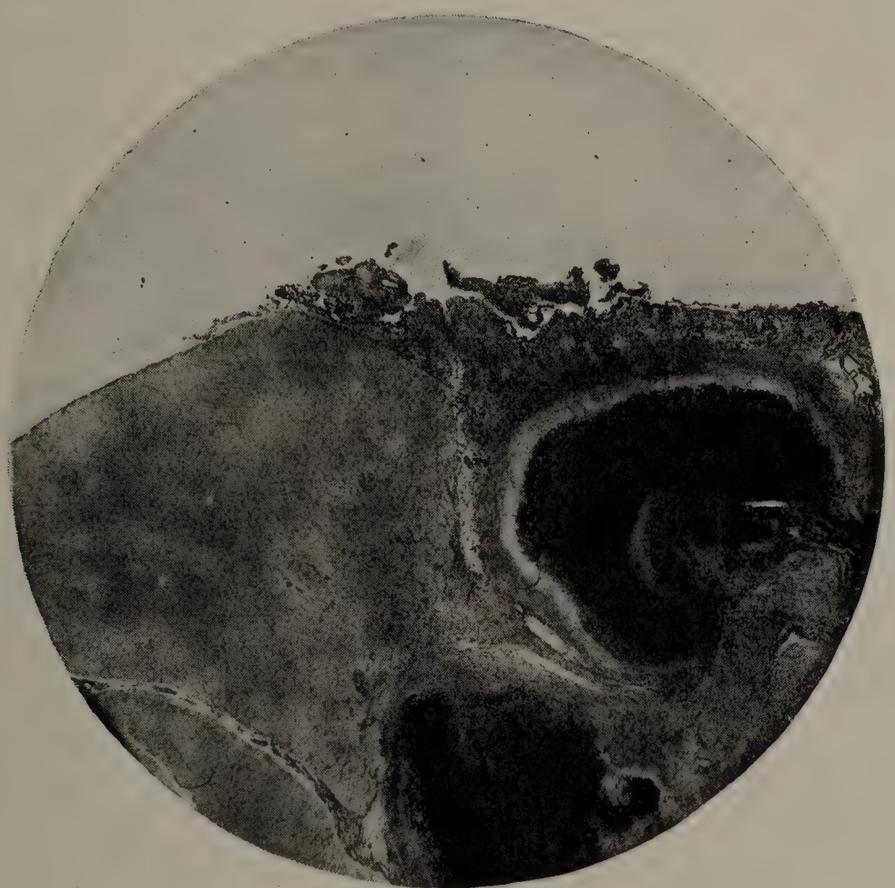


Fig. 385.—Gumma of brain.

2. **Syphilitic Meningitis of the Base.**—This is perhaps the most common trend in neurosyphilis. A widespread syndromy may develop within three months after infection, but the majority of cases are of slower evolution, one to five years. In some the symptoms have appeared as late as twenty-five years after infection. Its most frequent site, at the beginning, is in and about the interpeduncular space, thus almost invariably involving the optic chiasm. From here it tends to spread in all directions, pressing into the sulci, thickening the meninges, by infiltration, by arterial disease, or by gummatous growth. Usually all three types of pathological alteration are found. The gummatous masses not infrequently invade the brain structures as well, grow about the emerging or entering cranial nerves, and even involve the bones of the skull, and the upper cervical vertebræ. Thus, spinal meningeal infiltrations almost invariably accompany this basal syphilitic meningitis. Whereas, the diffuse, conglomerate types are more frequent, isolated vascular disease, circumscribed gummata, or

[1] Petrén: Mitt. a. d. Grenzgeb. d. med. u. Chir., 1910, **21**, Heft. 5.

other simpler manifestations of the disease may occur, in which latter case the syndromes are apt to be less complicated.

The clinical course of the more frequent types of basal syphilitic meningitis often resembles that of general paresis, especially in the beginning, but the gradual extension of the infiltrating or gummatous developments causes cranial nerve symptoms which soon permit a differential diagnosis.

Headache is a frequent and early sign, often preceding other symptoms by weeks, months, or even years. It has the nocturnal exacerbations so long described as characteristic of syphilitic headache. It is complained of variously as boring, stabbing, pressing, etc., and percussion at the base may show tenderness, though less frequently than in convexity meningitis. The cervical vertebral complications spoken of often result in stiffness of the neck.

An early implication of the optic nerve is to be expected. It shows itself (20 per cent. to 40 per cent. of the cases) either as a pressure neuritis (neuritic atrophy) of the nerve in one disk, then in the other, later, if headache and vomiting or other signs of intracranial pressure are prominent, choked disk in both eyes is apt to be present. Atrophic degeneration and optic neuritis are less often found. Marked diminution in visual acuity may be present without any disk evidences of disease.

The oculomotor nerves are frequently and usually irregularly involved. It is characteristic of basal syphilitic meningitis that successive branches are implicated. First one eye may show a ptosis, then perhaps an internal rectus palsy, then the other eye may show a dilated pupil, slow in its reactions to light, then ptosis develops here. Occasionally the accommodation reflex is lost. A series of cases will show a great variety of oculomotor palsies. A true Argyll-Robertson syndrome is not infrequently obtained.

Other cranial nerves become involved. Variations in the corneal reflex, in the sensibility, pain, anesthesiæ of the face, point to a trigeminal complication. A peripheral facial palsy may be presented. In some individuals the deeper-lying cranial nerves (IXth, Xth, XIth, XIIth) are caught in the syphilitic extension, with their characteristic symptoms. The cochlear and vestibular nerves are frequently involved.[1]

The mental picture is usually very striking. It is frequently that of a slowly developing apathy, or of a mental hebetude, which slowly in a few weeks, advances to coma, or unconsciousness, with periods of acute confusion, possibly violent delirium. There is a marked variability in these patients from week to week and also considerable differences in different patients. Some patients develop a sort of drunken delirium; others are heavy and stupid and apathetic; others are furiously violent.

One special feature is frequently met with. This is a rapid alteration in the mental picture, when, after a period of acute confusion or

[1] Lloyd: Jour. Nerv. and Ment. Dis., August, 1919. Kraepelin: Loc. cit., 9th ed.

deep coma, the patient becomes almost practically clear within a few hours or in a day. Such a variation may follow a period of severe convulsive seizures. Careful tests will reveal an underlying series of defects it is true, but from a lay viewpoint the patient may appear to have made a complete recovery so far as his psychosis is concerned. Without treatment, however, the patient again develops his apathy, confusion, delirium, or coma and not infrequently dies in this state. Sometimes death results by suicide as the patient develops, slowly or rapidly, a distinct depression with possibly persecutory ideas.

Thus at various times, these patients show the old-time "diagnoses" of acute confusion, dementia, mania, melancholia, paranoia, etc. This alone indicates the futility of regarding the symptom pictures which have gone by these names as *diseases,* some for hundreds of years. Happily present-day psychiatry, largely under the influence of Kraepelin's teaching, recognizes them as only the protean and kaleidoscopic picture-formation of a single disease process, neurosyphilis, with or without complications.

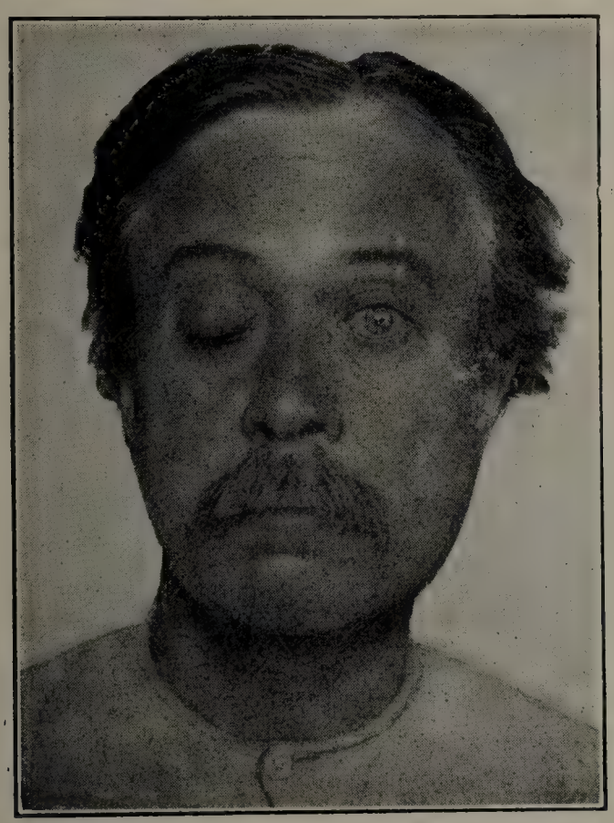


FIG. 386.—Cerebral syphilis ptosis. Third nerve palsy. (Jelliffe.)

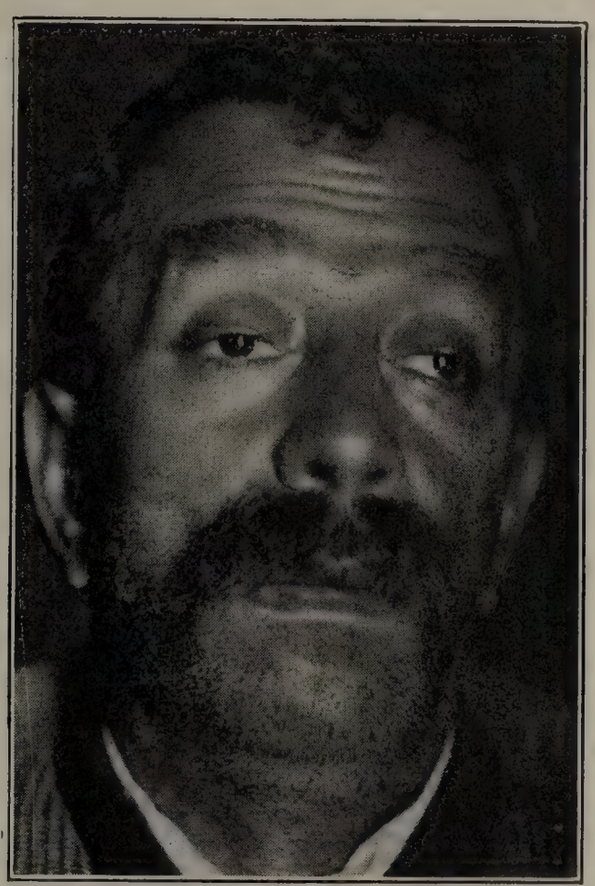


FIG. 387.—Cerebral syphilis with third and fourth nerve palsies. (Sharp.)

Biological and cytological methods have permitted this definite change in attitude, and have shown the essential and close relationship of many diverse neurological and psychotic syndromes.

3. **Syphilitic Meningitis of the Convexity.**—This differs from the former only in the trend of its symptoms. The pathological processes are practically identical. Many individuals show that the process is general, involving both the base and the convexity; in some instances

the pathological changes being more marked on the convexity than on the base.

Convexity syphilis, like the basal variety, may be a fairly localized affair, or it may be diffuse. It may be limited to the meninges, or involve the bones, or the brain, or, as is most usual, all three.

Here, headache is a prominent sign. It is paroxysmal, and often shows a nocturnal increase in severity. Percussion affords valuable evidence, as localized tenderness is very common.

The general syndrome of brain pressure is usually less emphasized. Nausea, vomiting, giddiness, may be present, but are usually late in development, or more transitory. Optic nerve changes are

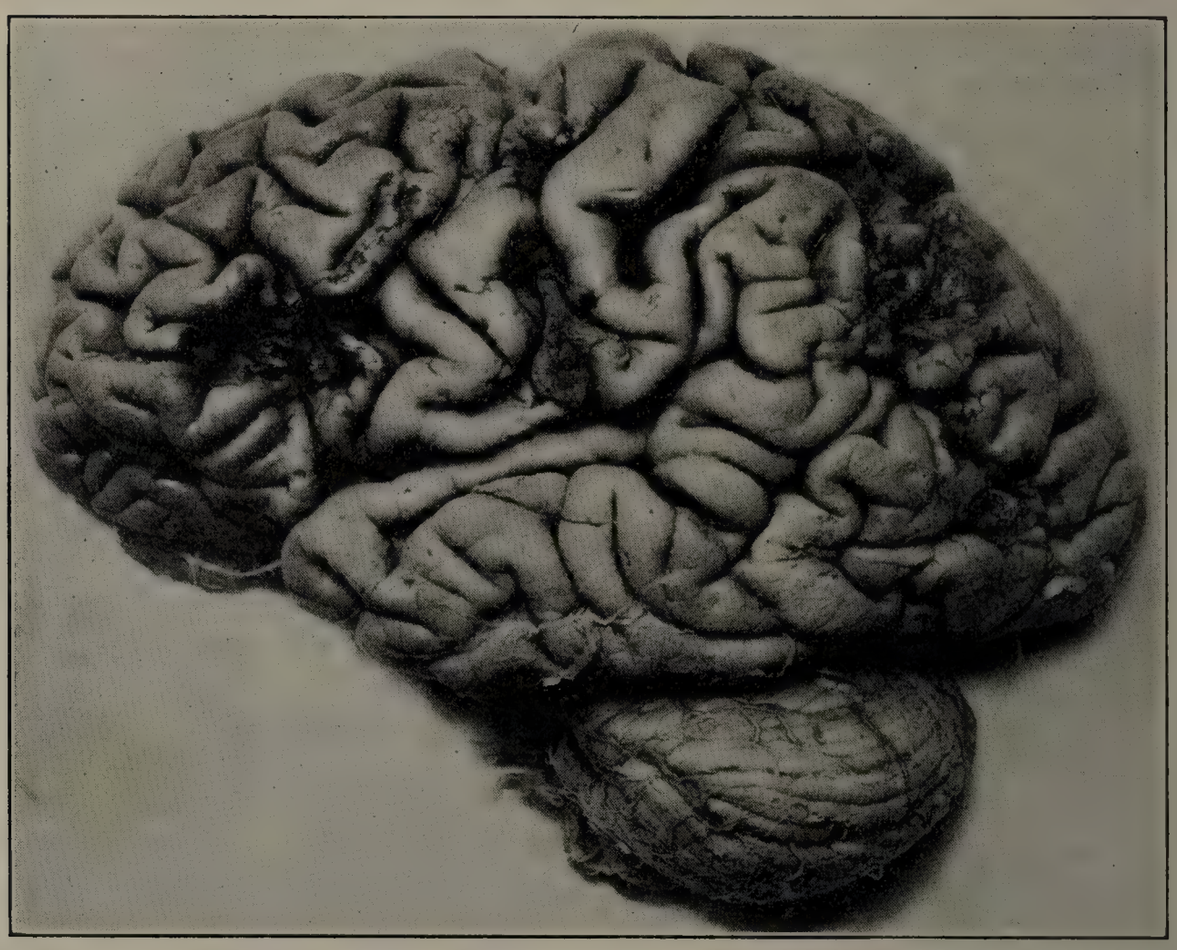


FIG. 388.—Cerebral syphilis of the convexity. Chronic epileptiform convulsions.

less frequent. Isolated symptoms are more prominent. Epileptiform convulsions indicate that the process is in or about the motor areas. Not infrequently the attacks are of the Jacksonian type. Involvement of Broca's convolutions produces temporary or more enduring motor aphasic attacks. Sometimes these aphasic attacks clear up in an hour or so, or in a few days. Minor speech difficulties indicate beginning involvement of the complicated speech pathways. Complete motor aphasia develops only, as a rule, with hemiplegic or monoplegic accompaniments. Pseudobulbar palsy attacks indicate a bilateral involvement, probably both cortical, less frequently cortical on one side, and subcortical on the opposite side or striatal.

Monoplegias of varying types are not infrequent. Bergmark[1] has devoted a large monograph to their study. Sensory disturbances, hemianesthesiæ, astereognosis, haptic hallucinations are met with.



Fig. 389.—Syphilitic meningitis with giant-cell formation.

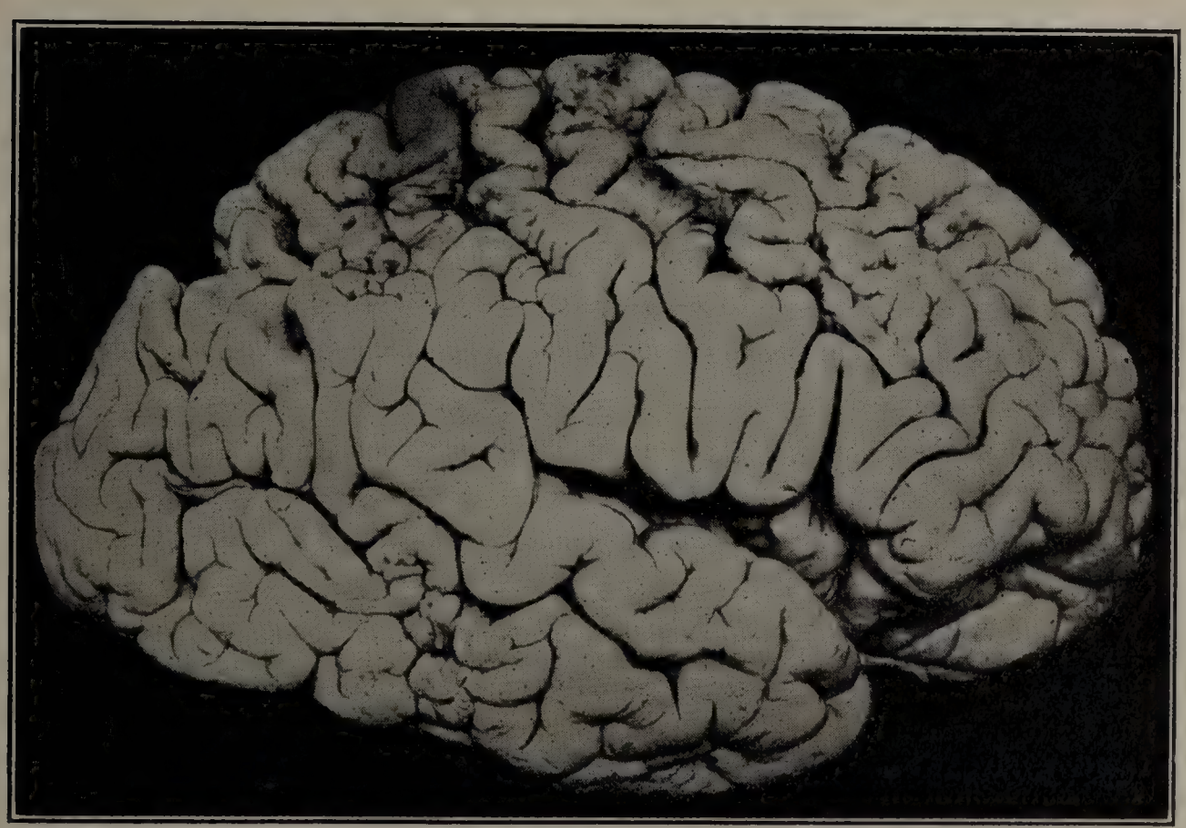


Fig. 390.—Chronic epileptiform convulsions due to syphilitic leptomeningitis and syphilitic vascular disease.

[1] Brain, 1911.

With diffuse meningo-encephalitic changes the picture of general paresis is assumed, and it is practically impossible to differentiate this trend in the process. The clinical pictures may be as various as those of paresis. Possibly the only means to distinguish them is that of a rigid application of the serological, cytological and chemical criteria. With these laboratory tests a practical insight may be gained into the more fundamental trends of the syphilitic process.[1]

4. **Cerebral Syphilis.—Vascular Types.**—In considering the symptomatology of patients who are thought to have cerebral syphilis, attention may again be called to the fact that there are no dividing lines between cerebral syphilis, basal meningeal syphilis or convexity syphilis. Rarely does one find a pure basal or convexity meningitis without some involvement of the cerebral substance on the one hand, while conversely it is as rare to find syphilitic processes strictly limited to the cerebral substance, and not involving the meninges. One can postulate pure types for purposes of description only, but disease is rarely a respector of one cerebral tissue more than another, although there may be a tendency to greater meningeal or vascular, or central predominance in one or another patient.

Symptoms.—It is for this reason that one is constantly reminded of the multiplicity of symptoms found in cerebral syphilis. In this connection it would not be without profit to glance for a moment at the diagnosis of certain cases reported by Nonne in his monograph several times alluded to. The patients were illustrations of basal, or convexity, or encephalic brain syphilis, usually combined forms. The short descriptive diagnoses run as follows: Specific headache a year after infection, with secondaries in skin and mucous membranes; headache and pupillary anomalies; headache and obstinate vomiting, with tertiary testicle signs; progressive simple dementia cured by treatment; progressive dementia with defect; combination of convexity meningitis and paresis; gummatous meningo-encephalitis of convexity with general symptoms, choked disk and antisyphilitic treatment unavailing; surgical treatment of gummata with cure; Jacksonian epilepsy; cortical epilepsy, choked disk, paresis of left leg; cortical epilepsy, optic neuritis, arterial hemiplegia; cortical hemiepilepsy and general cortical symptoms; arterial hemiapoplexy with hemiepileptic convulsions; generalized epileptic seizures; hemianesthetic attacks with cortical signs; uremia, etc. Such illustrations might be almost indefinitely continued. They are not the exceptions, they are the rule. One is tempted to indulge in the generalization that one hundred consecutive patients with the types of cerebral syphilis under consideration would show one hundred different

[1] The student should consult the periodical literature, for this branch of study is constantly evolving toward more and more pragmatic criteria. See current literature in such neurological magazines as the Revue Neurologique, Journal of Nervous and Mental Disease, Rivista di patologia nervose e mentale, Zeitschrift f. d. ges. Neur. u. Psych. Ref. and also the important Jahresbericht über die Fortschritte u. Leistung a. d. geb. d. Neurol. u. Psychiat., Berlin, Karger (discontinued during the World War) or the Zentralbl. f. d. ges. Neurol. u. Psychiat. Springer: Jahresbericht of the same publishers, Am. Jour. Syphilis.

clinical syndromes. Practically all of those just enumerated belong to the convexity types of cerebral syphilis. Their enumeration may prove of service in localizing the process.

A similar series for the basal types may be equally of service: Thus the clinical diagnoses run: Gumma of right frontal lobe; pressure neuritis of optic nerve; choked disk with general cerebral symptoms; bilateral neuritic optic atrophy; recurrent optic atrophy; hemianopsia; bitemporal hemianopsia; homonymous left-sided hemianopsia with right-sided abducens palsy: temporal hemianopsia; cranial nerve and epilepsy hemianopsia; hemianopci pupillary reaction,

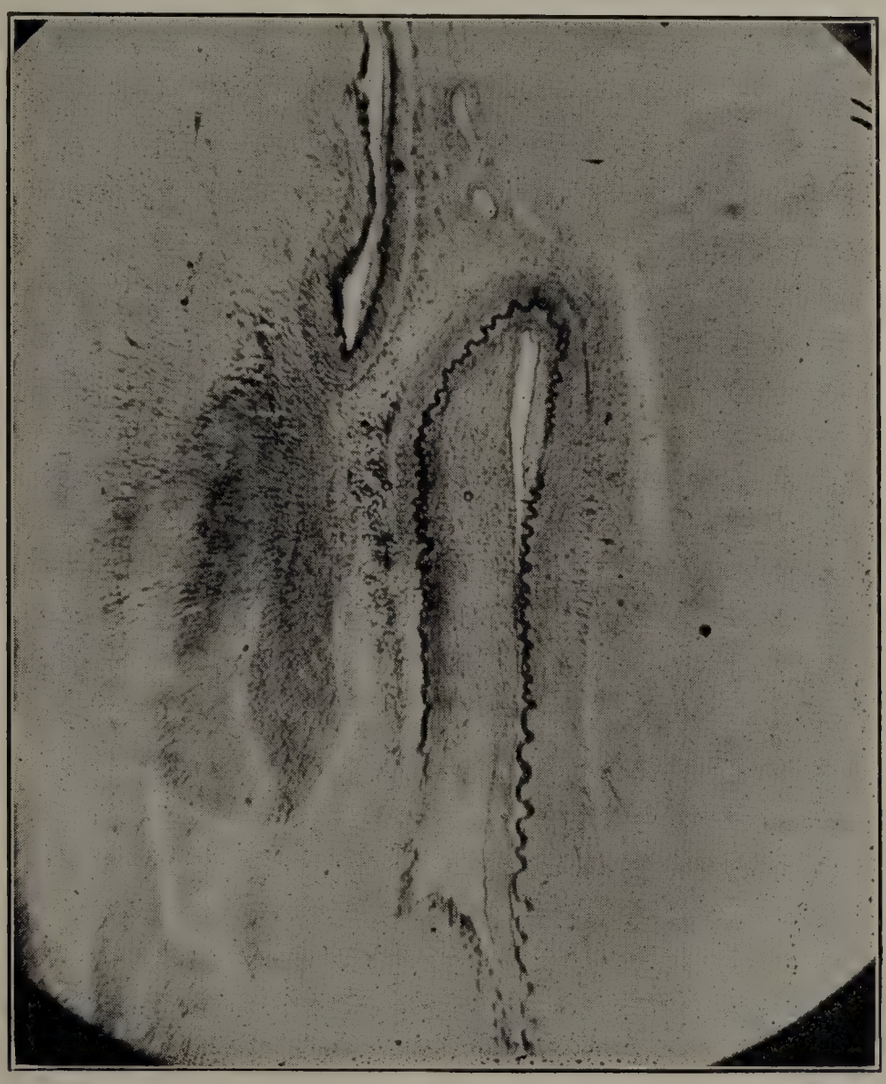


FIG. 391.—Syphilitic meningitis. Endarteritis of basilar artery.

oculomotor palsy; epilepsy, oculomotor palsy; ptosis; internal rectus palsy; optic atrophy; partial oculomotor palsy, fifth, seventh palsy; second, third, fourth, fifth, sixth nerve involvement; fifth, sixth, seventh, eighth nerve, right arm and leg palsy; seventh, eighth, psychosis (paranoia acuta) with manic moods; third, fourth, sixth, tenth, eleventh nerve palsies; second, third, fourth, sixth, seventh with epilepsy; third, fourth, seventh, polyuria; isolated internal ophthal moplegia—to mention any more would be to needlessly extend this chapter. The lesson such findings indicate is obvious. This multiplicity of syndromy in neurosyphilis has been a commonplace of neurology now for two or three centuries, but the student unfortunately

is rarely interested in history. Even professed specialists are constantly rediscovering what has been known often for decades if not centuries.

Eliminating, as far as possible, the varied syndromes of cortical or basal neurosyphilis meningo-vascularis, and limiting the discussion of the present section to these trends of cerebral syphilis due more particularly to arterial disease, what is its more frequent symptomatology?

In the first place it may be mentioned that the vascular types of brain syphilis also may be found very shortly after infection—as short a time as two or three months. Naunyn in a thorough study found that 48 per cent. of 335 cases reported on by him developed signs of cerebral syphilis within three years. On the other hand, forty years have been known to elapse between infection and the development of a cerebral syphilis.

Here the prodromal symptoms are usually headache, dizziness, sleeplessness, irritability, inability to apply one's self continuously to one's work, lack of interest in work, etc.; in general, the so-called *neurasthenic syndrome*.

The neuropsychiater sees such patients who have had their teeth pulled, their tonsils gouged out, their eyeglasses refitted, or eye muscles cut; their cervices amputated, their ovaries removed, from 9 inches to 6 feet of gut cut out, all of the endocrine glands dispensed, orthopedic apparatus applied, weeks of colonic irrigation, with or without electrical rigmaroles. Psychoanalyzed by the lay pretender. These patients often go through every fad and fiction of medicine before a competent examination reveals the underlying factor.

Headache.—The headache is usually very disagreeable; it usually has a migratory character—here, there and elsewhere; usually dull, it is at times boring. It is inconstant, intermittent, often, not by any means always, worse at night than in the daytime. It may disappear for weeks or months, and then suddenly reappear. It may also be the only sign of cerebral syphilis for months or even years.

Dizziness.—Dizziness, in shorter or longer attacks, is very significant of vascular disease. It is usually associated with the headache but may appear as the single symptom of brain syphilis. Like the headache it is apt to be increased by mental or physical work.

Insomnia.—Insomnia is frequent, often obstinate, quite variable and not infrequently sleep is made irregular and non-restful by the sense of heaviness in the head or actual headache.

Psychical Disturbances.—Psychical disturbances are the rule in those patients with arterial disease of the cerebrum. They become more or less apathetic, lose interest in their work; are unable to work because of forgetfulness or inefficiency. With this there is increasing irritability, an inability to size up the situation. Such severe disturbance is arrived at only after some time as a rule.

Abnormal Sleepiness.—Abnormal sleepiness, coming on in attacks, is not infrequent—such periods of torpor or apathy often intermitting

with periods of anxiety or of acute restlessness or excitement. Periods of stupor or semicomatose states may occur. They often presage more distinct neurological signs, being based as they frequently are, upon sudden extravasation, infiltration or thrombotic plugging off of the blood from small areas of brain tissue.

Many patients with cerebral syphilis of this general character remain in this condition, it may be, for some time; they show a picture precisely similar to certain patients with general paresis. It is in this general group that the greatest difficulties in diagnosis occur. As has been previously stated, complete serological, cytological and chemical laboratory studies are important for differentiating the trends in the present state of knowledge, and even these will sometimes be confusing.

Brain tumor is also to be thought of in diagnosis. Paresis, brain tumor, possibly gumma, cerebral syphilis, at times cannot be distinguished one from another clinically. With brain tumor, noncomplicated by syphilis, the absence of the four reactions affords a positive criterion.

Local Symptoms.—As a rule, however, the greater number of individuals with cerebral syphilis develop local symptoms, and neurological rather than psychiatrical syndromes come into relief, or the latter are intermingled with the former. Palsies develop. These are transitory, partial, not widespread, or may be severe, complete, and permanent, showing various hemiplegic syndromes, according to the anatomical site of the major disturbance—usually thrombotic—cortical, capsular, midbrain, peduncles, pons, or medulla—the symptomatology of the different forms of which are dealt with elsewhere. (See Medullary, Pontine, Midbrain, Interbrain, Thalamic, and Peduncular Syndromes.)

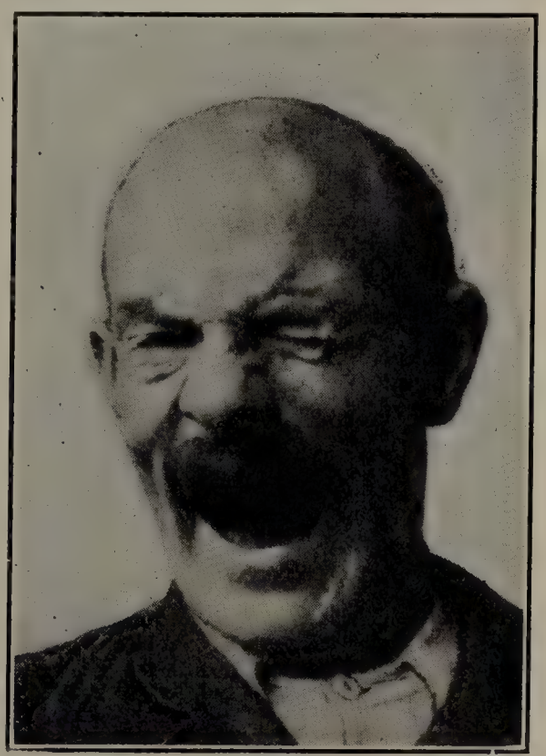


FIG. 392.—Pseudobulbar palsy from syphilitic disease. (Tilney.)

Successive attacks of mild and transitory palsies are very significant of cerebral syphilis. Monoplegias are not infrequent, one arm, one leg, one side of the face, possibly the cortical speech areas with, in case of double lesion, pseudobulbar palsy. Minor speech disturbances are extremely frequent, tremors of the facial muscles usually accompanying the stumbling, stuttering or drawling speech.

A list of the usual clinical diagnoses similar to those already outlined for basal or convexity meningitis would show a multiplicity of phenomena no less complex in the neurological field and certainly infi-

nitely more varied in the mental symptom pictures. As these latter will be discussed more in detail in the section on the psychoses no further mention will be made of them here.

Parenchymatous or Central Types.—General Paresis.[1]—It is usual to separate paresis as well as tabes from other syphilitic disorders of the nervous system, under the general caption of para- or metasyphilitic disorders. Fournier was largely responsible for this, and to paresis and tabes he added a number of other disorders, in other parts of the body, to which he applied the term para- or metasyphilitic.

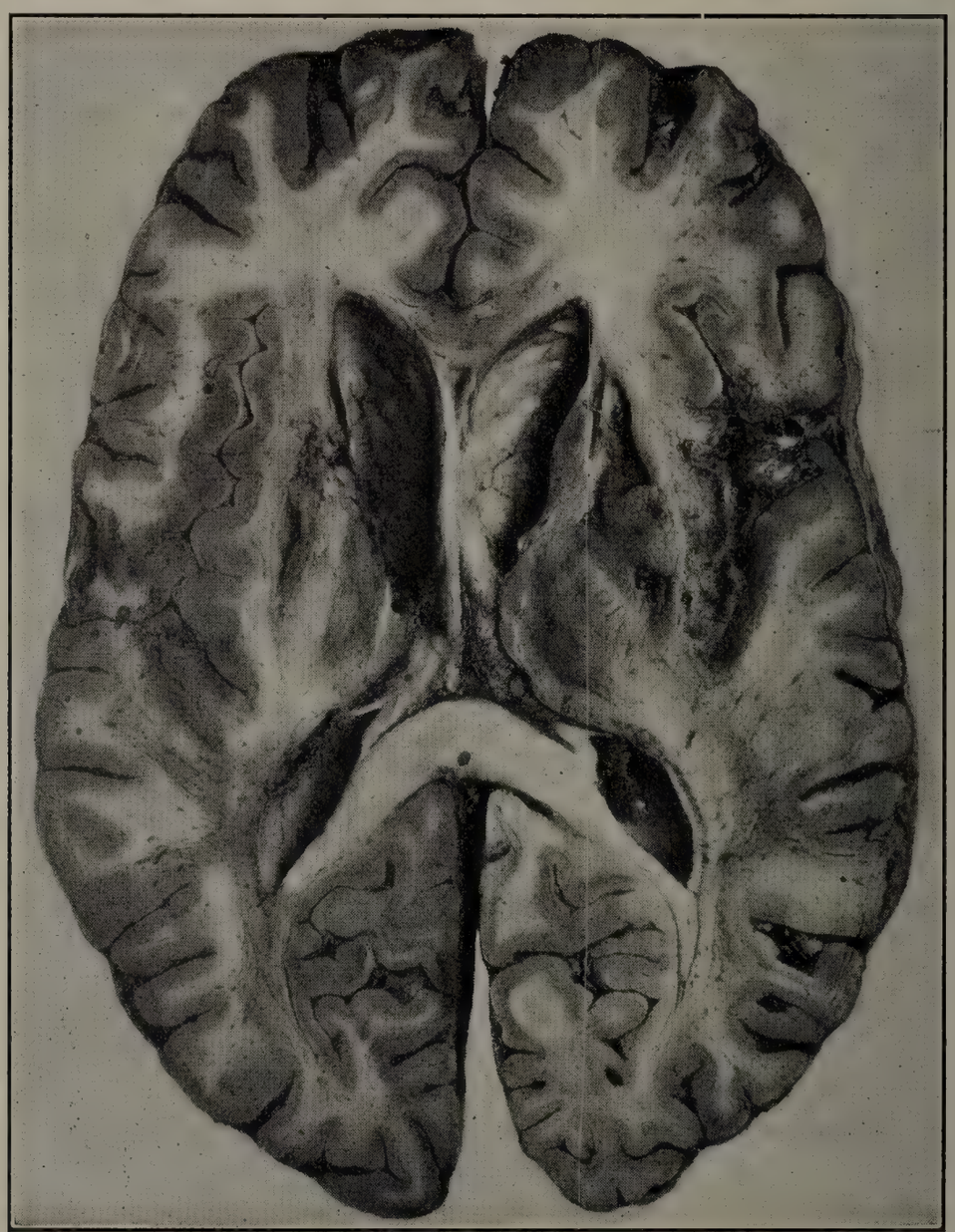


FIG. 393.—Cerebral syphilis (arterial type) with softening. Aphasia, hemiplegia, advancing dementia. (Lafora.)

Just why para- or metasyphilis is not known, especially so far as the nervous system is concerned. Many ingenious hypotheses have been formulated with the purpose of explaining the differences between paresis and tabes, on the one hand, and other types of neurosyphilis,

[1] See Kraepelin's masterly study (9th ed., 1927), compiled just before his death, by far the best study available. Earlier edition available in English, Nervous and Mental Diseases Monograph Series.

secondary or tertiary, on the other. It would serve little purpose to enumerate them in detail, since none has as yet compelled conviction.[1]

To return to the syphilitic etiology of paresis. It is practically conceded "no syphilis, no paresis." We are not speaking now of those few individuals who, either because of the presence of brain tumor or the existence of arteriosclerosis, or of other cerebral disorder, show a close *clinical* resemblance to paresis.

Finally the findings by Moore and Noguchi of *Treponema pallidum* in twelve of seventy paretic brains serve to render more certain the relationship of the organism to the disease. These findings have been amply confirmed by a number of observers in the cerebellum and aorta and generally diffused in the organs of the body (Jahnel).

But syphilis does not by any means necessarily lead to paresis, fortunately. The most recent studies of Mettler show that about 2 per cent. of those infected with syphilis develop paresis. A considerably larger percentage develop vascular cerebrospinal syphilis—how large, can only be surmised.

The question then arises, How is it that in certain cases a disorder arises, from twenty-five and a half years after infection, according to the primary age at infection, the younger the longer the latent period,[2] which, while closely resembling many forms of cerebral syphilis, is termed paresis? In other words, Why para- or metasyphilis? Bolton[3] believes that those patients develop paresis who originally had a constitutionally defective type of cortex. This only begged the question. Here may be pertinently discussed all of the contributory, accelerating, or aggravating or mobilizing factors, such as toxins, infections, traumata, surmenage, malnutrition, etc.[4]

Von Pirquet's conception of "allergie" opened a line of thought which led McIntosh and Fildes to formulate the view which many observers now hold with regard to the nature of parasyphilis. Von Pirquet showed that all infections led to a change in the character of the reaction of the tissues to the poison. This might take place in two directions; they might respond less and less to infection ("hypoallergie") until at last they ceased to respond altogether and became "anergic." But, on the other hand, a change in the opposite direction might occur; the reaction might appear both more quickly and with greater violence to a smaller dose of the poison. This he speaks of as hyperallergie and brought into this category the phenomena, described by Richet, under the name of "anaphylaxis."

The conclusions arrived at by Fildes and McIntosh[5] are as follows: (1) Parasyphilis of the nervous system is a purely clinical conception. It is a diseased state which may affect any part of the brain or spinal

[1] McIntosh, Fildes, Head and Fearnsides: Brain, 1913, vol. **36.** McIntosh and Fildes: Brain, 1914–15, vol. **37.** Spielmeyer: Histopathologie des Nervensystems. 1922. Greenfield: Pathology of Nervous System.

[2] Meggendorfer: Ztschr. f. d. ges. Neurol. u. Psychiat., 1921, **63**, 9.

[3] Bolton, J. S.: The Brain in Health and Disease, London, Edward Arnold, 1914.

[4] Scharnke: Arch. f. Psychiat., 1921, **62**, 766.

[5] McIntosh, Fildes, Head and Fearnsides: Brain, vol. **36**, Pt. I.

cord; the manifestations of tabes and paresis can only be erected into two clinical divisions by an arbitrary selection of signs and symptoms. Some forms of progressive muscular atrophy, lateral and combined sclerosis, primary optic atrophy and periodic epileptiform attacks may be equally definite manifestations of parasyphilis. (2) Parasyphilis is slightly, if at all, amenable to antisyphilitic treatment with compounds of arsenic and mercury, probably because these bodies do not enter the essential lymphatic structures of the central nervous system. (3) Parasyphilitic stages are peculiarly liable to arise after mild syphilitic infection. (4) In paresis, and in active, untreated cases of tabes, and tabo-paresis, the cerebrospinal fluid yields a positive Wassermann reaction, often of great strength. (5) With acute or chronic syphilis of the nervous system, other than parasyphilis, the behavior of the cerebrospinal fluid depends upon the extent to which the spinal cord and its membranes, including those of the brain-stem, are affected. Thus, most cases of meningomyelitis show a strong positive reaction in the cerebrospinal fluid, while cerebral lesions tend to give a weakly positive or even a negative reaction. (6) Antisyphilitic treatment has a profound effect on the positive reaction in syphilitic meningomyelitis, and the cerebrospinal fluid may give a negative reaction after a few months. But in cases of parasyphilis no positive change occurs in consequence of such treatment, within any comparable period. (7) Whatever views may be held of the primary and secondary manifestations in syphilis, Fildes and McIntosh believe that all tertiary and parasyphilitic manifestations are expressions of the reaction of hypersensitized tissues (hyperallergie). That is to say, during the previous stages of infection, the tissues have been so altered that they react more violently to a smaller dose of the spirochete or its toxins. Gummatosis is the reaction of hypersensitized connective tissues and blood-vessels, while parasyphilis is a hyperallergic reaction of the essential nerve elements and neuroglia. (8) The consequence of this hyperallergic reaction in the tissues of the central nervous system is death of any set of fibers or cells which happens to be attacked, and proliferative reaction on the part of the neuroglia within the same territory. Thus, the clinical manifestations of parasyphilis are an expression of the reaction and necrosis of hypersensitized areas of the nervous system evoked by reappearance of the *Treponema pallidum*. (9) This hypersensitive (hyperallergic) state of the tissues of the central nervous system is produced, in all probability, by the passage of the spirochetes or their toxins up the nerves from the skin and mucous membranes during the secondary period. But it is also conceivable that it may be due to a slight encephalitis during this stage of infection. The headache and lassitude, unaccompanied by any gross nervous lesions, which so frequently occur during the secondary stage, possibly represent clinically the period during which sensitization occurs. Other factors, direct concussive trauma, toxins, malnutrition, excessive surmenage, etc., cannot be excluded as yet in the total causal nexus.[1] (10) Thus,

[1] See Hauptmann: Zeit. f. d. g. Neur. u. Psych., 1921, vol. **70**; 1926, vol. **102**, on problems of immunity.

parasyphilis is a clinical conception which comprises the manifestations of a series of diseased states. From the pathological point of view the term is inadmissible. These states depend on the reaction of hypersensitized tissues to the spirochete or its toxins, and this reaction is as truly syphilitic as the production of gummata. The difference between

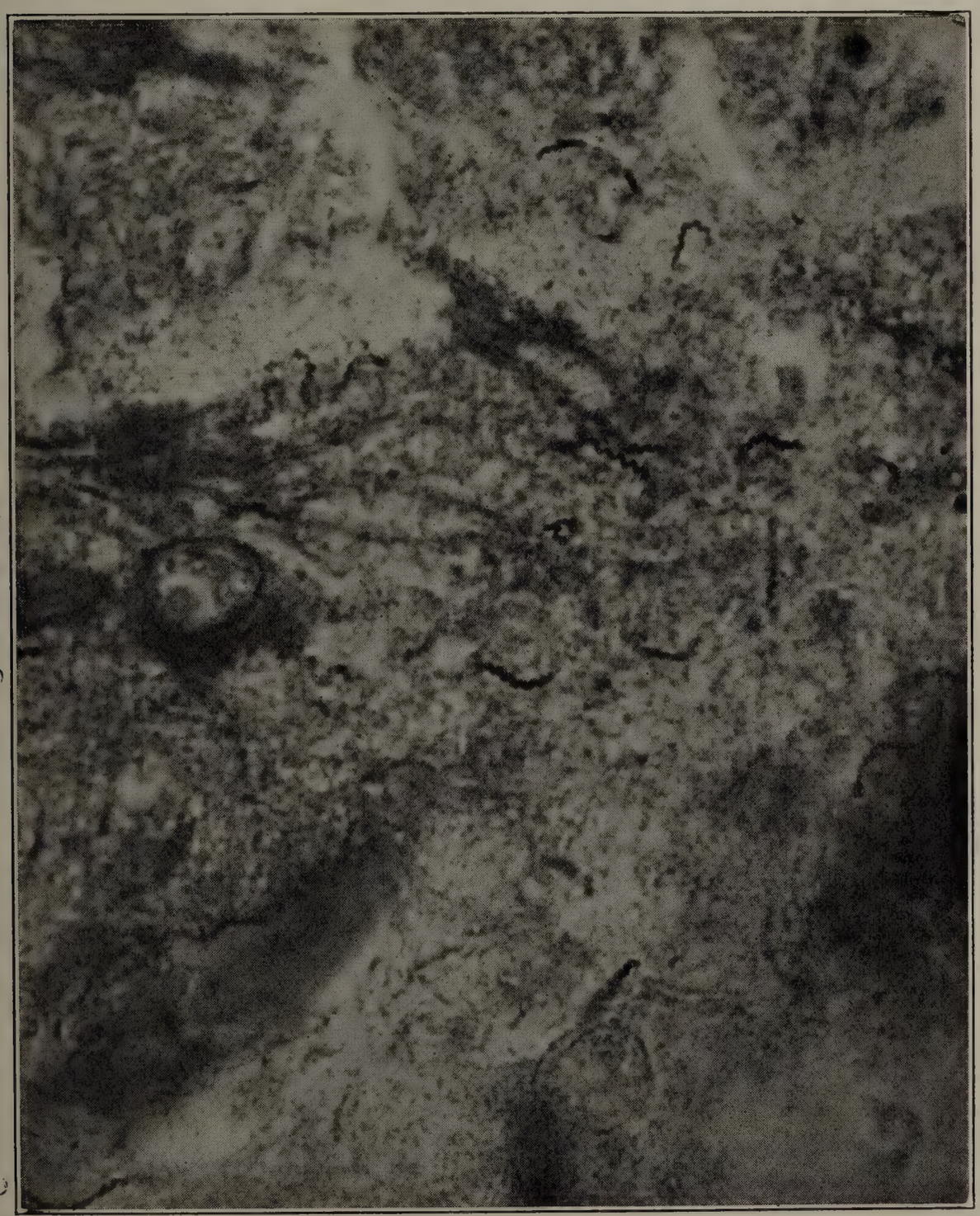


Fig. 394.—Treponema pallidum in the brain of a paretic. (Moore.)

the consequences of the tertiary and of the parasyphilitic process lies in the nature of the tissues participating in the reaction. In the one case the connective tissue is capable of repair, and the focus is readily reached by the remedial agents. In the case of parasyphilis, reaction of the essential nerve-elements leads to their death, and the antisyphilitic remedies cannot readily reach the spirochete.

We cannot here go further into the pathology of the parenchymatous types of neurosyphilis, but must refer the student to the studies particularly of Alzheimer, Nageotte, Nissl, Warthin,[1] Krause,[2] Lafora,[3] Kraepelin,[4] Nonne[5] and others.

Symptoms.—The syndrome is characterized by a bewildering multiplicity of forms, which, shifting in the individual patient from month to month, at times even from day to day, prevent any clean-cut descrip-

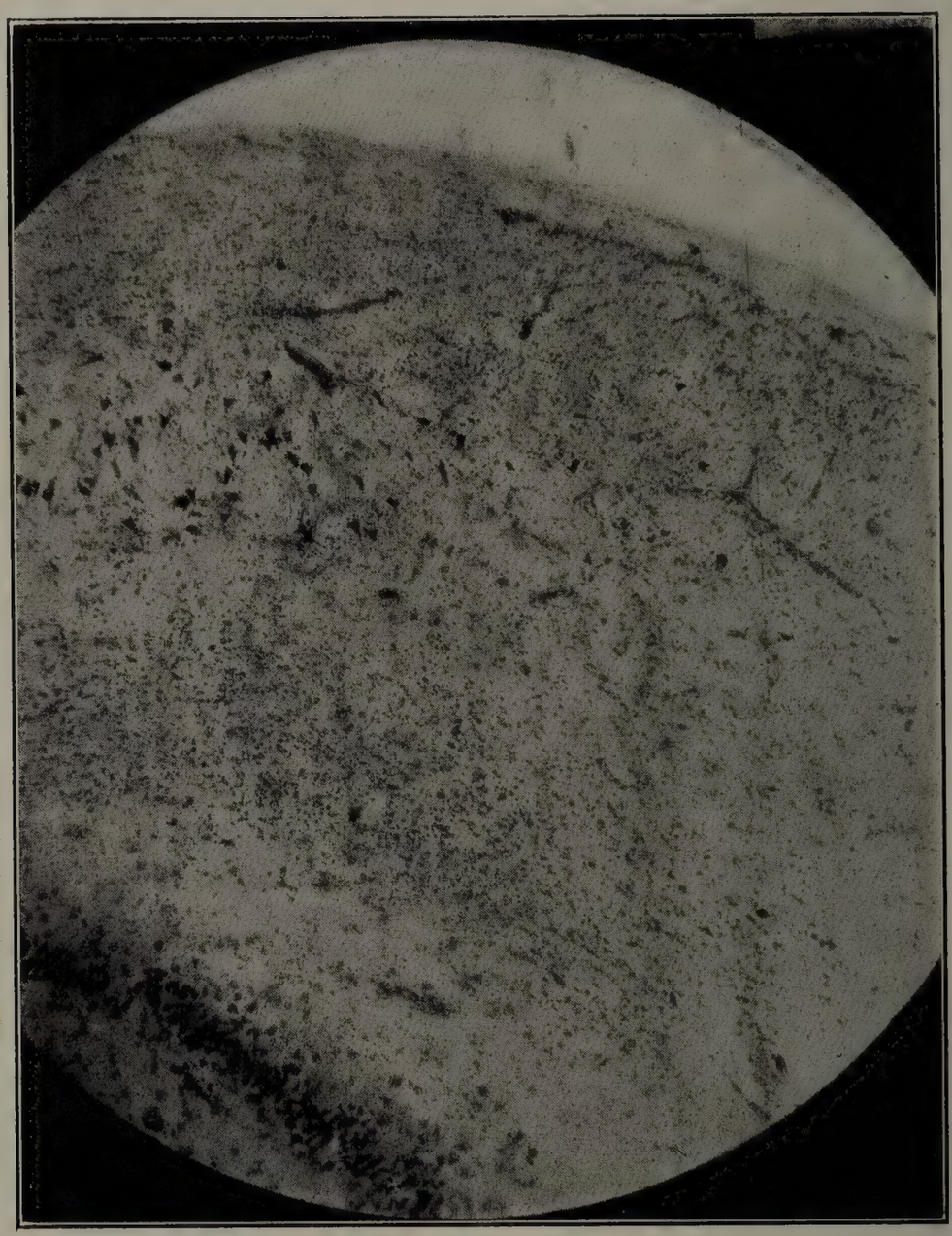


Fig. 395.—Ammon's horn in paresis. Pyramidal cell layer has disappeared, vessels infiltrated. Clinically, convulsive seizures marked.

tion that will embrace its many vagaries. Attempts have been made to create empirical types. Thus, Binswanger, in a notable study, created three types based on certain anatomical peculiarities—meningitic-hydrocephalic, hemorrhagic, and taboparetic forms. Such an anatomical subdivision has no reliable clinical counterparts.

[1] Jour. Lab. Clin. Med., 1918.
[2] Beiträge zur pathologischen Anatomie der Hirnsyphilis, Berlin, 1915.
[3] Diagnostico y Tratamiento modernos de la neurosifilis, Madrid, 1920.
[4] Kraepelin: Loc. cit., 9th ed.
[5] Nonne: Loc. cit.

Attempts at the erection of clinical types are perhaps slightly more encouraging, for many patients run a course more or less susceptible of literary descriptive generalization.

The earlier monographs of Simon and Krafft-Ebing, and the latter ones of Joffroy, Obersteiner, Klippel, and Kraepelin[1] suggest the following groups:

1. Simple dementing types.
2. Simple depressed types.
3. The expansive. or so-called classical type.
4. The agitated types.
5. The irregular types with localized symptoms, Lissauer, tabo-paretic form.
6. Juvenile paresis.

Before even attempting a description of these purely artificial delimitations, pictures which are constantly shifting and showing combinations of details, a brief glance at the chief symptom components is advisable.

These have frequently been divided into the mental and physical, but as this is a purely arbitrary distinction it will not be emphasized here. As has been noted, a diagnosis of an impending paresis may be made, at times some years before its onset, by the findings in the cerebrospinal fluid, but attention is here first focussed upon the mental picture. A peculiar psychical weakness is one of the early phenomena. This has been badly termed neurasthenic by some. A difficulty in perceiving external impressions shows this intellectual loss. In the early stages it may require special study of reaction times, which are usually lengthened, but soon absent-mindedness, inattention, loss of details, forgetfulness of important facts, become apparent. There is a gradually developing loss of ability for prolonged mental effort; in conversation finer shades of meaning are lost, the patient is no longer alert and keen, as perhaps has been his normal habit. The mental deterioration going on leads to many changes in his usual conduct, until the patient may be no longer quite sure of himself in his customary surroundings.

Certain patients develop a state of dreamy consciousness, as though in a mildly intoxicated state.

Increased fatigability is another early symptom. Much has been written of the preneurasthenic stages of paresis. This excessive fatigue may prevent him from starting anything new—sometimes he even falls asleep while at work or in conversation.

Defects of Retention and Memory.—Retention and memory soon commence to show defects. Careful studies in the early stages have shown difficulties in association, lessened capacity for learning, disturbance in attention, often with good retention. The patients forget recent happenings more readily, not knowing what has transpired a week ago, yesterday, sometimes a half-hour ago. These grosser

[1] General Paresis, Nervous and Mental Disease Monograph Series No. also 15, 9th ed., 1927, in German. Also see Rev. Neurol., 1922.

defects belong to the later stages as a rule. The memory of time relations gradually slips away, the patient being unable to arrange successive phases in an orderly series. Thus, many of these patients



FIG. 396.—Ganglion-cell degeneration and rod cells in paresis. (Lafora.)

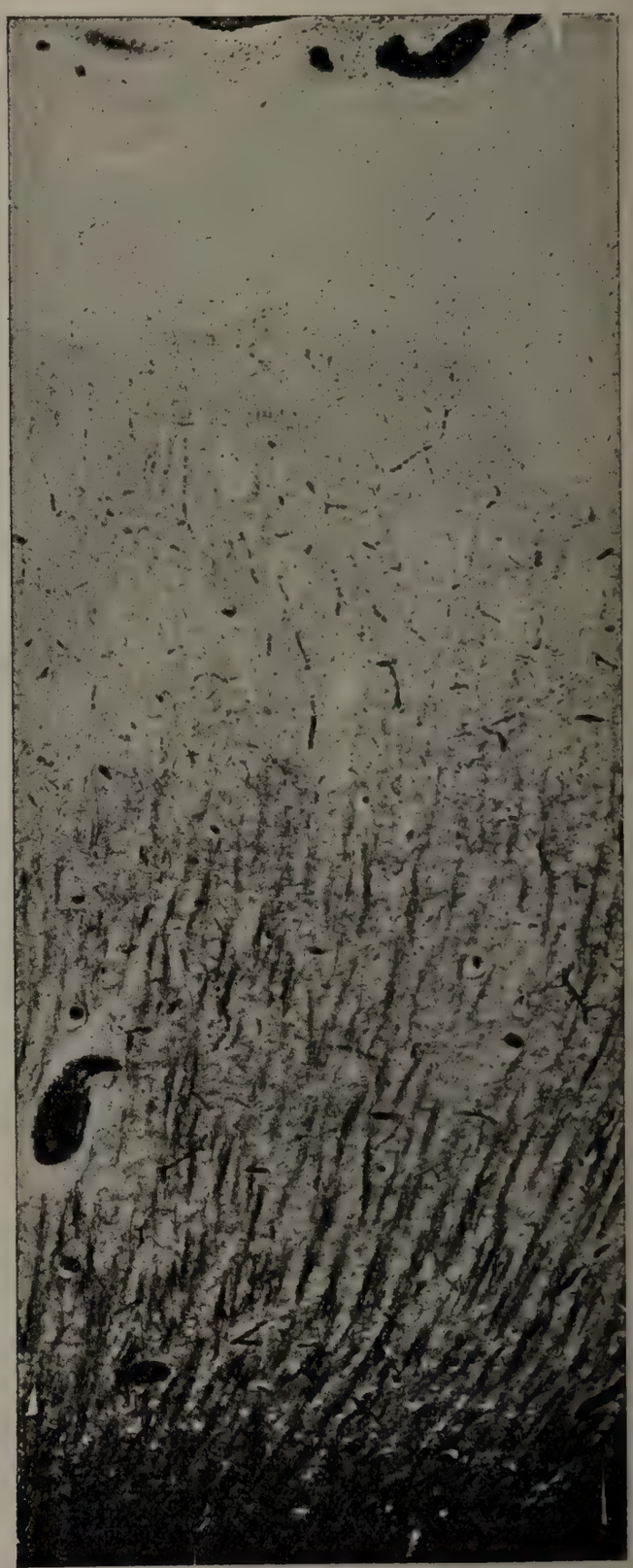


FIG. 397.—Axis-cylinder degeneration in paresis. (Kraepelin.)

show the greatest defects in their appreciation of time differences, when married, age of oldest child, and related striking facts of life. In later stages all sense of time may become effaced.

Impoverishment of Ideas.—A gradual impoverishment of ideas takes place, varying in degree from month to month and showing also great variability with different patients. Those associations most in use

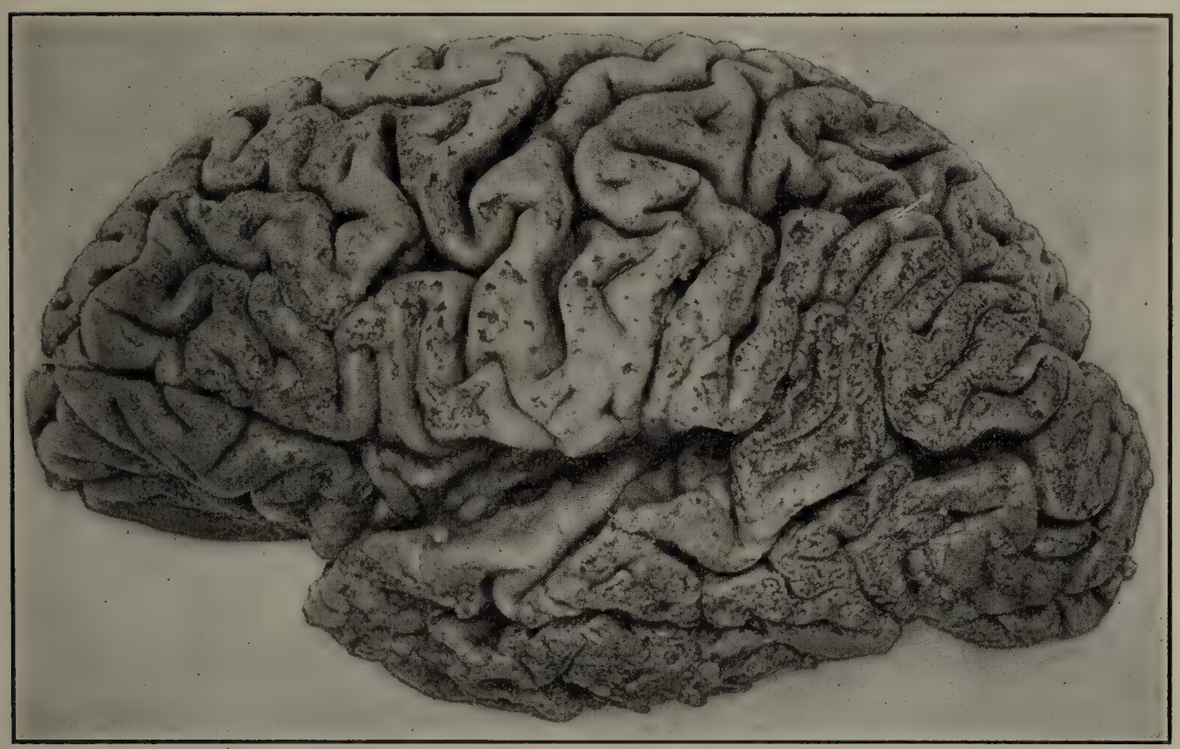


FIG. 398.—Brain of a paretic, showing marked atrophy. Flaking of the cortex due to adhesions of the pia. (White.)

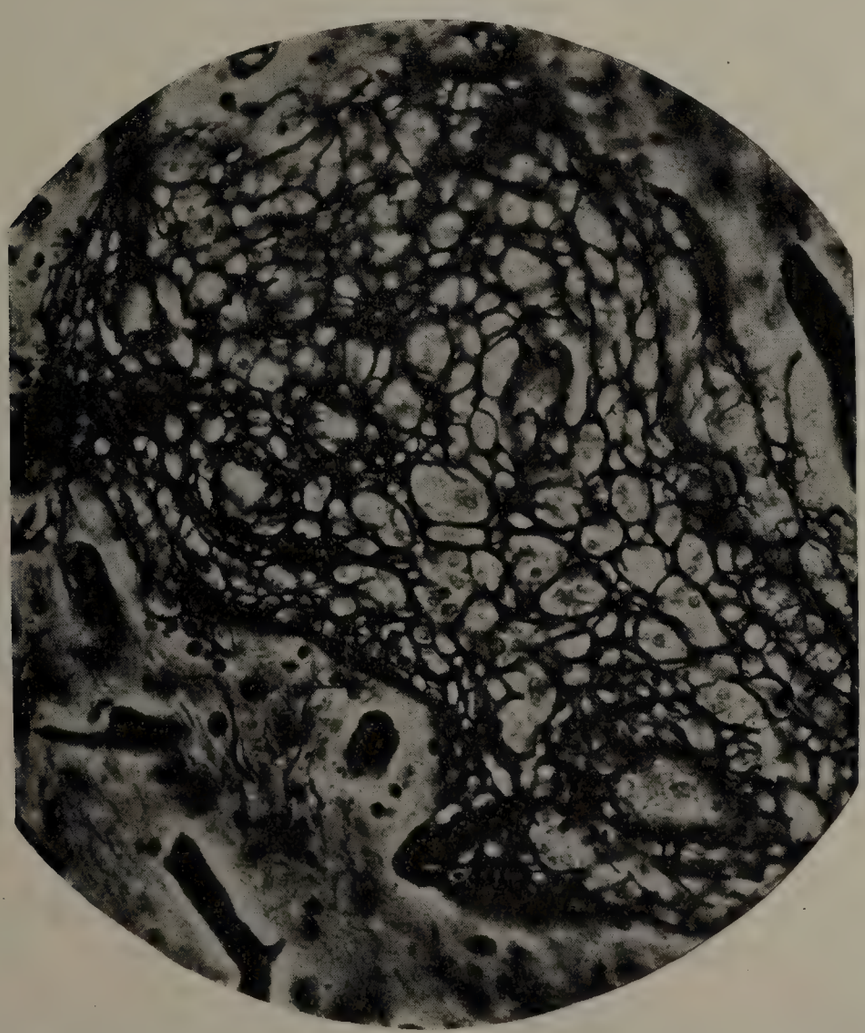


FIG 399.—Organization of Virchow-Robin lymph spaces in general paresis. Tannin silver. (Achucarro.)

usually persist the longest, and all tests should take into consideration special aptitudes. In the later stages the patients have lost practically all of their mental possessions—the gap at times being filled in by retrospective, confabulatory reminiscences. Certain of the patients resemble patients with a Korsakoff syndrome.

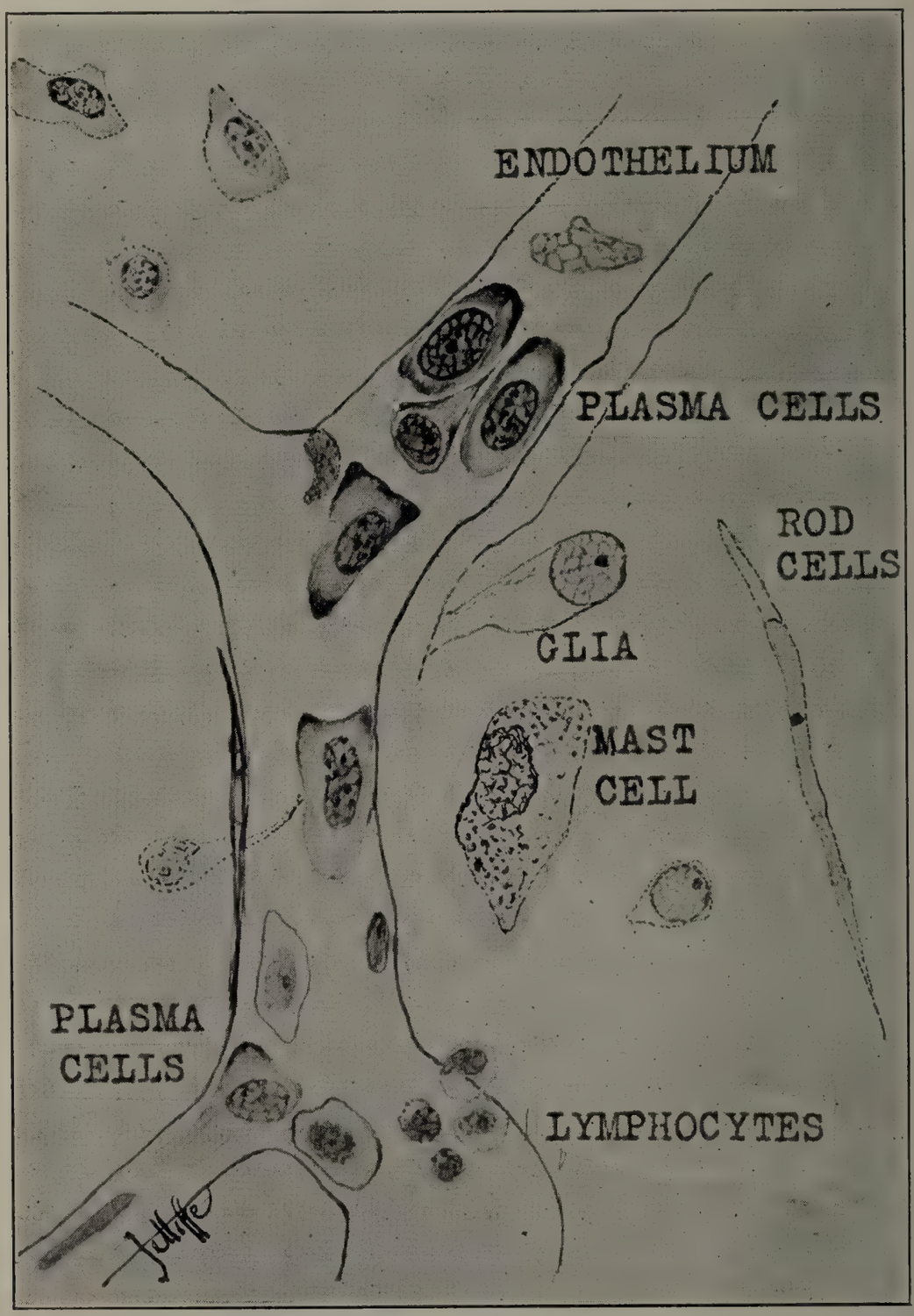


FIG. 400.—Histological changes in paresis.

Loss of Judgment.—Loss of judgment naturally goes on *pari passu* with the general psychical disintegration. In the early stages even refined methods of testing, such as those of Gregor, show that uncertainties, contradictions, logical lapses are not infrequent. The patients are easily distracted by sound associations, 9 x 9=99, etc. As this loss of judgment goes on the patients may make the most absurd plans. They do the most unusual things, often involving their entire

fortunes and playing havoc with all of their carefully woven social fabric. Dream world and real world become hopelessly confused in this fundamental psychic crumbling.

Hallucinations, illusions, and changes in simple sensory perception are found, but they are not, as a rule, prominent features in paresis.



FIG. 401.—Paresis. Cortical changes. Epileptiform convulsions. (Lafora.)

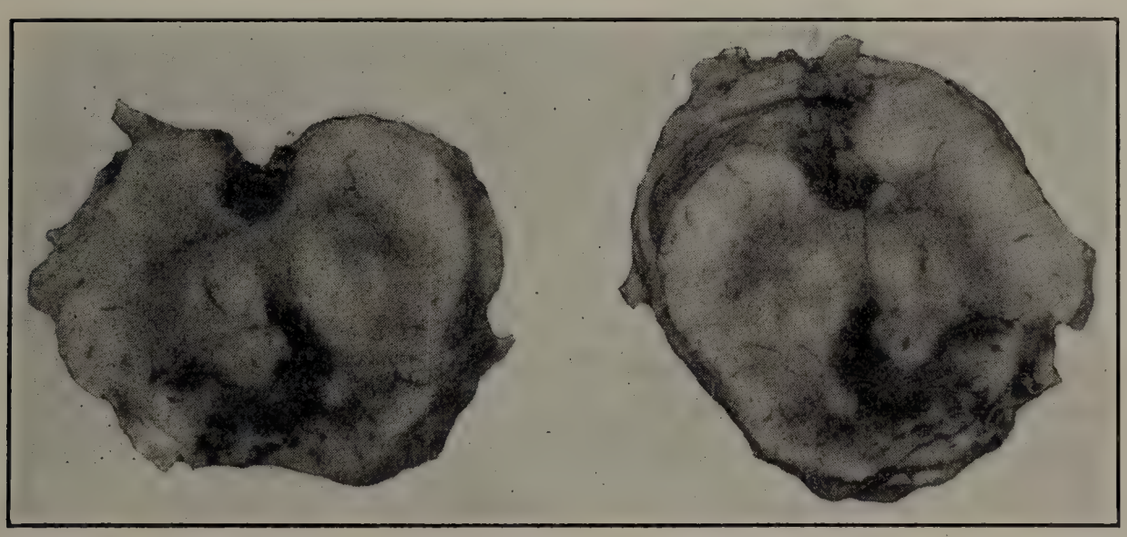


FIG. 402.—Paresis with Korsakoff syndrome; Wernicke's polioencephalitis superior.

Delusion formation is naturally present in many instances, although certain patients may go through the disorder with but few delusional developments.

Delusions.—The delusional ideas vary immensely—they are usually senseless and fantastic and when combined with active creative fan-

tasies, as they frequently are, especially in agitated or excited periods, pass all bounds. These patients think in millions, billions, quadrillions, etc. They are princes, kings, emperors, potentates, priests, Christ, God, supergods. They have rubies, pearls, diamonds, emeralds; two wives, a dozen, a harem, thousands of beautiful women, etc.

These delusional ideas, simple or fantastic, are also liable to great lability. They are always changing; contradictory as well as unconscious. New ones come, old ones go, revivals take place. Progressions may go backward: Now they have millions, next moment have thousands; now a king, in ten minutes a fine soldier. One can at times, by talking with these patients, expand or contract their delusional exuberance almost at will.

The same characteristics may be noted in regard to delusions of a depressive or hypochondriacal nature. Nihilistic delusions such as believing they have no stomach, no heart, or are dead are not infrequent in these types.

Alteration of Emotional Activity.—The disposition or emotional reactivity is involved, as is the intelligence. As a rule the patients in the early stages are hyperexcitable—others, however, are markedly depressed. They are apt to be touchy, surly, cross, even having violent outbursts for the most trivial events. There is often a distinct damper in their higher ethical feeling, so that the stimulus of conversation, the joy of music or art, of various social relations gives way to a careless indifference, often at great variance with the psychomotor activity of the patient.

As the disorder progresses the mood is apt to be colored by the delusional interpretations. Anger and laughter may follow one another in quick succession, and a great variety of fleeting, changeable, often contradictory, emotional states are passed through.

Character Alterations.—The character alterations are predominant. Decision is progressively lost; instability and foolhardiness alternating with obstinancy and perverseness. Initiative is reduced, and the patient may become as clay in the potter's hand; such periods often alternate irregularly with impulsive heedlessness. Kraepelin relates the case of a patient who stepped out of a second-story window to pick up a cigar that he happened to notice on the walk beneath him. Criminal actions may be committed in just the same manner as the case of the paretic who shot at Mayor Gaynor, of New York. Suicide may occasionally take place in the same manner. Stealing is by no means infrequent, and sexual misdemeanors and crimes are extremely prevalent. This blunting of the repressions inculcated by the force of civilization is particularly noticeable, and predominantly in the sexual sphere. Hence results the frequent telling of lewd stories, consorting with people of quite inferior social status, exhibitionism, shameful and open masturbation, and even genital assaults.

Osnato emphasizes that it is the abnormal types of personality that are more apt to develop paresis as a result of luetic infection and that

the forms of psychological reaction will conform to the types of personality possessed by the individual.[1]

Neurological Signs.[2]—Here one finds not infrequently in the beginning phases a dull, heavy headache. Hyperesthesia often precedes the blunting of any special sense, and various localized disturbances, such as word-blindness, word-deafness, auditory hallucinations, apraxia, asymbolia, astereognosis, indicate a special localization for the time being in more or less definite cortical areas. Optic nerve atrophy occurs, at times early, in from 5 to 10 per cent. of the cases. Special changes in the optic disk are recognizable in from 12 to 50 per cent. of the cases.

Changes in Cutaneous Sensibility.—Very frequently cutaneous sensibility is modified—sharp pains, numbness, itching, etc., occur, and in those forms recognized as taboparetic these often show the special localizations of the tabetic. Out of these changed sensations delusional interpretations frequently arise. A general insensibility to peripheral stimuli develops later, and the patient may then pay little attention to any kind of irritant, heat or cold, full bladder, distended rectum, etc. Occasionally such patients mutilate themselves, cutting off a finger, or the tongue, or the testes in order to get rid of what seems to them a foreign body.

Motor Incoördinations.—Motor incoördinations, from initial trembling to more high-grade ataxias, apraxias, adiadokokineses, Romberg, asynergiæ, are common. Intention tremor is not infrequent and perseveration is almost never missed in the later stages.

Disorders of Speech.—In speech the motor difficulties have been specially studied since Esquirol first laid stress upon such changes in mental cases. Frequently beginning with slight stumbling, a slurring over certain letters or syllables, *r*, *l*, etc., the paretic develops very characteristic speech anomalies. These come out with marked prominence in the use of test phrases—electricity, Methodist Episcopal, organization, truly rural, third cavalry brigade, etc.—when certain letters are repeated, stumbled over, or elided. Paraphasia, aphasia, ataxia of speech, perseveration are among the frequent later developments until only a mumbling may be possible in the last stages. Similar changes take place in writing.

Eye Symptoms.—In the ocular movements, analogous difficulties are observed and in the pupils one observes a variety of changes. Statistical studies show these pupillary anomalies to be extremely frequent. Differences in size from 50 to 80 per cent. (Rache); distortion of the pupillary outlines, 74 per cent. (Joffroy); Argyll-Robertson pupil, 50 to 70 per cent. (Westphal, Junius, Arndt, etc. Many of these pupillary anomalies undergo considerable variation, changing from time to time even without treatment. Loss of consensual light reflex, as already noted, is often one of the earliest, and at the same

[1] See his interesting analysis of the content of the paretic mental fabric in terms of preparetic character type in Jour. Nerv. and Ment. Dis., May, June, 1919.

[2] Macfie Campbell: Focal Symptoms in General Paresis, 1914.

time one of the most persistent of the pupillary anomalies. Early retinal changes of minute exudative types are frequent.

Convulsive Phenomena.—Convulsive phenomena, epileptiform or apoplectiform in character, are rarely missed in paresis. They are usually of the cortical epileptic type. They often occur early in the disease or may punctuate any period in its development. At times limited, they more often are generalized, and frequently have prodromata, such as dreamy states, motor incoördination, thickness of speech, twitchings, etc., as a rule occurring early. As isolated phenomena, typical status attacks may be observed, with as many as 100 or more epileptiform crises in twenty-four hours. An attack in the very early stages may last only a few seconds; the patient suddenly sinks back on his chair, and is all right in a few moments—while, on the other hand, status attacks may persist a week or even more. Unconsciousness is usual, though it may be very slight or fleeting. A vast variety of focal residuals have been described.



Fig. 403.—Set of pictures of paretic convulsion. (Kraepelin.)

Similar changes may be observed on the sensory side of the nervous system, and so-called psychic equivalents, as in the more classical epilepsies, are frequent.

Statistical studies show the very great frequency of these attacks, Obersteiner recording them as often as in 90 per cent, of his patients; while Junius and Arndt in their extensive study of Berlin material, give them as occurring in 53 per cent. A personal study (J) of 200 cases showed them in 78 per cent. of the patients. Kraepelin believes that treatment in bed limits the number and frequency of the attacks; his Munich statistic show an incidence of about 65 per cent.

Alterations in Reflexes.—The tendon reflexes—triceps, radius, knee-jerks and Achilles—are usually positively involved, either excessive, in the greater number of cases, or diminished, especially in those patients with posterior cord involvement, which is frequent.

When the deep reflexes are found to be increased other symptoms of involvement of the pyramidal tracts are not infrequent. Babinski reflex, very frequently Chaddock's external malleolar sign, at times the paradoxical reflex of Gordon occur. Possibly there is an ankle-

clonus, and spasticity in gait is present. If, on the other hand, the deep reflexes are diminished, other signs of involvement of the position sense and deep sensibility fibers, travelling the posterior column pathways, are usually found. Ataxia, Romberg, girdle sensations, anesthesiæ, etc., pains of the radicular type, are also often encountered in these taboparetics.

In most of the patients there is great variability in the two sides. Occasionally one finds spasticity of one and hypotonia and ataxia of the other, and combined symptoms are to be expected in the later stages, especially in those patients with prominent cord localizations.

In the final stage contractures occur in the bed-ridden patient. They are unable to do anything and muscular twitches, spasms, localized atrophies, and a veritable museum of anomalies is to be looked for.

Findings in Cerebrospinal Fluid.—The findings in the cerebrospinal fluid have already been discussed. Suffice it to say here that they are of paramount importance and a diagnosis of paresis without the signs obtainable in the cerebrospinal fluid must always be regarded as lacking in a most important element.

Enough has been said to show that the clinical picture of paresis may be closely counterfeited by a number of other pathological states —notably brain tumor, cerebrospinal syphilis, arteriosclerosis, chronic alcoholism, sleeping sickness, etc.

The findings in the fluid are very definite. A positive four reactions, the fluid used in small quantities—0.05 to 0.2 c.c.—is almost certainly diagnostic of paresis, yet at times it would appear that positive four reactions are found in other syphilitic processes which do not behave like paresis. The gold-sol reaction usually offers typical paretic curves.[1] The newer tests offer much of interest.

Vasomotor and Trophic Disturbances.—Vasomotor and trophic disturbances may appear early and come and go; among them skin eruptions, such as herpes, and pemphigus are the commoner types met with. The ready appearance of bed-sores and abscesses indicates the lowered resistance of the skin and subcutaneous structures.

The bodily temperature may show considerable variation, even on opposite sides of the body; it is usually subnormal in the later stages, save following convulsive seizure. Sleep is irregular, especially in the excited stages, when the paretic may not sleep for days—whereas in torpid stages or in those quiet, dementing forms the patient sleeps or is in a doze much of the time.

The appetite is capricious, and the bodily weight is apt to fall off in the early stages and during excitement, to become much increased in the torpid, quiet states.

Disorders of the bladder, and incontinence of urine and feces, all sooner or later come within the outlines of the picture.

Remissions.—One clinical feature which is very striking is the tendency of this disorder to show marked remissions. In certain

[1] Kaplan: Serology in Nervous and Mental Disease, loc. cit.; Rawlings: Am. Arch. Neur. and Psych., Aug., 1919.

respects this is a general law in disease processes, but in paresis it appears most striking because of the almost miraculous change that takes place in the patient. Such patients are about to die; they become absolutely helpless, convulsion follows convulsion; they know nothing, are bed-ridden, have to be fed, soil themselves, and are reduced to breathing, heart-beating automata. They may remain in this condition for weeks and months, and then pick up a little, and then more and more, and within a space of six weeks to three months many such patients appear to be almost well and like themselves. They have risen from the dead, and strange to say, although the relatives, friends, and business associates have been told over and over again perhaps, for they should be, that this is not a cure, that it is only a remission of symptoms, the patient is frequently restored to all his civil rights and given full control of his affairs. In the majority of cases this is disastrous; he may launch out into new lines, involve his fortune, marry unwisely, and then after a few months, perhaps a year—the longer remissions on record have been five or six years—the average is about six months—the symptoms return, often in rapid progression, and usually lead to death after variable intervals of from six months to a few years.

Forms.—To return now to the subject of the forms—those more or less artificial groups which for the purposes of description psychiatrists agree upon.[1]

The symptomatology of paresis varies within such wide limits because of the extent and distribution of the pathological changes; because of the individual make-up of the patients, and because the disease process not only affects the highest psychological levels but strikes deep into the physicochemical and sympathetic foundations upon which these higher levels are built. The disease presents therefore, a combination of psychological symptoms more or less explainable at that level coupled with disintegrations of a much more material character and stable organization.

1. *Dementing Form.*—That which characterizes this general group is the progressive mental deterioration with motor paresis. Excitements, convulsions, extravagant delusion formations are not prominent and when present are transitory. In these individuals there is the early period of nervous irritable weakness, with loss of mental alertness, moodiness, inability to work, forgetfulness, and steadily increasing poverty of thought. Naturally, the picture beginning in this way may suddenly change. This is sufficient to throw the patient into another group—but if the development is of the slow, progressive nature, gradually advancing mental weakness, fleeting delusional ideas, often with childish, weak-minded features, these are the general symptoms of the dementing type of paresis.

2. *Depressed Forms.*—Here anxious depression is in the foreground of the mental picture. Hypochondriacal, delusional states are promi-

[1] See White, W. A.: Outlines of Psychiatry, 11th ed., 1926.

nent. The patients continually complain about bodily discomfort; have lost their intestines, or have destroyed their manhood by masturbation or sexual excesses. These delusional ideas become more and more nonsensical. In many instances the hypochondriacal ideas are dependent upon fancied sinful actions or wrong-doing. They are great sinners, they must be protected from the police, they fear they will be sent away. Such patients often have persecutory ideas, and when such are prominent early in the disease, before there is marked deterioration, they are frequently regarded as "paranoiacs" especially by those who are taught to regard names as disease entities and seek for a *diagnosis* from a "pathognomonic" symptom. These paretics with persecutory ideas have pronounced hallucinations of hearing. Notwithstanding these hypochondriacal or persecutory, delusional interpretations, these patients are markedly indifferent; they are apathetic, talk and move in a monotonous, dull manner, and take little interest in their surroundings.



FIG. 404.—Simple dementing form of paresis.



FIG. 405.—Paresis. Demented type of facies.

Thus the energy, the libido not being able to go forward in the affairs of life, a marked pathological introversion takes place, and regression takes hold of all sorts of nonsensical, childish, infantile, and archaic fantasies. Pathological projection also is common, and one has a regular chaos of pathological mental mechanisms. The sense of reality is so markedly impaired, and the affective relativity so cut off, that the nonsensical beliefs have no corresponding or adequate emotional relationships. The personality is fragmenting and disintegrating.

Childish regressions of hero formation appear. The patient is a god, a king, an emperor; like Jack and the beanstalk, he is miles high; as in Gulliver, he is a great giant; as in Midas' touch, or Aladdin's lamp, he breaks the bank at Monte Carlo, or is the owner of immense gold mines, fabulously valuable jewels, etc.

Contrasting states of great inferiority, weakness, poverty, cause them to be very fearful, easily confused, easily lost; they beg for protection, hide from anger, or ask piteously for food, preserve scraps, etc.

They become unmanageable in bed, and finally in many the agitation and fear develop great resistance and violence. Self-destruction may be attempted, mutilation occasionally occurs. Most of these attempts, however, are fragmentary, non-sustained and bungling.

Stuporous states show a contrast to this marked violence. They may persist for weeks, months, or even years. The patients lie stupidly, "depressed," or anxious in bed, unclean and unmanageable. Special rigidities, catatonic-like in their nature, may develop.

The special statistics show that from 15 per cent. to 20 per cent. of the material in some of the larger European hospitals and clinics may be in general thrown into this depressed category.

3. *Expansive Types.*—This general type has been for years considered "classical," yet they are not as frequent as the dementing types. Thus, Kraepelin gives 30 per cent. in his Heidelberg series, Junius and Arndt 27 per cent, in their Berlin material. It is to be regretted that the specialist has failed to emphasize this feature, which is of so much value to the general practitioner, and has confused the issues by speaking of a "change in type." It is of more value to insist upon the comparative rarity of the megalomanic features of paresis, since, as the average medical man has been taught to recognize paresis by this sign, it is not to be wondered at that so much delay has occurred before the recognition of paresis. The emphasis should not be laid upon the comparatively rare expansive cases.

In this megalomanic type the boastful ego rises to superior heights. Everything is seen from the standpoint of a feeling of abundant energy. At first the ideas are those of great exaltation, within the bounds of normal human experience, but soon the patient looses his earthly bonds and soars to superhuman unrealities. His strength is appalling; his education superior to any others in the world; he speaks ten, nay, all languages; has all wealth; all power; figures mount from thousands to millions, to pages of ciphers. And in kaleidoscopic changes, and great individual variation one learns of many marvels of superior excellence only dreamed of in childish fantasy, or seen in the boasts of primitive peoples.

One feature of this frightful megalomania, which has its very great ups and down, should never be overlooked; namely, the tendency for such patients to commit genital indiscretions, even atrocities; or to engage in the most foolhardy enterprises, thus jeopardizing life and property.

This feature in paresis is of so much importance that special attention

should be devoted to the legal measures which should be invoked to prevent the worst consequences of this mental disturbance.

That this megalomania has a distinct deterioration background is seen in the frequent combination of a poor clerk, in a state institution, who speaks of the million-dollar novel he is writing. It consists of a few miserable scrawls on toilet paper, or on the edges of a daily newspaper. This is only a type. Such inconsistences may be read of in the classics of psychiatry, from the work of Arnold, in 1700, to the present time.

These fantastic, exalted, euphoric states very frequently elaborate on sexual themes. Thus the patients have hundreds, millions of wives or concubines—"Solomon was a piker in this matter," boasted a Bellevue patient. The children are more numerous and more beautiful than any promised to the ancient Hebrew heroes.

One patient, mentioned by Kraepelin, could lift ten elephants, was two hundred years old, 9 feet tall, was a beautiful Adonis, weighed 400 pounds, had an iron chest, an arm of silver, a head of gold, 100 wives, 1000 million boys and girls, his urine was Rhine wine, and his feces were gold.



FIG. 406.—Paresis, showing grandiose type.

The illustrations might be repeated *ad infinitum*. They are to be found in richer or poorer elaboration, in shorter or longer intervals of excitement, in this exalted euphoric type, but one may see a hundred paretics, as a general practitioner may see them in the early stages, and never get a ghost of an idea of such experiences. Of this 100 some time, sooner or later, 25 to 30 of them will be liable to exhibit the exalted, euphoric, megalomanic picture here indicated rather than described.

Consciousness is usually much clouded in this form, especially while the delusional projections are in their full growth. Time, place, the great world, is a dreamy, far-off place of little moment to the mind engaged in its ambitious program. Continuity of thought is practically impossible, and chaos and anarchy exist. In such minds hallucinations are frequent.

The mood is happy, overflowing with schemes for good deeds and generosity, and all-embracing in its brotherly love. But coherence is not to be expected. Hypochondriacal ideas, such as delusions that there are worms in the head may rest in bizarre connections with the delusion of being a great philosopher, a Shakespeare, etc., and changes in mood are of frequent occurrence. Weeping follows ecstasy, and is

replaced by beatific, sublime happiness. Sudden, passionate excitement leaps up under restraint, to subside, or to be diverted by such a trifle as a falling leaf, or a ring at the door bell.

The great psychomotor excitement is a striking feature, and one difficult to manage. These patients walk miles, are on the go, meeting people, busily engaged in everybody's business, making plans for self and others, and, when he is confined, the paretic's violence knows no bounds. He is transformed into a raving animal.



FIG. 407.—Excited paretic. (Kraepelin.)

Throughout all of the excitement, divertibility, constant changing of plans, mixture of silly pleasure and superficial sadness, there is the note of great deterioration of critique and emotional degradation and degeneration which shows particularly in the conventions relative to one's person. Carelessness in dress, uncleanliness, grossness in eating, loss of finer susceptibilities, coarse expressions, frank immoralities—these are but a few of the possibilities in such lax conduct.

In watching such patients from day to day, one is struck by the immense variability in the picture. The ideas of grandeur may all vanish, the patient denies he ever said any such thing, he may get angry in a dispute over the matter, and then launch into a magnificent grandiloquent invective of collossal outlines.

As the dementia increases, these large ideas may entirely disappear, or be preserved, and appear on the surface only as a few words, or murmurs, "good to eat," "fine women," "millions," etc.

Finally, in the later stages, the patients all sink to a more or less common level—"sans everything."

Among the expansive forms may be found the quick, galloping patients who die within a short time. Increasing experience seems to show, however, that these excited types indicate a very severe reactive process, and hence, if they do not die in the height of the reaction (galloping cases), they provide the greater number of the more stationary and protracted forms—those who make a partial recovery with defect, and who later disintegrate. Remissions seem to be common in this type as well.

4. *Agitated Forms.*—Those patients who show a predominant motor activity in the beginning may be said to be grouped here. Great restlessness runs through the entire picture. The mental content is very variable—euphoric, depressed, hypochondriacal, mood colorations flit in and out. Galloping cases are usually grouped here, in which an extremely rapid and fatal course is present.

This is really only a subgroup of the preceding type, only artifically separated off by reason of the more consistently persistent psychomotor restlessness. Remissions are frequent, as are also the apoplectiform and epileptiform attacks. The pathological process simply has a wider extension in the motor areas.

The acute delirious cases, somewhat resembling delirium tremens of alcoholism, and independent of it, are arranged by Kraepelin in the agitated group.

5. *Irregular Types.—Lissauer,* etc. These patients, showing irregular forms of development; neurosymptomatic groupings, hemiplegias, etc., are here brought together. The hemiplegic and taboparetic groups are the more frequent.

Taboparesis.—Taboparesis is the most striking of these irregular forms and deserves a further outlining.

It has been assumed by many, especially by neurologists (Schaffer, for summary, 1912) that tabes may be regarded as a spinal paresis, and paresis a cerebral tabes; that is, the disease varies only by reason of the greater severity of the process in the one or the other localization.

Kraepelin, on the other hand, accentuates the opposing psychiatric view, that whereas the two disorders are undoubtedly fundamentally syphilitic, yet they are two different kinds of processes, and that when the symptoms of tabes are added to paresis the changes in the cord are not exactly similar to those found in tabes limited to the cord. The

different findings in the cerebrospinal fluid in the two disorders would point to some sort of a difference as well. There are no real differences. The discussion is only worth while if it will lead to a solution of the very practical question: What makes the pathological lesion locate itself in one or another region? There are few facts as yet available to enable this question to be answered.[1]

Clinically, taboparesis shows in a combination of the symptoms observed in the two forms. Raymond and Nageotte would have it that every paretic would show tabetic signs, if he lived long enough. In those patients with pronounced tabetic onset one finds the frequent pupillary anomalies, the diminution or loss of the patellar reflexes, Romberg sign, ataxia of lower or upper extremities, or both, hypotonia, lancinating pains, crises, and arthropathies. These are found in the more definite taboparetics closely associated with the psychical disturbances already outlined. In the more classical tabetic patients the mental disturbances, to which Cassirer and O. Meyer have devoted their attention, are very distinct from those of paresis.

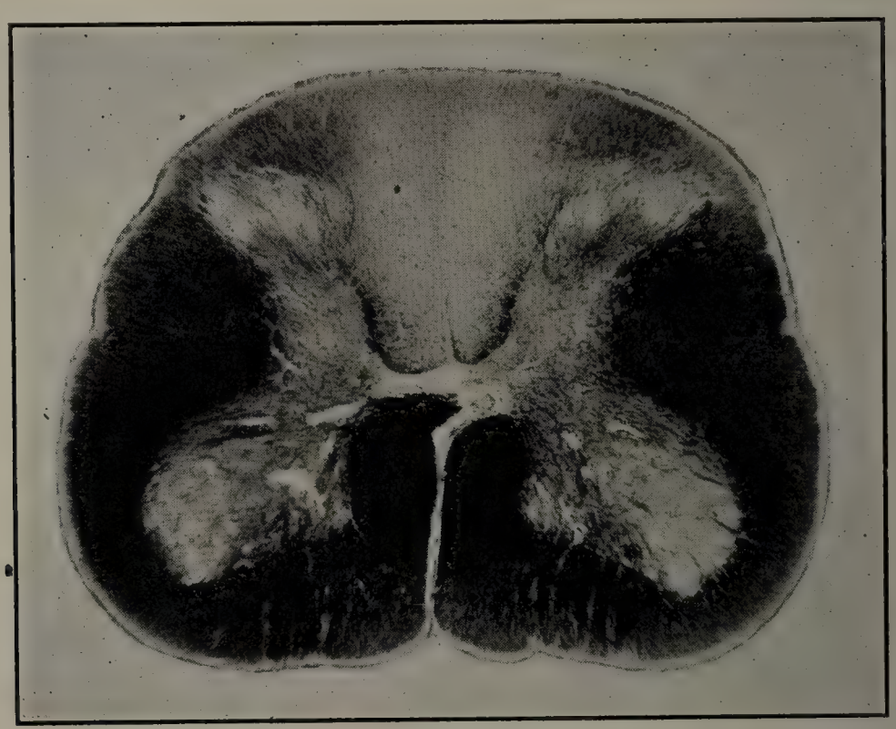


FIG. 408.—Paresis with tabetic changes in spinal cord.

6. *Juvenile Paresis.*—This form is quite distinct. It was apparently first recognized in 1877 by Clouston. Gudden in 1894 gave a monographic study of 20 cases. Following this the reports mounted so that by 1903 at least 141 reported cases had appeared in the literature. Kraepelin's daughter[2] has given the latest (1920) monograph on the subject. It appears at the present time not infrequently, since the Wassermann-Plaut findings offer such certain criteria for its determination. Such tests seem necessary, since the clinical picture may be so

[1] See Jelliffe: Discussion, New York Neurological Society, March, 1919; Jour. Nerv. and Ment. Dis., August, 1919.

[2] Toni Schmidt-Kraepelin: Ueber die juvenile Paralyse, Berlin, 1920. Kraepelin: Loc. cit. Nonne: Loc. cit.

extremely variable—hence it was overlooked—many patients dying diagnosed as "imbeciles."

Here the patient may make a comparatively normal development to five or ten years of age—certain non-developmental forms probably belong here, but are now disregarded. Then the child's mentality seems to drop. In older children, ten to sixteen, this drop is more apparent. Poor memory, bad motor adaptation and gradual deterioration appear. Childishness, fabrication, excitements, and depressions, fears, and anxieties, are frequent. Epileptiform convulsions appear—



FIG. 409.—Juvenile paresis in the emaciated stage.

many juvenile paretics are gathered into the almshouses and epileptic colonies as "epileptics with feeble-mindedness"—and after a course of three or four years, with gradually deepening mental disintegration, the patient dies. The histopathological changes are identical with those of the adult form.

It is noteworthy that the age of onset, from seven to twelve years, is the same length of time that in the adult form elapses between infection and the outcrop of the parenchymatous disease.

Interpretation of the Paretic Psychosis.—The interpretation of the mental symptoms of paresis may advantageously be approached from

two angles: In the first place, from a consideration of the personality make-up previous to the onset of the disease process. The disorder of mind must be a disorder of that which existed before and therefore the character of the mental symptoms is directly dependent upon the type of individual. Osnato[1] believes that it is the abnormal types of personality that are more apt to develop paresis following luetic infection.

The grandiose type of paretic symptom we believe falls quite in line, in its interpretation, with this concept of organ (cortex) inferiority. The destructive luetic process produces as it advances an ever increasing mental inefficiency which is compensated for in the only possible way because of its organic basis, namely by fantasy. The patient begins to build up delusions of power to compensate for the progressing weakness and these delusions increase in improbability as the destructive process results in increased mental weakness. This pyramiding of the delusions of grandeur (the patient is not only worth hundreds of thousands, but millions, trillions, quadrillions, etc., finally having to invent words to express the size of his fortune) goes on and on in a vain effort to overtake the results of cortical (mental) destruction. Real inefficiency is compensated by an effort to think himself efficient; efficiency is sought to be brought about by th nking hard enough that it is really so; results are sought by thinking of them as attained—the "all-powerfulness of thought."

5. **Syphilitic Psychoses.**—In this section are included the psychoses which are associated with cerebral syphilis and with tabes. In the present state of knowledge a clear distinction cannot be made either on pathological, clinical, or psychological grounds between the so-called metasyphilitic and the more clearly syphilitic conditions, and there are undoubtedly all sorts of gradations between these two practical divisions.

Forms.—*Neurasthenia.*—Kraepelin speaks of a syphilitic neurasthenia—a form of mental disturbance much written upon by earlier authors. The preneurasthenic phase of a cerebral syphilis or of a paresis is not now under review. It is apt to appear shortly after infection and manifest itself in a nervous discomfort, difficulty in thinking, irritability, disturbance of sleep, pressure in the head, variable and changeable discomfort and pain. To these may be added slight depression, dizziness, confusion, anxiety, slight difficulty in finding words, temperature variations, paresthesiæ, and nausea.

Many students prefer to interpret these symptoms as a direct result of the infection, and not as a circumscribed syndrome; but slight vascular changes, minute pupillary alterations, and particularly evidence of meningeal irritation as shown by the spinal fluid lymphocytosis, point in the direction of its being something more than a simple, infectious reaction.

[1] Personality in Paresis, Jour. Am. Med. Assn., February 16, 1918; Jour. Nerv. and Ment. Dis., May, June, 1919. See Hauptmann, loc. cit. Hollos and Ferenczi: The Psychical Disorder in Paresis, Nerv. and Men. Dis. Mon. Ser. No. 4.



FIG. 410.—Brain of a patient with a syphilitic psychosis of acute maniacal type. Enlarged and engorged vessels. Syphilitic meningitis. (White.)

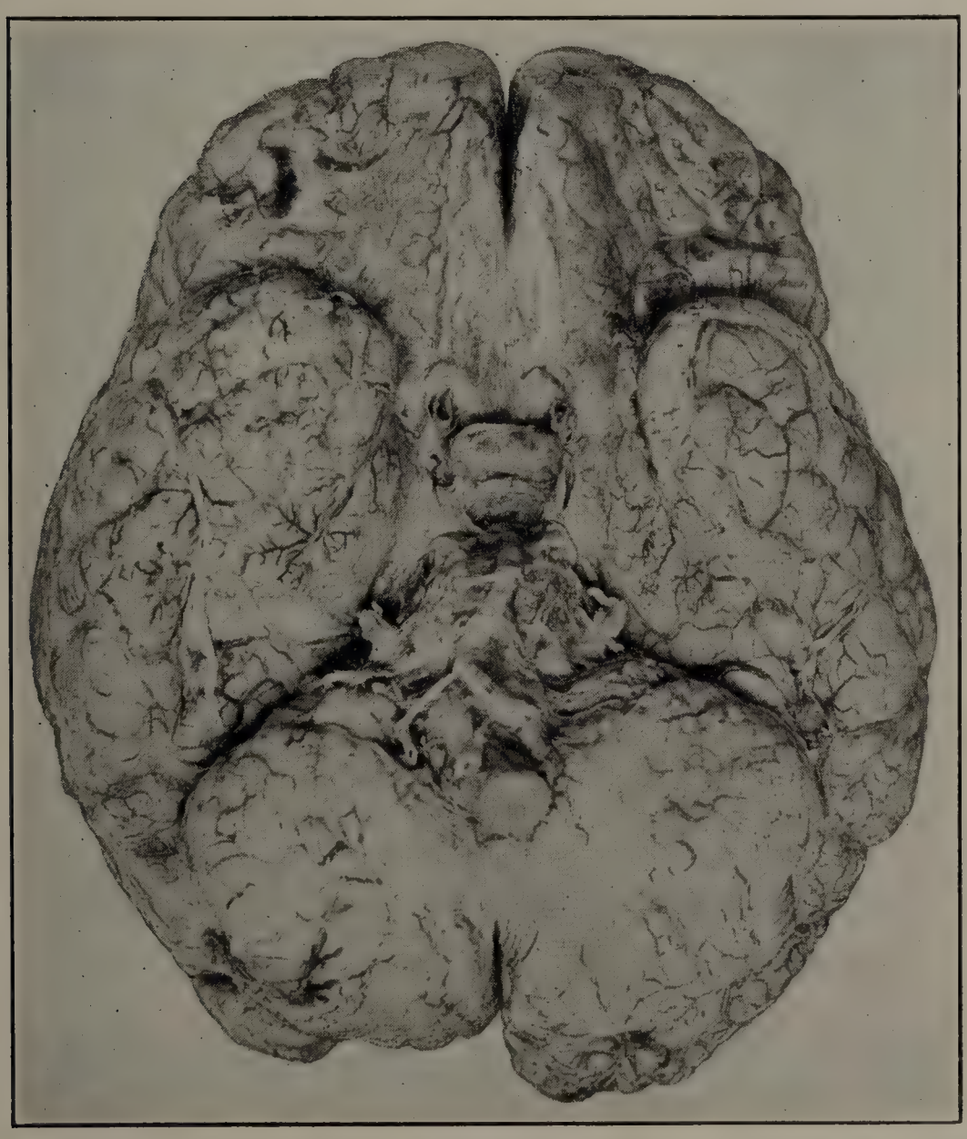


FIG. 411.—Brain of a patient with a chronic syphilitic psychosis of maniacal type. Syphilitic leptomeningitis, pachymeningitis. (White.)

Plaut has described the *psychoses* which are associated with cerebral syphilis and with tabes in ten groups. The groups are as follows:

1. *Simple Luetic Weakness of Mind.*—This is the weakness of mind which usually goes with gross lesion of the brain, marked by hemiplegia or monoplegia. It is generally the result of the blocking of a considerable vessel by a thrombotic process and usually occurs in relatively young people. There is no well-defined type of mental defect resulting, as this is dependent, of course, upon the location of the lesion and upon the make-up of the individual. There may be depression or euphoria or a simple forgetfulness and indifference. Occasional cases of arteriosclerosis occurring early in life simulate this condition very closely.



Fig. 412.—Brain of juvenile paretic showing marked atrophy. (White.)

2. *Syphilitic Pseudoparesis.*—Here a group of cases is found which seems to occupy all portions of the territory between the true syphilitic psychoses and paresis. On the mental side the distinction between pseudoparesis and paresis is practically impossible to make. Persistent auditory hallucinations, however, seem in experience to have pointed quite strongly to pseudoparesis. The most reliable diagnostic criterion is the behavior of the cerebrospinal fluid toward the Wassermann reaction, it being often negative in vascular syphilis and positive in paresis. While this is not an absolute differentiation, and larger doses of the serum may produce the positive reaction, it is still, however, one of the most important differentials. It has to be remembered, too, that some cases of paresis are found with negatively reacting fluid, and rarely cases of lues with positively reacting fluid.

3. *Paranoid Forms Combined with Tabes.*—In this group are found patients who do not show any considerable deterioration, but present

ideas of persecution with numerous auditory hallucinations over a considerable period of time. There is no self-reproach, they remain

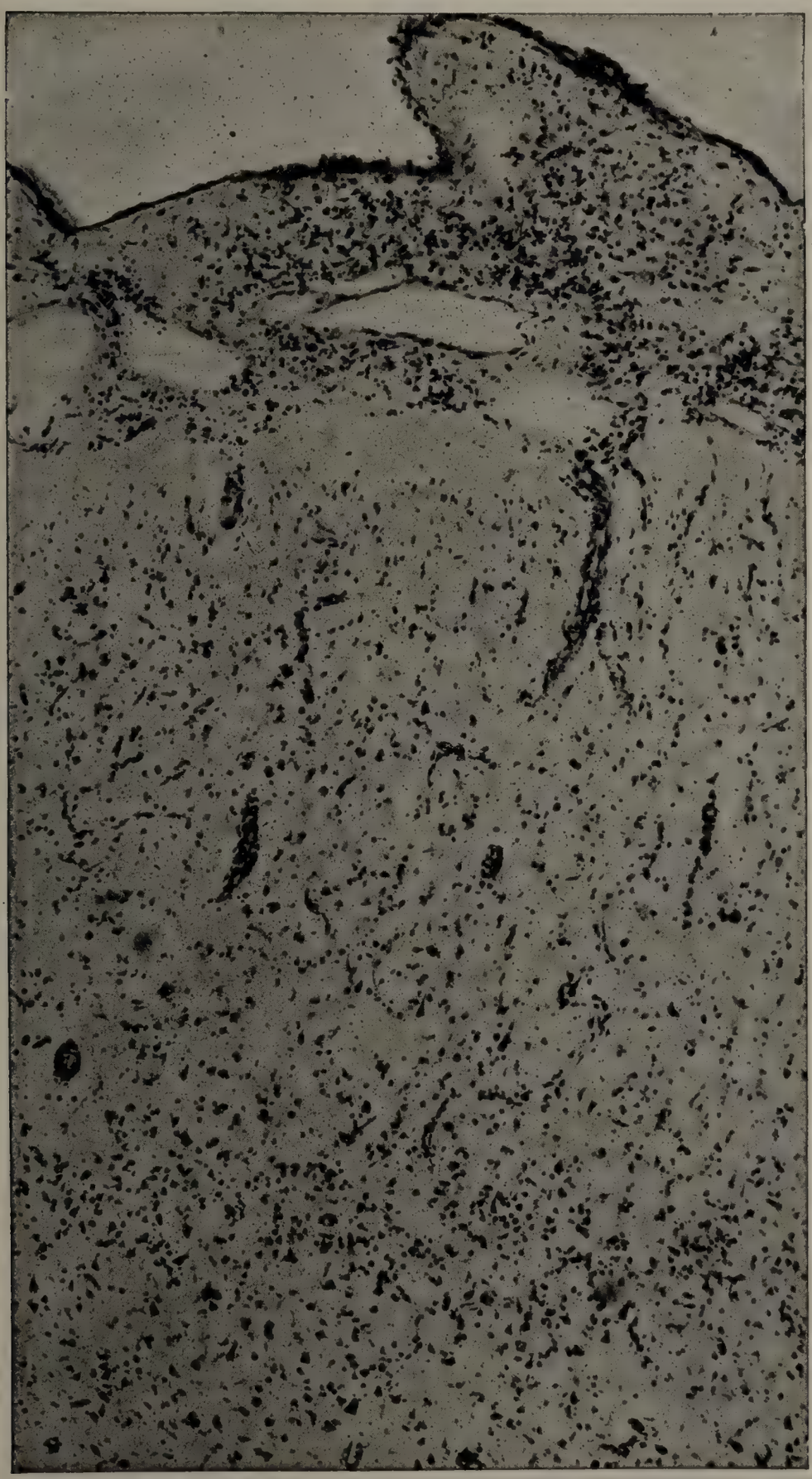


FIG. 413.—Devastation of cortex in paresis. (Kraepelin.)

lively and affable, and what seems to be peculiar, present marked hallucinations of common sensibility accompanied by fantastic ideas.

4. *Paranoid Forms without Tabetic Symptoms.*—In this group are presented paranoid ideas combined with auditory hallucinations, rather resembling the alcoholic hallucinoses. A pronounced delusion of jealousy was present in one case described by Plaut. They have to be separated from manic-depressive psychosis and particularly from dementia precox. The separation from the latter is made from the absence of catatonic signs and failure to develop marked evidences of defect.

5. *Certain Epileptic Forms.*—These appear to be due to the endarteritic changes in the small cortical vessels and may be combined with paralytic phenomena which develop as a result of the participation of the larger vessels in the disease process. The cases resemble very closely genuine epilepsy and must be differentiated by the neurological and serological findings. A case described by Plaut showed transitory dream states.

6. *Short Hallucinatory Confused States.*—These resemble the confusions associated with the epileptic forms and the French have considered them in the same class with the crises.

7. *Psychotic Disturbances Associated with Syphilitic Cardiac Disease.*—This condition probably develops most frequently in connection with syphilitic aortitis.

8. *Psychoses Resembling Manic-depressive Psychosis.*—Here conditions are grouped which superficially very closely resemble the manic-depressive psychosis. As a rule, however, there is something to attract attention as indicating at least an aberrant form. On the mental side the delusions are more grotesque, more out of harmony with the personality of the patient, or show an unreasonableness which is not commensurate with the degree of excitement. On the physical side, of course, inactive pupils should lead to a serological examination. Occasionally such episodes occur a long time before the outcrop of frank symptoms of metasyphilitic disease.

9. *Mental Disorder Due to Syphilis as a Psychic Trauma.*—Here a psychogenic psychosis which is more apt to take a depressive form is included.

10. *Hereditary Luetic Mental Disturbances.*—This envisages psychopathically defective subjects and weak-minded children with luetic etiology. The exact relation between lues and various forms of weak-mindedness is not accurately known, but it is known that a large number of the feeble-minded group are luetic. Syphilitic brain disease may occur in early infancy and proceed for some time, producing only transient symptoms, perhaps an occasional convulsion, and ultimately lead to serious defect.[1]

6. **Tabes.**[2]—**History.**—In any historical presentation a sharp distinction must be made between the name tabes dorsalis, and the syndrome as now understood. So far as is known, the former had its origin with Hippocrates, the latter, if one accepts the post-Columbian origin of

[1] See behavior of colloidal-gold tests in differentiating certain of these types by Rawlings: Loc. cit.

[2] See valuable monograph by Maloney: Locomotor Ataxia, New York, 1917. Decourt: l'Ataxie aigue tabetique, Paris, 1927.

syphilis, could only have come into existence among Europeans and their descendants after the fifteenth century.

The various interpretations given to the Hippocratic term throughout the ages is a chapter of surprises. Spermatorrhea, gonorrhea, leucorrhea, gleet were its initial meanings, with or without signs of organic disease of the cord; when combined with cord signs—myelitic processes, usually tuberculosis—Pott's, etc.—it was called tabes nervosa or myelophthisis. Out of this mass the syndrome of the present day was separated. It was natural that excessive venery should have been attributed as the cause. As late as the early part of the last century (1804) the term persisted in the sense of a "nervous weakness," *i. e.*, what today one would designate a "sexual neurasthenia," or a "sexual phobia." So far as can be made out from Littre's Hippocrates this is exactly what Hippocrates wrote about under the term tabes dorsalis. Even in the theses of Harless and Brera, in the early part of the nineteenth century, the relation between the cord, the vessels of the lower abdomen, and secretions from the genito-urinary organs was held to be a close one.

As to the earliest observations of modern tabes, the traces are very indistinct. To the clinicians of the sixteenth and seventeenth centuries, the confused mass of paraplegias was practically insoluble, and it would appear that it was only in the beginning of the nineteenth century that the process of differentiation took place. E. Horn (1816) called attention to a number of the important features including blindness; Weidenbach (1817) attempted to make a separate disease of it—he contested the inflammatory origin—said it had nothing to do with consumption, although still unable to break away from the belief in the excessive venery etiology then rampant. Schesmer (1819) described the peculiar gait in an unmistakable manner, while W. Horn (1827) emphasized the real absence of a true paralysis, and spoke of an ataxia wherein this affection was different from other forms of myelitis. Decker (1838) called attention to the swaying and unsteadiness with closed eyes, which was taken up by Romberg three years later and rechristened Romberg's sign.[1]

Pathologically, the characteristic cord signs were not unobserved. Hutin (1828) describes them, Ollivier of Angiers (1837) gave the picture reproduced here in part, while Cruveilhier (1832-1845), in his Atlas, gives masterly clinical and pathological descriptions.

Romberg in the first edition of his Lehrbuch gave greater precision to the description, and Steinthal (1847) threw together the incomplete paralysis (or ataxia), the Romberg sign, and the characteristic gait. Later Romberg, in the second edition of his text-book (1851) gave greater precision to the concept, and gave a classical description. Finally, Duchenne, in the years 1852 to 1858, elaborated the general idea, and gave the first complete and adequate description of the disorder. It may be said that Horn and Romberg had practically made

[1] See Neuburger: Aus der Vergangenheit der Neuropathologie, Wien. klin. Wchnschr. 1911; in collected essays of F. H. Garrison; also interesting history of neurology by Garrison in Dana's well known text-book, 11th ed., 1926. See Jelliffe, Fifty years of American Neurology, Semi-Centennial Vol., Am. Neur. Soc., 1924.

out of the general tabes dorsalis collection a special tabes dorsalis collection in the sense of present-day conceptions, in the physiological and pathological fashioning of which Todd (1847), who seemed to grasp the fact that incoördination and posterior columns were related, Rokitansky (1854), Virchow (1855), Türck (1856), Landry (1858), and Gull (1858) made lasting contributions. The recent history gives us the names of Charcot (1868), Vulpian, and Topinard, and in 1863 three monographs appear by Eisenmann, Leyden, and Friedreich, while the later contributions of Westphal (1875), Argyll-Robertson (1869), Marie, Redlich and Obersteiner, Nageotte, Oppenheim, Dejerine, Goldscheider, Erb, Nissl, Schaffer, and Alzheimer record the detailed studies in etiology, symptomatology, pathogenesis and spinal cord conditions of the last two decades.[1] Finally, the even more recent work of the serologists, particularly Wassermann, Citron and Plaut,[2] and Noguchi has given the final word regarding the etiological factor, syphilis.

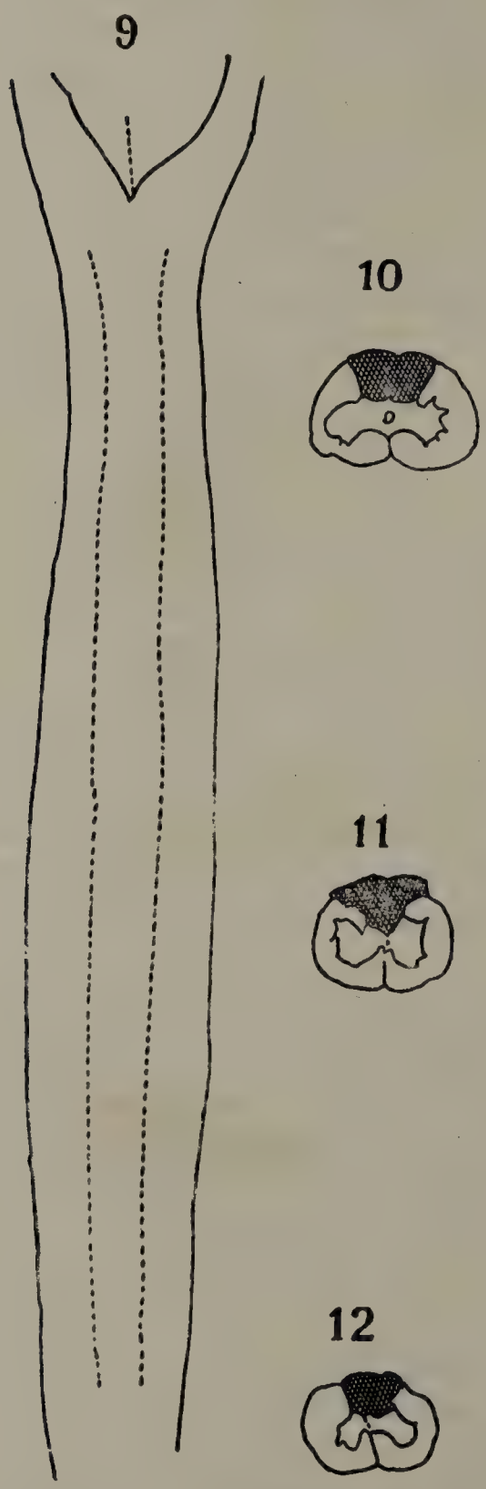


FIG. 414.—Illustration of the cord of a tabetic given by Ollivier of Angiers in 1837.

Etiology and Occurrence.—Syphilis is an essential factor in the production of tabes. The statistical method had almost proved the syphilitic etiology of tabes, but with the advent of the objective methods of Wassermann and his students all doubts have vanished. In the more recent work of the most competent serologists syphilitic substances are found in the blood serum of practically every case (Plaut, *loc. cit.*). Furthermore, the cytological examination of the cerebrospinal fluid shows the presence of cellular exudates characteristic chiefly of the syphilitic processes, and as will be seen in the consideration of the pathological features, the syphilitic nature of many of the findings is beyond controversy. Finally, *Treponema pallidum* has been found in the spinal cord areas in tabetics.

[1] Jelliffe: On Some of the More Recent Literature of Tabes Dorsalis, Pathology and Etiology, Int. Clin., 1907, **2**, 257. Later literature, see Schaffer: Lewandowsky Handbuch and work by Massary, 1909. For early history consult E. Meyer: Kritisch-Historische Beobachtungen, Strassburg Diss., 1880. Papers by Neuburger: Loc. cit. Nonne: Loc. cit.

[2] The Serodiagnosis of Syphilis in Psychiatry (Nervous and Mental Disease Monograph Series, 1911).

Pseudotabetic syndromes are known to occur in multiple sclerosis, in tumor, in caries, from accident, gunshot wounds, in poisoning by alcohol, pellagra, hypothyroid states, diabetes, ergot, etc.

What secondary factors are necessary to determine why this or that patient infected with syphilis should develop tabes cannot yet be answered. Less than one-half of 1 per cent. of the infected develop the disease, so that other factors are demanded on *a priori* grounds to explain why one syphilitic individual develops it and ninety-nine syphilitics do not; and this is true only for certain races. The many cases of congenital tabes following a syphilis from the same source, suggest variations in the virus. Similarly the large number of tabetics who are known to have followed the wake of certain syphilitic prostitutes (Morel Lavallée, Erb, Brosius—glass-blower cases). Another factor may be due to variations in resistance. (See hyperallergy discussion in paragraph on paresis.) Jelliffe[1] has reported a partial pseudotabetic syndrome of hypothyroid origin, and it may well be that syphilis plus some one or may be several factors are essential for the development of a tabes.

It is highly doubtful if any of the many causes usually mentioned, such as exposure to cold and wet, trauma, excessive venery, etc., have anything to do with the after-development of a tabes. The multiplicity of assigned causes makes it more than probable that none are primarily concerned. Ford Robertson's hay bacillus is certainly not a proven secondary factor. Trauma is highly doubtful. It may cause a pseudotabes from lesions in the cauda equina chiefly. Of heredity little is known. Charcot, Borgherini, Erb, and Gowers have laid much stress upon it.

Occurrence.—The majority of cases occur in the fourth decade, but this is largely due to the fact that the disorder comes on ten to twenty years after infection, and syphilitic infection usually occurs between the twentieth and thirtieth years. The disease may appear at almost any period after infection (fifteen to seventy years of age). The average runs from thirty-five to forty, the average interval after infection about fifteen years, with extremes at four to thirty-five. Dejerine and Raymond report cases forty-five and fifty years after infection. In certain races with a high syphilis percentage (Algiers) tabes is practically unknown, and there is every reason to believe that the syphilis has been present there since the fifteenth century. In other races from three to five of every one hundred syphilitics develop the disease. In certain countries the ratio of men to women is 4 to 1, in others 10 to 1.

Occupation seems to play only that role that speaks for increased opportunities for syphilitic infection.

Symptoms.[2]—The symptomatology of tabes is markedly diverse, and whereas it seemed at one time that it presented a more regular

[1] Jelliffe: Hypothyroidism and Tabes Dorsalis, New York Med. Jour., March 2, 1921.

[2] See Maloney, loc. cit.

picture than other nervous disorders, accumulating experience shows that typical pictures, so-called, are the exception rather than the rule. In other words, there is no one symptom that may not be absent, and very few symptoms that may not be present in disorders other than tabes.

Nevertheless one can rely fairly well upon the following grouping of symptoms: Lancinating neuralgic pains, mostly in the lower extremities, usually preceding all of the other symptoms; paresthesiæ and related sensory disturbances, analgesiæ, hypoesthesiæ, loss of the tendon reflexes (patellar, Achilles), incomplete or complete Argyll-Robertson pupil, unilateral or bilateral ataxia in both extremities, Romberg's sign, bladder disturbances, hypotonia, ocular palsies, positive Wassermann in the blood and lymphocytosis in the spinal fluid.

A host of other symptoms may be present in individual cases, either early or late, but those just mentioned belong more particularly to the majority of the cases, and are usually sufficient to make an early diagnosis. A description of the individual symptoms will show somewhat of the proportionate occurrence, both in point of time and frequency.

Pains.—Severe lancinating pains occur in about 90 per cent. of the cases, and usually as an initial sign (60 per cent.). The patients complain in an almost stereotyped manner of having had sharp, severe, fugacious pains usually in the sciatic and crural distributions, which they speak of, and unfortunately are regarded even by physicians, as rheumatic. These pains come on in attacks, last a few minutes or more, several hours, a few days and then disappear to again recur. They may precede the development of other symptoms by a few months or even many years (twenty-two years—Erb). The average varies widely. They are an indication of the leptomeningitis or radiculitis which is one of the fundamental results of the syphilitic virus or products induced by it. Maloney (loc. cit.) contends they are mainly due to implication of the vegetative nervous system and groups the pain syndromes with gastric and other crises. The pains are usually of extreme severity and are much dreaded by the patient.

Whereas the distribution is predominantly sciatic or crural and radicular at first, the pains may be widely distributed, and may in fact start in any sensory root, cranial or spinal. Thus trigeminal neuralgia may be an initial sign, or the pains may affect the larynx, or the stomach, or the heart, the bladder, the testicles, the intestines, and give rise to various forms of crises, so characteristic and so much feared. The pains may be felt in the skin, or deeper lying structures.

Deep, boring pains are also present, usually later. Not infrequently the larger nerve trunks are somewhat painful to pressure. This fact may lead to confusion in separating an alcoholic or other neuritic pseudotabes.

Crises.—These have some relation to the pains of tabes, and are probably due to similar pathological alterations, but located in other vegetative reflex arc segments. The best known are the gastric

crises, noted as early as 1856 by Gull and recognized by Charcot (1868) as belonging to the general picture of tabes. The patients have sudden, violent gastric pain, radiating in all directions, and in the severe attacks accompanied by nausea, vomiting and great prostration. Like the pain attacks, these crises may last for hours, or a few days and then disappear for weeks or months to recur at irregular intervals. They disappear as rapidly as they come, and quite analogous to the lancinating pains, may be early or late symptoms. Certain cases of tabes begin with such crises. Such a beginning is frequently not recognized and has often led to an illy considered laparotomy.

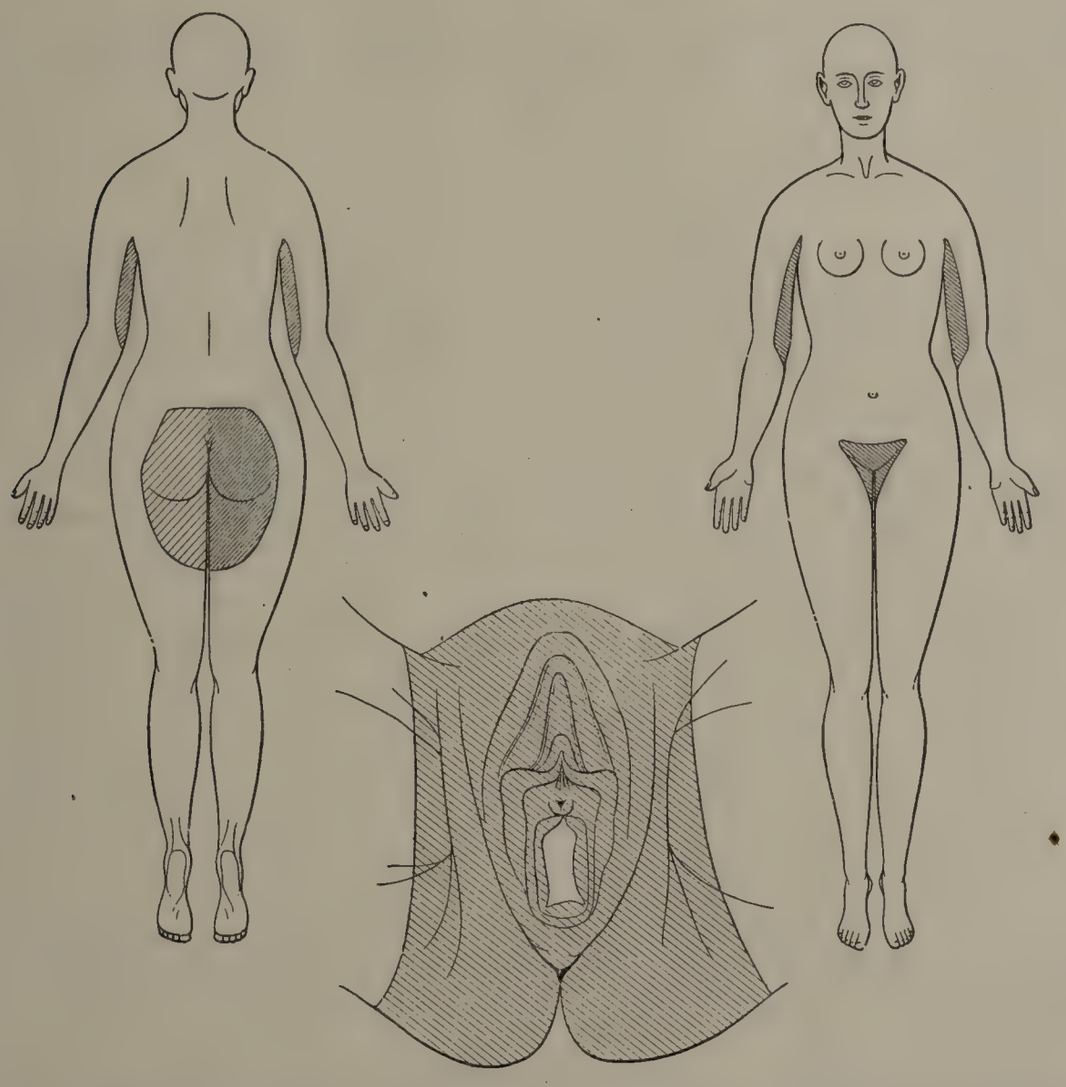


FIG. 415.—Tabes involving the cauda equina, showing the radicular distribution of the sensory disturbances. Tactile pain and thermal sensibility were involved. The patient first had pains and sphincter disturbances. The second dorsal root was also slightly involved, hence the sensory changes in the arms. (Dejerine.)

Similar crises affecting other internal organs have the same etiology and course. Thus there are intestinal colics with diarrhea, rectal pains with tenesmus and diarrhea, vesical crises with strangury, urethral crises, renal colic-like attacks, testicular crises, vulvo-vaginal crises, laryngeal and diaphragmatic crises with cyanosis and dyspnea. pharyngeal crises with obstinate hiccough, bronchial crises with cough, cardiac with angina-like attacks. Sneezing attacks have been described as an initial tabetic sign.

These crises are recognized to have as an essential component a loss of balance in the sympathetic-parasympathetic tonus mechanism. Jelliffe's cases of disturbance of this mechanism as seen in hypothyroid states is of interest in the analysis of these crises, as well as of the osteoarthropathies. Eppinger first called attention to these possibilities and Jelliffe and Pötzl have spoken of the further possibilities of the added influence of unconscious emotional factors as determining the localization of a symptom in accordance with the held-up craving. Kempf has elaborated this theme in his Psychopathology.[1]

Sensory Involvement.—As a result of the implication of the meninges of the sensory roots, alterations in the sensory functions take place. The pathological process in some is so gradual or mild as not to give rise to pain, and in many, initial paresthesiæ may precede the pains,

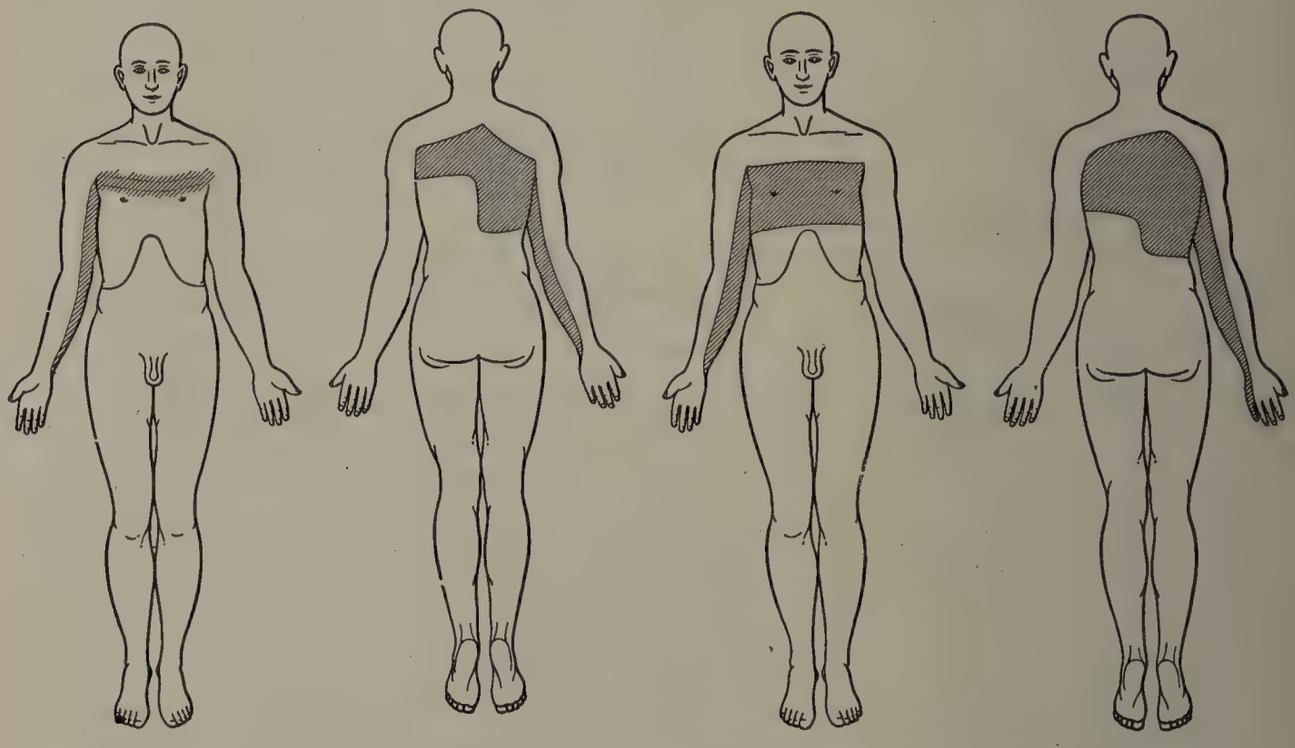


FIG. 416 FIG. 417 FIG. 418 FIG. 419

FIGS. 416, 417, 418 and 419.—Radicular distribution of sensory disturbances in tables; Figs. 416 and 417 represent the distribution of tactile anesthesia; Figs. 418 and 419 that of loss of pain and temperature sense. (Dejerine.)

but more often the same pathological process gives rise to both. Tingling, numbness, crawling sensations, flashes of hot and cold, slight benumbing of the tactile sensibility, causing the sensation of wearing a glove, or walking upon a textile are the usual forms. They may be expected almost anywhere from the region of the trigeminus through any sensory cervical nerve branch to the tips of the toes, although the ulnar region seems a site of special predilection. Occasionally they cause the peculiar girdle-band sensation, at one time considered so characteristic of tabes. A whole limb may be involved, but under any condition the tendency for the sensory disturbances is to show a radicular distribution (Dejerine) (Figs. 415–419).

Increasing sensitiveness is also frequent, so that the patient dreads the cold, or draughts, or sudden shocks, or the clothing, not only on

[1] Kempf: Psychopathology, Mosby, St. Louis.

account of the abnormal sensitiveness, but because these may bring on the severer pains. Tactile anesthesia follows this and is a direct resultant of the degenerative process in the root area. These are also irregularly distributed.

Other sensory functions also become involved. The *pain sense* may become lost in irregular areas. Retardation in the carrying of pain impulses is present at times. Complete loss is a sign of a complete lesion. Maloney gives three signs of a partial lesion: 1, a raised sensory threshold; 2, delayed perception; and 3, a sensation of imperfect contact. There may also be numbing or loss of *heat* and *cold* sensibility. Bony sensibility as tested by the tuning-fork also may be absent and shows irregular distribution. In general, deep sensibility is more profoundly affected than epicritic sensibility. Maloney states that this is due to the fact that the vegetative nervous system is largely concerned in deep sensibility and is the sensory system mainly affected in tabes.[1]

Since, as has been pointed out, Ranson[2] identifies the cutaneous "protopathic" system of Head with certain unmyelinated fibers of the posterior spinal roots, this conception, as maintained by Maloney, is probably the correct one. In 1912 Ranson published his discovery of unmyelinated fibers in the dorsal roots. These are mostly cutaneous nerves, and they practically all enter the cord in bundles which lie laterally to the myelinated fibers of the same root. The former are called by Ranson the "lateral division" of the dorsal root. A much smaller number of myelinated fibers is found also in this lateral division. All the neurons of the lateral division run up or down, in Lissauer's tract, a very short distance—usually less than a segment. That is to say, these fibers run into the gray matter at or near the level at which they enter the cord. Their intraspinal course suggests at once that they are the fibers of pain and temperature sensations, since it is known that the afferent impulses underlying these sensations pass through the gray matter as soon as they reach the cord.

Ataxia.—The most prominent sign of the sensory involvement just noted is seen in the gradually (sometimes suddenly) developing ataxia. The fibers conducting the impulses from the joints and the muscles to the chief organ for their coördination, the cerebellum, are degenerating, and there results an imperfect knowledge of the position of the joints and of the states of muscular tension necessary to the proper performance of motor functions. There results a hypotonus and swaying of the arms in the finger-nose test and finger-finger test, and of the legs in the knee-heel test, and in walking. The patients are partially or completely unaware of where their limbs may be, and are unable to control the same, save to a certain degree through other avenues, namely, the eyes. According to Maloney this unawareness

[1] Maloney: Locomotor Ataxia, New York, 1917. Decourt: Ataxie aigues, Loc. cit., 1927. Gennerich: Ztschr. f. d. ges. Neurol. u. Psychiat., **30**, 545. Spielmeyer: Ztschr. f. d. ges. Neurol. u. Psychiat., **84**, 257; **91**, 627. Pandy: Ztschr. f. d. ges. Neurol. u. Psychiat., **89**, 589.

[2] Am. Jour. Physiol., 1916, **40**, 571.

is due to the suppression by the pysche of the feeble, imperfect and delayed postural images which arise from the musculature in the area of the affected nerve roots and which owing to the misleading nature of their information are suppressed in favor of vision. Hence with closed eyes all of these signs of ataxia are markedly increased. With this great uncertainty of movement there is no muscular paralysis, and little loss of muscular strength, save as the patient generally becomes weaker.

Gait disturbances are accompaniments of the ataxia, and are extremely characteristic. They were described by Schermer as early as 1819. In the early stages the patient notes a difficulty in going up and down stairs, or finds himself insecure on uneven surfaces. He stumbles and at times falls. At night he finds it more difficult to get about, and he soon notices that he must keep his eyes glued to his legs or the surface on which he is walking if he is to be able to control them. Later he must walk with a cane, and his legs are thrust somewhat wider apart, are thrown somewhat irregularly forward, and are then brought to the ground with a sharp stamp, the knee being stiffened or even bent slightly backward—overextended—at times so much so as to cause him to fall.

His step becomes quicker and more stamping, until he reaches a point where, unless he has trained himself to walk anew, he becomes bed-ridden. Maloney has demonstrated that the preataxic, ataxic, and bed-ridden stages are not so much stages of structural demolition as stages of mental deterioration, stages of loss of cerebral control over voluntary movement. The manner of rising from a chair or sitting down soon becomes very characteristic. The ataxia in the upper extremities, which is usually less than that in the lower, affects the patient's writing, the buttoning of the clothes, his dressing, etc. His hands are constantly moving—one finger and then another is raised or lowered, or the wrist turned. Oppenheim has called particular attention to these spontaneous movements, which closely resemble a static ataxia, and are found throughout the body.

Other muscles naturally may be involved in the ataxia, particularly those of the face, mouth, tongue, larynx, pharynx in which case speech, singing, swallowing, etc., are affected. Many patients die of aspiration pneumonia through ataxia in the swallowing apparatus.

Romberg's sign is another result of the loss of position sense. It may be an early sign, but is more apt to develop later in the disease. Many patients without well-developed Romberg are unable to balance themselves on one foot, and further, a mild Romberg may be more readily demonstrated by having the patient bend slightly forward.

Tendon Reflexes.—Westphal first emphasized the importance of the diminution or loss of the tendon reflexes—notably of the knee-jerk and the Achilles reflex. These belong among the initial symptoms in the larger number of cases. The knee-jerk may be first diminished on one side, best demonstrable by the Jendrassik method, or lost, and

this for years, perhaps, before the development of a complete Westphal phenomenon. The Achilles-jerk is lost in a similar manner and not infrequently even before the loss of the knee-jerk (Babinski method).

These tendon-reflex changes are all referable to the degenerations in the root zones and sensory columns.

Cranial Nerve Involvement.—Any one or all of the cranial nerves may be implicated. Loss of smell is rare.

Pupillary Reflexes.—Here a striking phenomenon is observed. Patients with tabes—as with many other syphilitic affections of the medullary or midbrain region—show a diminution or loss of the pupillary light reflexes, without any loss of the reflex of convergence or of accommodation. This is the *Argyll-Robertson* phenomenon. It is

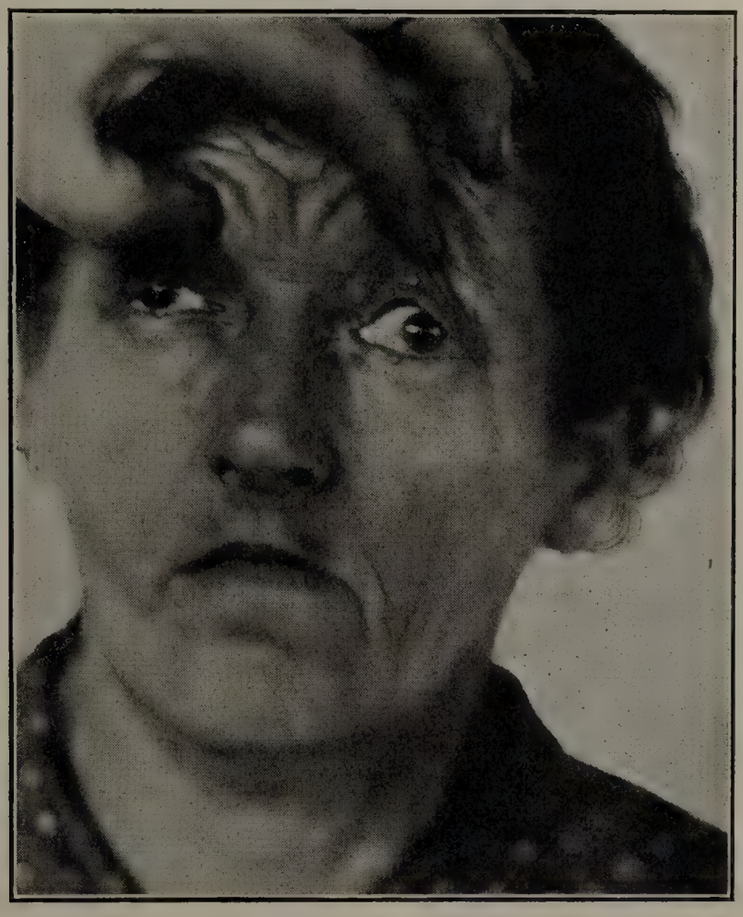


FIG. 420.—Tabes. Third and sixth nerve palsy. (Jelliffe.)

present in over 60 per cent. of the cases, and may be present for many years without other symptoms. The pupils are apt to be at first irregular in size, and also not infrequently, in shape. The light reaction is at first less prompt—usually in one eye before the other—later both eyes are involved. Myosis in marked degree is then apt to develop. Loss of the consensual light reflex is one of the earliest signs of this pupillary change (Weiler). The sympathetic dilatation of the pupils is also soon diminished or lost.

Optic nerve changes are frequent and may occur early. There is an irregular gray atrophy, with narrowing of the macular vessels and gradual narrowing of the visual field, first for colors and then blindness, partial or complete, with advanced optic atrophy.

Ocular Palsies.—These very frequently belong to the early symptomatology and are often fleeting. Persisting ocular palsies are more often encountered in the later stages of the disease. These palsies in tabes occur quite irregularly and show themselves as atonic ptoses, irregular diplopias—from fourth or sixth or third nerve affections—and rarely show signs of a complete ophthalmoplegia, internal or external.

The *trigeminus* involvement may show not only neuritic pains as already noted, but also trophic disturbances of the cornea, herpes, irregular development of the bony parts, etc. The motor root may also be partly paralyzed.

Facial palsy occurs, but not often.

The *acoustic* is occasionally involved in both cochlear and vestibular branches, producing unilateral or bilateral deafness, or vestibular nystagmus, Ménière-like crises, etc. (Bonnier's syndrome.)

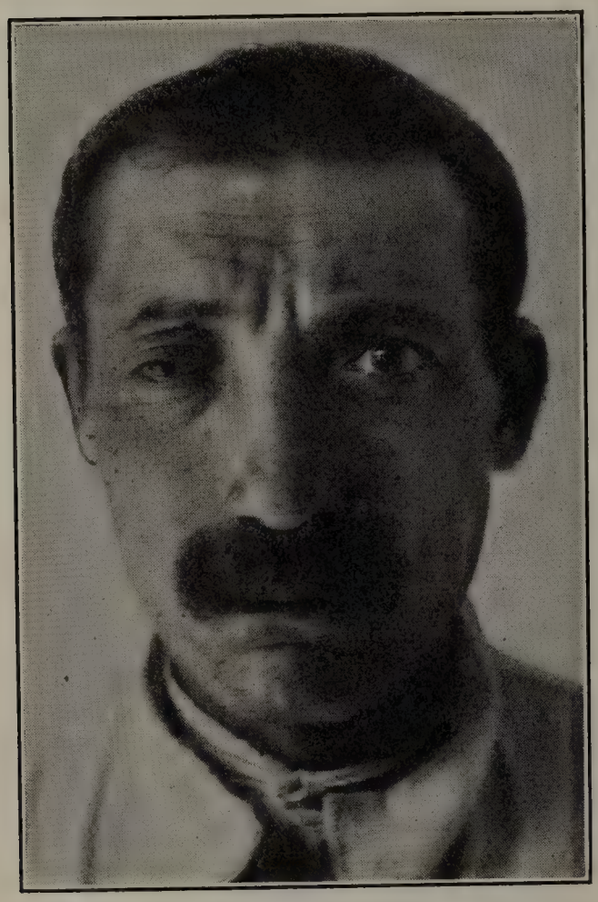


Fig. 421.—Tabes. Third nerve palsy.

The *vagus* and *accessory* nerves



Fig. 422.—Tabetic arthropathy of the rib. (Jelliffe.)

are frequently implicated, causing crises already noted, and also slowing of the pulse, dyspnea, tachypnea, etc. Taste is not infrequently lost. Bulbar palsies are frequent.

Visceral Symptoms.—The bladder is usually involved at some stage of the disorder. The crises have been referred to. Weakness of the bladder is the most usual occurrence. Cystitis usually develops and is a cause of general weakness. Incontinence is not infrequent. Constipation is also frequent, but fecal incontinence not common. Loss or increase of erotic desire is an early sign; impotence is common; continuous priapism unusual.

Trophic Symptoms.—These may involve any of the tissues of the body, but particularly skin and bones. *Arthropathies*[1] are very frequent,

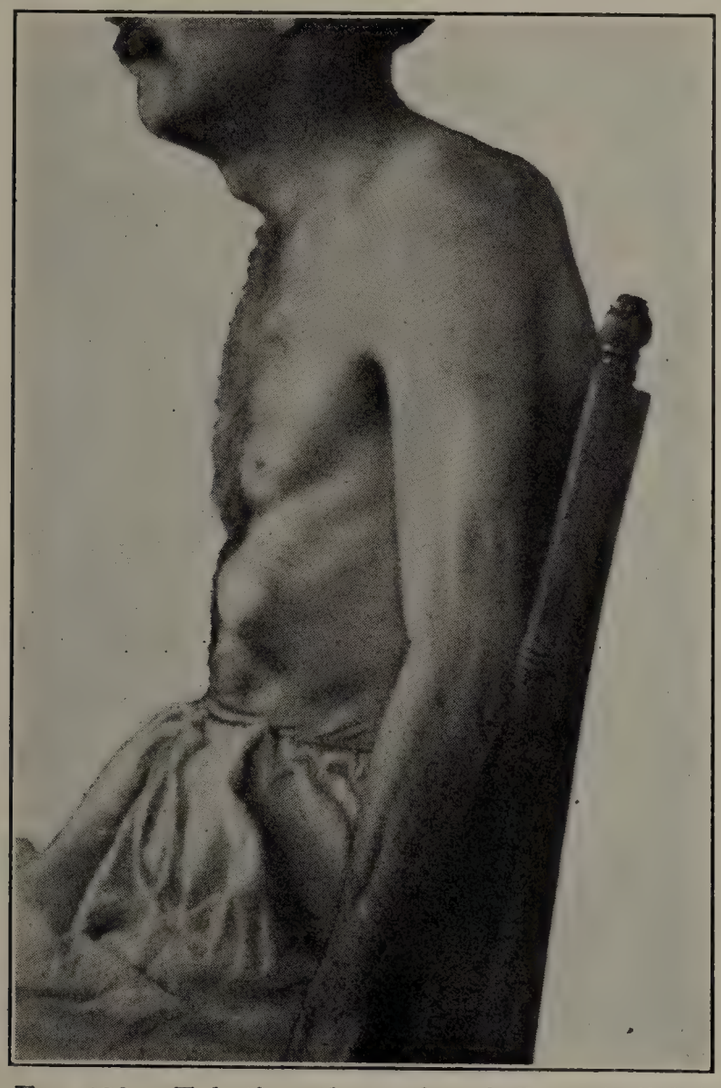


FIG. 423.—Tabetic arthropathy of ribs. (Jelliffe)



FIG. 424.—Tabetic arthropathy in the wrist. (Jelliffe.)

[1] Flatau: Die trophische Störungen bei der Tabes Dorsalis, Berlin Thèsis, 1888. Barré: Les osteoarthopathies du Tabes, Paris, 1912. Also see Mme. Dejerine et Ceillier on the osteoarthropathies in parplegics from spinal cord injury, loc. cit. Grassheim (Ztschr. f. d. ges. Neurol. u. Psychiat., 1921, **72**, 119) has collected the recent literature, but gets no further than general statements relative to the participation of hormones. Recent literature in Quarterly Cumulative Index Medicus.

usually occurring after the disorder is well advanced, not infrequently as an early symptom. The knee-joint is the joint of special predilection. The arthropathies usually develop with surprising rapidity—with

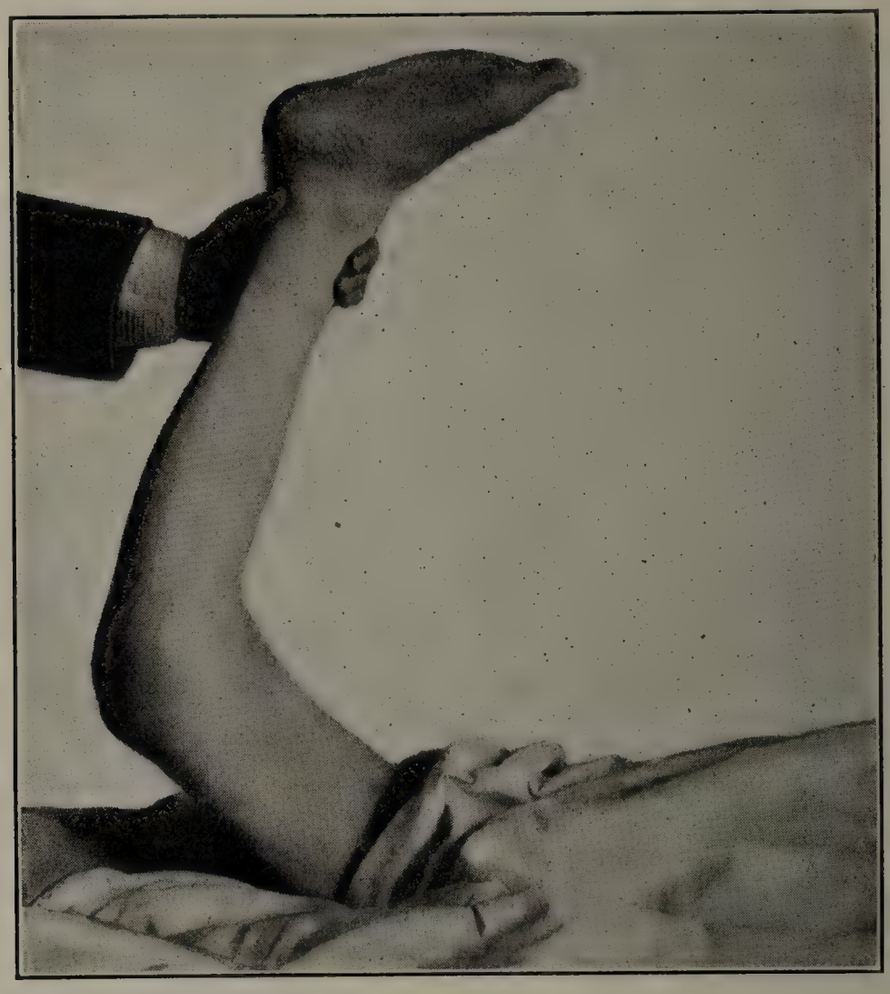


Fig. 425.—Tabetic arthropathy with destruction of the joint and hypotonus. (Jelliffe.)



Fig. 426.—Charcot joint. (Jelliffe.)

edema and swelling but none of the usual signs of a rheumatic joint; then new bone formations take place, with or without subluxation. Almost any joint may be affected, even the jaw.

Fragility of the bones is a further complication. Perforating ulcers of the feet constitute another trophic disorder. These trophic changes constitute further evidence to the fact of the implication of the vegetative (protopathic) fibers in tabetic pathology.

Muscular Atrophies.—Dejerine[1] described these in 1889. An initial neuritis may give rise to an early muscular atrophy, but atrophies are not common.[2]

Blood Serum and Cerebrospinal Fluid.—The Wassermann reaction of the blood serum is positive in most of the cases of tabes. The cerebrospinal fluid is positive in from 60 to 80 per cent., and Plaut is of the opinion that it will be found to be more often so with improved technic. Nonne and Hauptmann, by using larger quantities of serum, have established this. It seems to be less often positive than in paresis —which, in view of the opinion held concerning the identity of the two disorders is food for reflection regarding the local reaction of the brain and cord proper.

There is a marked lymphocytosis of the fluid, which is usually found in the early stages of the disorder, and is an index of the meningitic syphilosis usually found.

The colloidal-gold reaction may give curves resembling paresis, especially when there is some mental extension, but as a rule the curve is not classical.

Course and Variation.—Tabes is essentially a chronic disorder, usually mildly progressive, developing fully after many years. In a small number of cases it runs an acute course, causing death within a few years. Acute ataxias of more benign course (Guillain) are to be looked for as well.[3] In others it is stationary and in this sense gets well.

Duchenne (1858–1859), in his memorable description, made a division into three stages, which have been somewhat modified. The periods usually considered at the present time are as follows: (1) Prodromal or preataxic stage; (2) ataxic period; (3) paralytic period.

Such a division is of arbitrary value only; no two cases are exactly alike. The preataxic stage may last many years, or there may be none, ataxia and paralysis developing with extreme rapidity. The symptoms are so many, and the times of their appearance so variable, that a so-called typical course is the exception rather than the rule, yet a not unusual course is one extending over about ten years, with two or three years of pains, with or without crises, then the gradual development of the loss of knee-jerks; Argyll-Robertson pupils, gradually increasing difficulty in walking, worse in the dark, Romberg—then the patients are confined to bed, and then the stage of paralysis and atrophies. Maloney correctly denies the existence of the paralytic stage and rightly calls it the surrender stage, the stage in which the ataxic will not pay the tax in effort which movement demands.

[1] Rev. de Mèd., 1889.

[2] See Lapinsky: Arch. f. Psych., vol. **40, 42.** Lakacs: Zeit. f. d. ges. Neur. u. Psych., 1914, **24.** Pitte: Zeit. f. d. ges. Neur. u. Psych., 1922, **76,** 275.

[3] Decourt, J.: l'Ataxie aigue tabetique, Paris, Doin, 1927.

Death results from the disease itself, or from complicating disorder —bulbar accidents causing pneumonia, laryngeal choking, cardiac syncopes, kidney complications, very often; intercurrent disease such as tuberculosis, in large part, pneumonia, typhoid, and erysipelas in smaller percentages.

Prognosis is always most sinister. Stationary cases are known, but a question concerning diagnosis may be raised respecting the cases in the older literature. With the newer objective Wassermann and cytological symptoms it is to be seen whether such stationary cases exist. Since such are found as paretics, it is not improbable that the same will hold true for tabes.

The patients with severe bladder complications usually do badly.

The duration of life has varied from six months to thirty and more years. The general average runs between ten to fifteen years.

Forms.—Certain trends are worthy of special mention as forms these are:

1. *Juvenile tabes*, like juvenile paresis, occurs in children from five to ten years of age, or in young adults apparently up to about twenty-five years. The higher age incidence is rare. The pupillary signs are early, and the bladder is soon involved. Ataxia and paralysis then develop. The objective serological and cytological changes are usually positive—the number of cases examined is as yet too small to permit wide generalizations, but syphilis of the parents is an essential.

2. *Late Tabes.*—The appearance of the disorder after the age of fifty is rare, yet cases are recorded in which the initial signs have come on as late as seventy years. One case is on record of infection at twenty, tabes at fifty-nine. After all it is mostly a question of when infection takes place—since cases of infection at eighty or over are known, it is practically impossible to determine the tabes age limit. No noteworthy symptomatic variations are known in late tabes.

3. *Rapid Tabes.*—Here the disease advances very rapidly, rendering the patient incapacitated in a few months, with death as a result of of the paralyses. The paralyses and atrophies occupy the foreground in the picture, the ataxias being less prominent. The pupillary signs are present. Death may take place within six months. Acute benign cases are occasionally seen. (Guillain, Decourt.)

4. *Slow Cases.*—These are the more usual cases already described.

5. *Stationary or Benign Cases.*—A particular type in which blindness comes on early and which follows a benign course was first described in 1881 by Benedikt. Dejerine and Martin (*Thése de Berne*, 1890) called attention to the fact that it is rare to find a case of tabes beginning with blindness that advances to the second stage.

Furthermore, amaurotic tabetic cases seem to have fewer pains. The pathological features are the same as in other cases, save as to extension, and the explanation of this variation is difficult to find. A certain diminution in the general symptoms in amaurotic tabes has been observed.[1]

[1] Maloney: Jour. of Nerv. and Ment. Dis., 1914, vol. **41.**

Diagnosis.—Little difficulty exists after the development of the Argyll-Robertson pupil, lost knee-jerks, ataxias and Romberg. Practically the only differential at this stage is a polyneuritic process, principally of alcoholic, rarely of diabetic origin. Here the pupillary disturbances are less in evidence, but can occur, and if present there are usually more grave cerebral symptoms and a diagnosis of taboparesis is more in question. The serological and cytological results determine a diagnosis almost at once. Still the most difficult cases to differentiate are those of alcoholism complicated with syphilis. Where the alcoholism causes a pseudotabetic picture and the syphilis gives its serological and cytological findings with slight meningitis affection as its only spinal or cerebral concomitant, the cases are diagnosed only with the greatest of difficulty.

Other toxic pseudotabes offer few difficulties—such as those due to ergot, diabetes, lead, pernicious anemia in submyxedemas, and the infectious toxemias. Here the pupillary signs are absent for the most part and the serum and spinal fluid normal.

Certain cerebellar atrophies and new growths cause symptoms closely resembling those of tabes, but the gait is more widely swaying, the individual movements have less ataxia, the absent knee-jerks, and Argyll-Robertson are wanting; and there are, moreover, to be found the definite signs of cerebellar involvement in the nystagmus and its alterations to the Bárány vestibular tests. Normal serum or cord findings are to be expected.

Multiple sclerosis can occasionally cause a tabetic syndrome if a patch should involve the sensory neurons but here the other symptoms, the nystagmus, the signs of spasticity, masked by the hypotonus, Babinski phenomena, etc., should afford the clue. Rare sclerotic patches in the pons and midbrain regions have caused unilateral Argyll-Robertson pupillary pictures.

Syringomyelia occasionally develops with an initial tabetic picture, but soon the classical dissociation shows itself, and permits a diagnosis. The blood and spinal fluid findings are also to be reckoned with.

Hysteria as an astasia abasia occasionally causes difficulty, but here careful examination can exclude the entire tabetic symptomatology.

Pseudotabetic neuritides and eye signs, *i. e.*, irregular pupils, gastric crises, may occur in masked myxedema (hypothyroidism). (Jelliffe, loc. cit.)

Pathology and Pathogenesis.—Both Ollivier d'Angiers and Cruveilhier gave descriptions of the general gross anatomy and both recognized the sclerosis of the posterior columns. Todd associated the sclerosis with the ataxia. Bourdon and Luys (1861) called attention to the relation of the sclerosis of the posterior roots and the posterior column sclerosis, since which time the development of knowledge concerning the pathological processes in tabes have been most actively studied, although be it said, without yet arriving at general uniformity.

Seen with the naked eye, the cord is usually markedly atrophied,

hence the origin of the old term consumption (tabes); the posterior roots are atrophied, sometimes more marked in one region than another. The diminution in volume of the cord also varies in places, being more pronounced, as a rule, in the dorsal and sacrolumbar regions than the cervical, and, as Cruveilhier noted, the atrophy preponderates markedly in the posterior columns. The pia and arachnoid are somewhat swollen but translucent with slight opalescences or cloudiness. Meningeal involvement is very frequent; a fact brought out more in recent years, and in strict conformity with the findings of the cellular contents in the cerebrospinal fluid.

Throughout the entire length of the cord one finds a graying discoloration, the margins are slightly sunken below the normal level, and the discolored areas are harder to the touch. The atrophy seems to cease with the sensory medullary nuclei—save in those cases where cranial nerves are markedly affected when irregular atrophies are encountered in the upper sensory neurons.

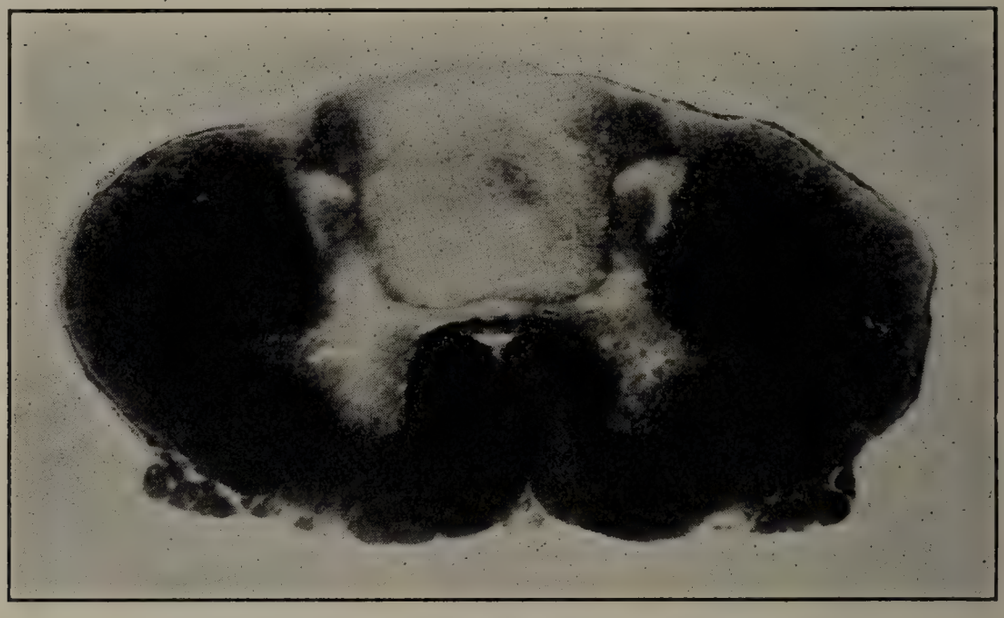


Fig. 427.—Tabetic changes in cord in paresis.

Histologically the picture is fairly uniform. There is a mild inflammatory thickening of the pia and arachnoid with lymphocyte and plasma-cell infiltrates. This leptomeningitis varies considerably in its localization, and in its intensity, and the vessels are not infrequently involved. The whole process closely approaches that of a syphilitic meningitis, but is less intense, and is rarely accompanied by the presence of spirochetes.

The posterior roots are irregularly atrophied; the sacral and lumbar roots may not show a single unchanged fiber, whereas the cervical roots are less involved, save in the case of cervical tabes, where the reverse holds true.

The posterior columns show the most uniform lesions. These are greatly diminished in volume. There is a gradual degeneration, often, however, more intense on one side than the other. The atrophy is not universal, for many fibers are intact. These are usually collaterals,

originating within the cord itself. The direct fibers in the columns of Goll and Burdach, from the degenerated posterior roots, are those chiefly involved. A great deal of variability exists as to the respective distribution of the atrophic degenerated fibers, but such bear a direct proportion to the distribution of the degenerated posterior roots. Certain portions of the cord escape, due to anatomical reasons; these are more particularly the commissural zones, Flechsig's centrum ovale, Gombault and Phillippe's triangle in the lumbosacral region, the cornucommissural zones, Schultze's comma tract and the fascicles of Hoche. (See Plate XII.)

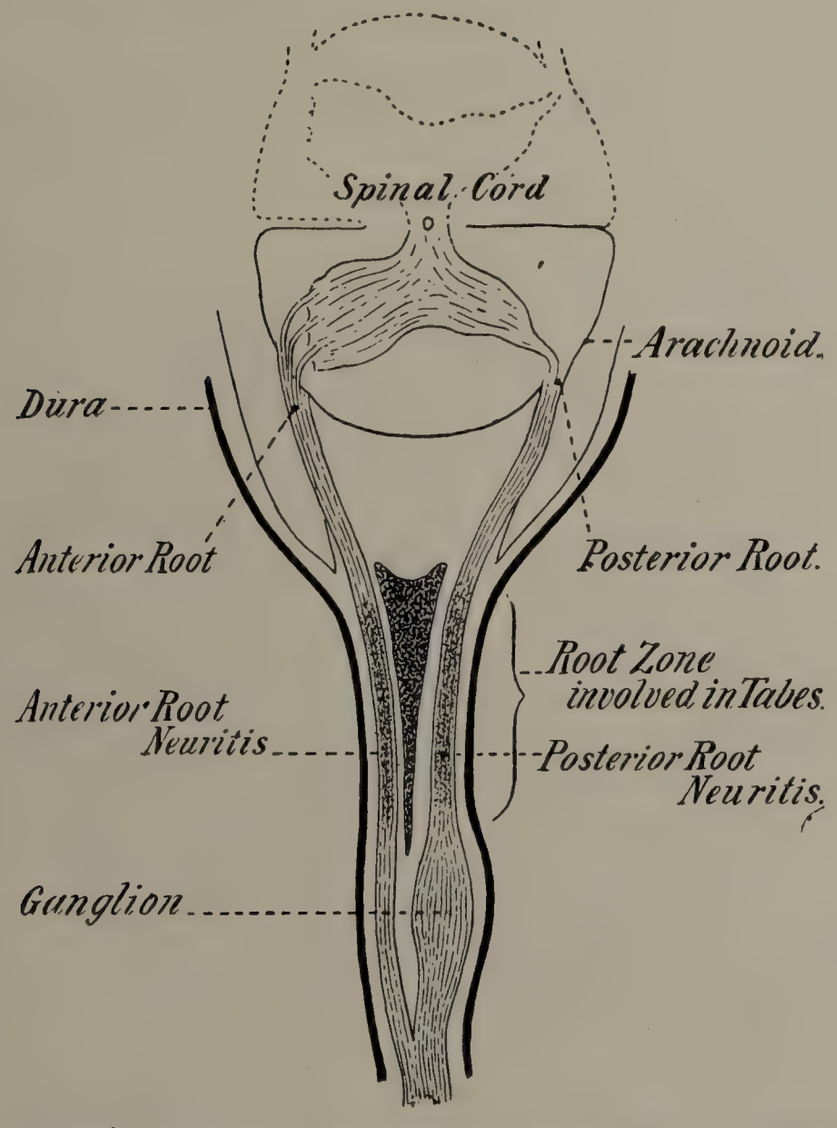


FIG. 428.—Nageotte's scheme representing the location of the chief lesion of tabes in the posterior root zone.

As a result of the disappearance of the fibers a secondary neuroglia infiltration takes place. This consists of glia with fine prolongations, and also spider cells. In the cases of taboparesis one finds lymphocytes and plasma cells within the cord substance. With a limited tabetic process they are not usually encountered.[1] Many ganglion cells of the posterior horns show degenerative changes. They are

[1] Alzheimer: Nissl's Arb., **1**, 14. See Spielmeyer: Paralyse, Tabes and Schlafkrankheit, Ergebn. d. Neurol. u. Psychiat., 1911; Histopathologie des Nervensystems, 1922; studies of Jahnel, Jacob: Ztschr. f. d. ges. Neurol. u. Psychiat., vols. **50-70**. Spielmeyer: Ztschr. f. d. ges. Neurol. u. Psychiat., 1923, **84**, 257. Pandy: Loc. cit.

diminished in size, there is definite chromatophilia, or vasculization and other signs of degeneration. The cells of the column of Clarke are diseased only to a slight extent.

In tabes with cranial nerve signs the degeneration can be traced in the medulla and pons with secondary atrophy of the sensory nuclei. Atrophy of the optic nerve is not infrequent.

Jendrassik, Schaffer, Epstein and Krauss have also shown that the brain cortex is not uninvolved even in typical tabes. There is a diminution in the nerve fibers, and alterations in the vessels and pia —there may be lymphocyte and plasma-cell infiltration even in the absence of pronounced mental symptoms. Cerebellar degenerations are also known. The anterior horn cells do not always escape. In the patients with pronounced muscular atrophy many motor cells of the anterior horns are found degenerated. Similar changes are known where there are bulbar, laryngeal or other cranial nerve palsies. The cerebrospinal fluid also shows the effects of the mild inflammatory process by its increased lymphocytosis; a count of over 10 cells to the cubic centimeter (Fuchs and Rosenthal chamber) is to be regarded as positive, 50 to 100 are not unusual. Plasma cells are also to be found.

Serologically a positive Wassermann is to be expected, although here the number of positive results would seem to fall below that obtained in paresis. Possibly paresis indicates a more acute process.

Nonne phase I reaction is also frequently positive.

The posterior ganglia are also affected in many cases, although not uniformly. There is atrophy and destruction of the cells, and proliferative inflammatory exudates of the capsule. The changes are not sufficiently constant to permit one to assume that the primary part of the disorder is located in the posterior ganglia.

The peripheral nerves are also frequently found degenerated; Nonne says constantly; thus showing the complete degeneration of the sensory neuron, central as well as peripheral.

Vegetative System.—This shares in the general destruction and shows particularly in the regions involved by gastric or other visceral crises.

Pathogenesis.—Unanimity of opinion has not yet been reached. The various hypotheses evoked have upheld vascular (meningeal), medullary, radicular and neuritic theories, not to mention the idea of a primary system intoxication. The general tendency is to regard as *fundamental* a primary syphilis involving particularly the posterior roots—a modified syphilitic radiculitis as the most constant feature in the pathogenesis. The process is one of chronic specific hyperallergic reaction in which there is (1) an involvement of the posterior radicular fibers and of the peripheral nerves; (2) an extension to the vegetative nervous system fibers both sympathetic and autonomic; (3) to the motor nerve system.

Concerning the nature of this hyperallergy, if present, little is known.[1]

[1] See Head: Brain, 1913. Dind: Rev. d'Urologie, 1914.

7. **Syphilitic Meningomyelitis.**—Nonne has devoted a large portion of his noted monograph to a consideration of the lesions of syphilis of the spinal cord and its membranes. This is a general indication of their extreme frequency, yet most patients showing syphilitic lesions of the cord also show signs in the brain or its meninges. They are nearly all examples of cerebrospinal syphilis. For practical purposes, however, it has been found of value to arbitrarily divide this large conglomeration and discuss it under two captions: cerebral syphilis and spinal syphilis or meningomyelitis. This means simply that one is dealing with cerebrospinal syphilis with predominant cerebral and minor spinal symptoms on the one hand, or with predominant spinal and nerve root, with less prominent cerebral signs on the other. It again seems advisable to accentuate the purely pragmatic character of all such classifications.

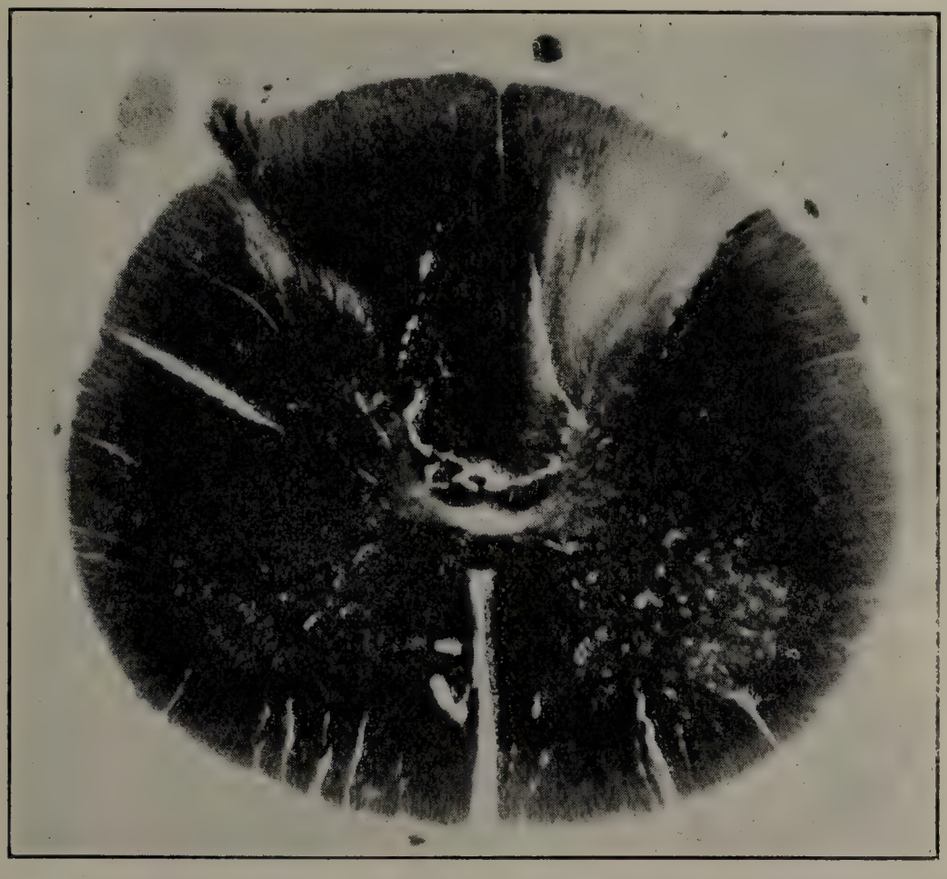


FIG. 429.—Meningomyelitis. Radiculitis. Degeneration of Burdach's columns.

Symptoms.—In considering meningomyelitis as a unit, further emphasis may be put upon separable symptom groups. Within this conglomeration again clinical neurology shows four fairly clear tendencies:

1. Syndromes due to pronounced meningeal implication.
2. Syndromes due to root and cauda equina disease. Radiculitis and neuritis. Syphilitic osteoarthritis of the spine is a frequent factor in these.
3. Myelitic syndromes due to indiscriminate transverse disease.
4. Syndromes of less extensive transverse lesions and fiber-tract isolation. System syndromes.

A combination of all would make a complete meningomyelic syndrome. This is a not unusual picture in a rapidly developing case; in its more chronic course the emphasis seems to be laid upon one or another of the just mentioned groupings.

These are characteristic syndromes of the early and secondary stages of syphilis. The syndromes may develop within a few months after infection, or only come on after many years. In the former case the acute myelitic changes are frequent, also root lesions (many neuralgias, sciatica, etc.). The later developing cases show more the systemic lesions and gradually advancing meningopathies (later secondary meningitis) with compression (spastic) phenomena.

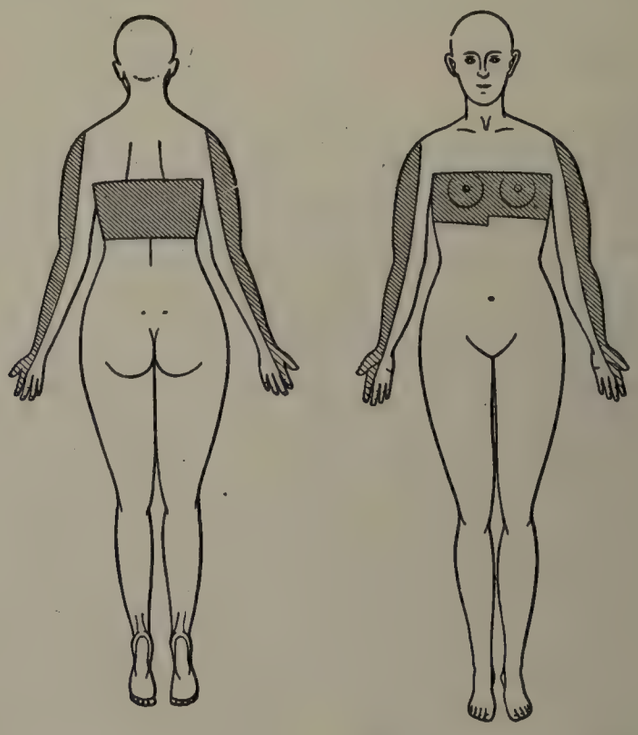


FIG. 430.—Radicular distribution of sensory loss in tabes, particularly to tactile pain and thermal sensibility. (Dejerine.)

In all one expects to obtain a positive Wassermann; cerebrospinal fluid Wassermann may be negative in early cases, but usually becomes positive; pleocytosis is frequent—often the cell count being very high, always indicating the grade of meningeal involvement. The lymphocytes are sometimes found before the stage of roseola; are abundant in the active secondary stages and less frequent in the tertiary stages of a meningomyelitis. The protein content varies considerably.

1. *Meningeal Syndromes.*—Severe pains are signs of meningeal involvement. They shoot across the shoulder-blade, in the neck, across the hips, dart down the arms or legs and cause a stiffness of the neck, the shoulders and the thighs. The spinal column is usually sensitive to pressure, and to percussion, and local intensities may show both these signs, and also the peripheral signs of a definite skin zone localization.

Pain is frequently preceded by paresthesiæ, like the crawling of ants, numbness and coldness. There is a gradual increase in the

reflex excitability of the cord, due to pressure and evidenced by increased knee-jerks, possible Babinski sign, Oppenheim or Chaddock signs. When pressure is exerted in the sacral segments bladder and rectal disturbances are frequent.

With chronic meningeal thickening these pressure symptoms increase markedly and spastic paretic phenomena augment, especially when gummata add their special pressures. Gummata may give rise to a "cord tumor" syndrome.

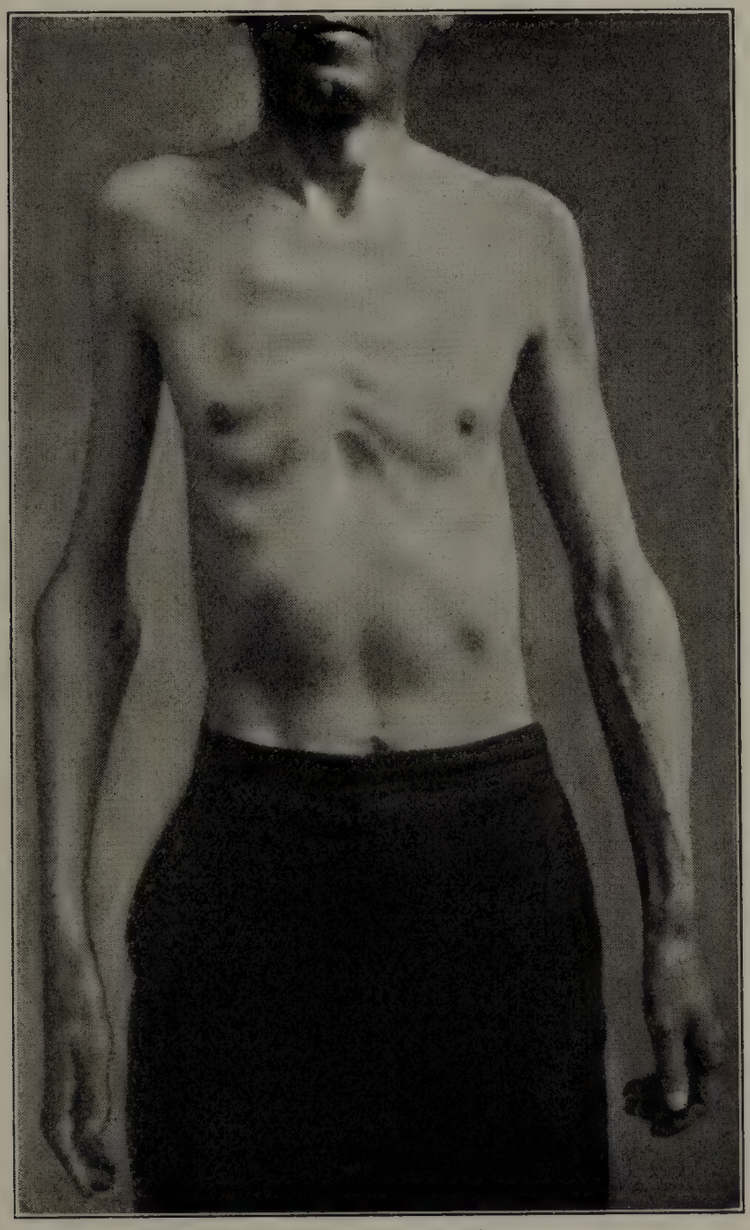


FIG. 431.—Syphilitic meningomyelitis with radicular symptoms, atrophy, etc. (Jelliffe.)

2. *Root Syndromes.*—Here pain is frequent and neuritic atrophies appear. Sensory losses of a root distribution are in evidence. Many show the characteristic reversal of epicritic touch loss being less extensive than protopathic (vegetative fibers) pain loss as pointed out by Head as characteristic of radicular localizations.

The atrophy of the muscles also follows the radicular distribution.

Many obstinate neuralgias are due to syphilitic radicular disease. Possibly one-half of the sciaticas are of this nature. Dejerine has put them as high as 80 per cent. in Paris. A very large proportion of

the brachial neuralgias, so long looked upon as rheumatic or gouty, or what not, are due to a syphilitic root meningitis.

Neuritic muscular atrophy from pressure on the anterior roots is further complicated by pressure on the anterior horns by the thickened meninges. Thus very anomalous atrophies result. When occurring in the eighth cervical and first dorsal region one obtains classical Klumpke paralysis with dilatation of the pupil, and narrowing of the palpebral fissure of the affected side. Lower localizations result in intercostal palsies, back-muscle atrophies, hip girdle, thigh or cauda lesions. In this latter situation striking dissociations are obtained, as in the upper arm region, and radicular sensory disturbances and lost reflexes are the rule with atrophies.



FIG. 432.—Pachymeningitis hypertrophica cervicalis. (White.)

3. *Myelitic Syndromes.*—These indicate the complete involvement of the cord and also point to intraspinal vascular disease, rather than to a meningeal lesion. Complete flaccid palsy (paraplegia) is the usual result. This is combined with sensory loss as well. The completeness of the sensory loss varies considerably and indicates the severity of the lesion. Absence of a lymphocytosis points to a purely vascular and usually focal lesion within the cord. The bladder and rectal functions are implicated as well.

In the regressive stage an increase in spasticity marks the subsidence of the inflammatory reaction and many anomalous syndrome mixtures result. This phase of meningomyelitis offers abundant

opportunity for very heterogeneous syndromes. A Brown-Séquard complex, poliomyelitis, tabetic syndrome with atrophy, amyotrophic lateral sclerosis syndrome—these are but a few of the possible combinations.

4. *System Syndromes.*—These occur not so much as residuals of the previous myelitic changes, nor as due to meningeal compressions, but represent disease in or about the long motor tracts, tractus corticospinalis, especially. They give rise to the forms of *primary lateral sclerosis* (Erb), some combined scleroses and particularly to clinical pictures closely resembling multiple sclerosis. Combined disease of the posterior and lateral columns is quite apt to be syphilitic.

Special localization of one or more of these forms of meningeal syphilis gives rise to the special forms of hypertrophic cervical pachymeningitis, which have been described by Joffroy and Charcot, and to Kahler's disease.

The anterior horns may be predominantly involved usually, however, as a result of a transverse myelitis (Nonne) giving the picture of a chronic anterior poliomyelitis.

In *hypertrophic cervical pachymeningitis* an enormous thickening of the meninges is found with or without gummata and located in the cervical region. Here root and compression symptoms are present. Pains in the neck and shoulder, stiffness of the cervical spine, shooting pains down the arms. Sensory loss may then show, particularly to pin prick with relatively intact sensibility to cotton-wool. The ulnar and median are particularly implicated. Fibrillary contraction of the muscles, atrophy and loss of electrical excitability occur. The special type of deformity known as preacher-hand is one of the frequent expressions of the involvement of the brachial plexus in the cervical meningitis.

8. **Congenital or Hereditary Syphilis.—Effect of Hereditary Syphilis.**—Serological studies have thrown much light on the question of the mode of transmission. This cannot be entered upon here. These studies as particularly carried out by Plaut, Mott and others have shown the enormous importance of transmitted syphilis in the pernicious effects upon the nervous system. Linser, moreover, has shown that two-thirds of the children of syphilitic parents show a positive Wassermann reaction, although much fewer show signs of congenital syphilis.

It may be recalled that Fournier stated the proportion as high as 98 per cent., and that 68.5 per cent. of the children died. This does not include the aborted offspring. Should these be reckoned, one could obtain a true idea of the morbidity of syphilis in the young. Hochsinger reports an interesting group of cases in this connection. In 72 families there was paternal syphilis. The mothers were not syphilitic. Seventy mothers gave birth to 307 children—110 stillborn, 166 syphilitic and 31 healthy. The healthy were all the last born save in four instances.

Of the children of tabetics, one obtains the same story from Mott,

Mendel and others. Either no children, many abortions, many dead children, few living and no one knows as yet the fate of these. Certainly one-half are doomed to disease and disorder of the nervous system.

It would appear that the common effect of such syphilitic infection is to reduce the resistances of the body and its powers for full develop-

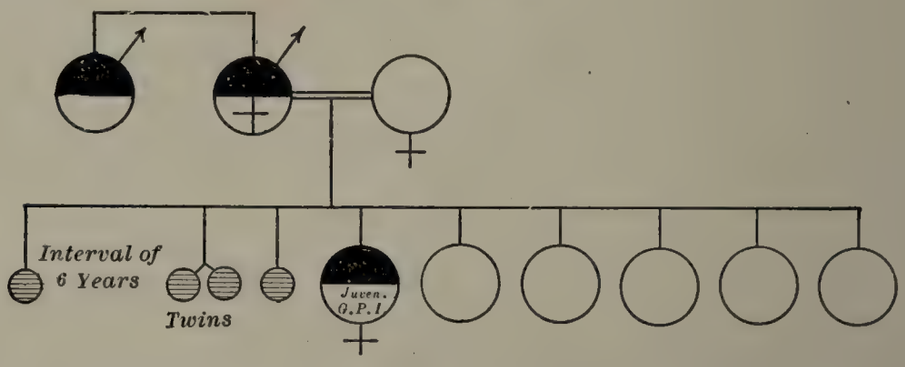


FIG. 433.—Congenital syphilis: juvenile paresis, at first considered as "imbecile." (Mott.)

ment both in the general body and nervous tissues. Syphilis diminishes the vital energy of the germ plasm prior to conjugation and can cause pathological variations in nervous structures just as it can transmit the disease through the germ cells. The abundant studies on alcohol and its influence on the germ cell affords an analogy in understanding how this takes place with another type of toxemia.

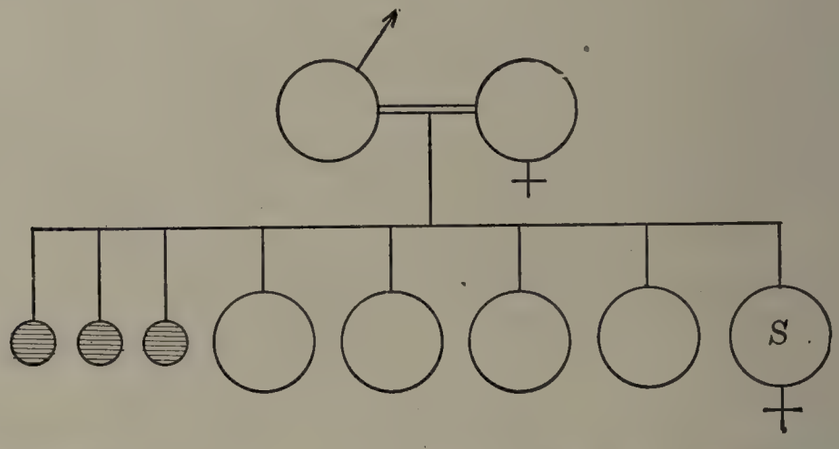


FIG. 434.—Congenital syphilis. Three miscarriages; then five children born alive and well. Last child snuffles, Hutchinson teeth. Did well in school, then deteriorated, noisy, maniacal; thought to be paretic. Autopsy showed generalized cerebrospinal gummatous meningitis, perivascularitis and endarteritis. (Mott.)

The classical formula of Fournier seems to hold—abortion, dead child, early death, living, healthy child. This is in need of amendment; it is worse. The formula reads: Complete sterility, miscarriage, abortion, stillbirths, children dying in infancy or convulsions, marasmus, meningitis, hydrocephalus. Then follow children who are comparatively healthy, but who in later life develop late hereditary syphilis.

A study by Hochsinger (1911) says that of 208 children of syphilitic parents who had been under observation over four years, 89, or 43 per cent., had some disease of the nervous system. Of these there were 9 cases of hydrocephalus, 2 of Little's syndrome, 6 epileptics, 2 paresis, 1 tabes, 6 Argyll-Robertson pupil, 36 extremely neurotic, 5 hysterical, 14 chronic headaches, 10 imbeciles. This is in strange contrast to the statements of Jonathan Hutchinson, who, but a few years ago, taught that nervous syphilis was negligible.

It has been abundantly shown that nearly every form of adult syphilis of the nervous system can be encountered in hereditary syphilis and, as Mott well says, if congenital syphilis were not so

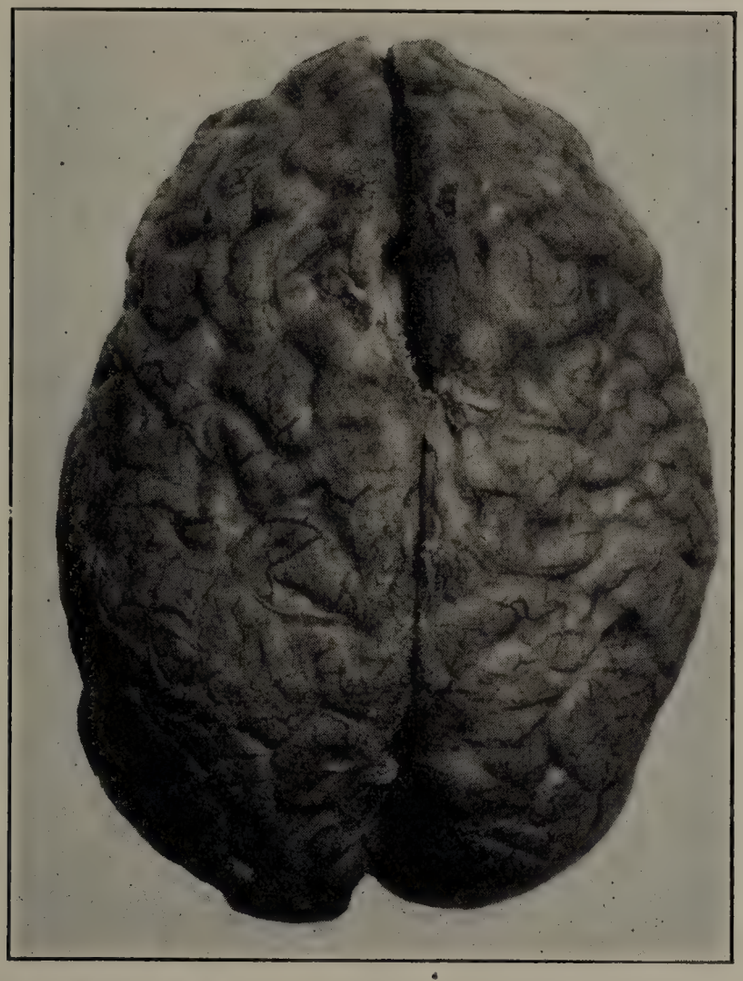


FIG. 435.—Brain of a congenital syphilitic idiotic child. (White.)

fatal to infant life the number of people suffering from syphilitic disease of the brain would be appalling. It would, therefore, be of little service to repeat what has already been written regarding nervous syphilis of adults as it appears in children, and the present discussion will be limited to a consideration of such forms of juvenile nervous syphilis as are present only in children. These are, particularly, certain forms of feeble-mindedness, of hydrocephalus, ependymitis, Friedreich's ataxia, primary optic atrophy and encephalitides or encephalomalacias, leading to various hemiplegic syndromes, often loosely grouped together as Little's disease. Juvenile paresis and juvenile tabes are among the commonest congenital disorders of later infancy or adolescence.

Congenital syphilis of the nervous system may show itself before or at birth; it may develop in earliest infancy or in adolescence; it may develop as late as twenty, or even forty-two to forty-three years (Müller). How long may the virus remain latent, finally to develop a definite syphilitic reaction? As yet the answer will depend upon the general bias of the answerer, rather than upon empirical data. It would appear from the evidence at hand that no definite age limit can be put upon the time when such a latent factor becomes activated by causes as yet unknown. That an activator of some sort plays a role in such disorders as tabes and paresis seems at present a justifiable hypothesis. When a newborn child, known to have been syphilitic biologically, can be followed throughout life, his serum reactions being tested from year to year, as is now being done, then the question can be finally decided.

In this connection a line may be devoted to the subjects of congenital syphilis "unto the third generation." While of late years doubts have been accumulating relative to this matter, certain positive cases are being reported. The rationale of this seems plain in view of the observations of recent years made by Levaditi, Bab and others, that *Treponema pallidum* may be found in the ovum, and in an apparently resting stage similar to the resting stage known for other flagellate protozoa closely allied to the organism causing syphilis.

The fact of the whole matter is that opinions and statistics relative to congenital syphilis of the nervous system, supported by clinical observation alone and uncontrolled by the available biological tests, are insufficient approximations and very insecure. When relied upon for negative conclusions they are harmful to the advance of thought, and detrimental to the relief of sick humanity.

Notwithstanding the importance and often the strikingly gruesome character of these late-appearing congenital cases the attention of the practitioner should be riveted upon the numerically preponderant miscarriages, stillbirths and early syphilitic deaths, if he would get in the right attitude toward the therapeutics of this disease, as it affects the nervous system.

In congenital syphilis, as has been stated, a replica of what has been found in adult syphilis may be expected. Pathologically speaking, the lesions are nearly always combined. There is a variable composite of endarteritis, of leptomeningitis, of pachymeningitis, gummata, large and small, localized or infiltrating gummatous neuritis, diffuse degenerative changes in the cells of the spinal cord, in the basal ganglia or of the cortex. Thus the clinical pictures are apt to be conglomerate and almost unanalyzable. Those more accentuated trends which permit a nosological term will be considered here.

Hydrocephalus.—As a result of congenital syphilis this condition has been suspected for two hundred years. Hasse, in 1828, Cruveilhier in his *Atlas*, von Rosen, in 1862, and Virchow reported definite examples of it. It arises in these congenital forms largely from syphilitic disease of the cerebrospinal fluid-producing structures—

choroid, ependyma, or from definite obstructive factors in the cerebral foramina, gummata, vascular swelling obstructing the iter, etc.

It is a not uncommon sequel of congenital syphilis and is undoubtedly more frequent than is realized. In Hochsinger's series of 362 cases of congenital syphilis 34, or nearly 10 per cent., showed hydrocephalus. In his series, which affords a fairly average review of the situation, the hydrocephalus began three to eleven months after birth; sometimes it was fetal. In 11 cases there were no nervous symptoms, *i. e.*, up to the time of reporting. In the others restlessness, sleeplessness, chronic vomiting, convulsions, contractures, nystagmus and feeble-mindedness were the objective phenomena.

The more usual clinical picture is that of a child, boy or girl, from three to six months of age, who following, or not, an insignificant blow on the head or some gastro-intestinal or bronchial disturbance, develops within a few days grave cerebral disturbances. There is great irritability and sleeplessness, screaming and kicking. The head is usually drawn back, the eyes and the fontanelles are apt to bulge somewhat. Vomiting is frequent and there are signs of oculomotor involvement. Internal strabismus from paresis of the external rectus is not unusual. It is frequently preceded by, or accompanied by, nystagmus and irregular pupils—often not responding to light.

Pain is present, as the child cries and struggles, and not infrequently the active movements of the arms—often highly spasmodic or convulsive—seem to try, in a blind reflex sort of way, to get at and brush away the source of it, *i. e.*, the head, pulling the hair, grasping and rolling the head. Spasticity, rigidity and other signs of intracranial pressure may at times be demonstrated. As a rule the temperature is only slightly, or not at all raised, and the minor signs of an epidemic cerebrospinal meningitis, *i. e.*, herpes, temperature, flushed and spotted skin are absent. The diagnosis of all of these infantile meningeal disturbances is fraught with much difficulty.

Feeble-mindedness.—Syphilis undoubtedly plays a much larger role in producing mental defectives than is suspected. The early statistics are comparatively worthless. They are quoted at great length even in modern works on idiocy, imbecility and the like.

The more correct appreciation of this chapter on syphilis and feeble-mindedness began with the studies of Fournier on parasyphilis. Those truths, somewhat uncontrolled, were forced upon him by his clinical observations. The early English, German and American figures varied from 0.1 (Shuttleworth) to 17 per cent. (Ziehen). Whereas the results following serological investigations start with the higher figures and mount upward, in some cases as high as 60 per cent. The American figures available (Atwood and Clark) showed that 20 per cent. of the idiots, imbeciles and morons at Randall's Island, New York, were syphilitic.

Intra-uterine feeble-mindedness is more or less a contradiction. Those children whose mental defects date to disease going on in the

uterus rarely live. Plaut expresses the opinion that feeble-mindedness may be regarded, so far as syphilis is concerned, as the result of an extra-uterine syphilitic disease undergone in infancy. In some there are signs of an acute brain disease. Some instances of recovered hydrocephalus show the signs of havoc in their inability to develop normally. Many others show no acute stage, but fail to develop. Many recorded observations are available to show the very gradual development of mental defect, without convulsions or fever, altogether without signs of organic disease, which arrived at a definite termination, and which left behind entirely stationary, perhaps even improvable, idiots or imbeciles.

That type of hereditary syphilitic child without any tangible disease, formulated by Fournier—his "enfants arriéres"—which is described as unintelligent, simple, silly, limited children, always behind, not infrequently shows the Wassermann reaction.

Again, one is convinced by the researches of others that mental defect in less-marked grade, or more properly speaking, along more restricted or special lines, is allied with this broad group on the basis of congenital syphilis. Thus Nonne reports cases of general irritable weakness of the nervous system. The patients are highly excitable, are extremely nervous, they are very moody, suffer from headaches, irregularities of appetite, sudden fits of passion—not associated with other forms of epileptiform analogies—and for whom mercury and the iodides work wonders.

Still another chapter has been opened in this hereditary syphilis problem in its relation to mental defect. It concerns many so-called psychopathic children. These children are bright, but they show marked ethical defects. Here one can conceive of the mental defect in terms of limited cortical control to the affective response of the sexuality and to the nutritional instincts. These children want and take without going around by the circuitous routes devised by cultural standards. Anatomically one can posit a defect of certain cortico-cortical association areas on the basis of the syphilitic poisoning.

Taking the whole group of feeble-mindedness, it is evident that clinically one cannot pick out the hereditary syphilitic child in all instances. Indeed, it should be emphasized that too much weight is given to the anomalies in physical structure—Hutchinson teeth, saddle nose, striæ about the mouth, prominent veins, scaphoid scapula, etc.—if one rejects those who from the heredosyphilitic class fail to show such anomalies. Nor can we recognize any certainly pathognomonic psychical anomalies. A careful neurological examination frequently aids in enlarging the group—particularly in the study of pupillary anomalies. The cytological tests are of the highest importance and every child born of syphilitic father or mother should be systematically examined by these cytobiological methods.

Treatment.—The treatment of syphilis of the nervous system appearing in any of the forms previously enumerated is often extremely satisfactory. In fact, at times one might say the results are often too

good, as the speedy relief not infrequently leads the patient to forego further treatment or causes him to pursue his course half-heartedly.[1]

Cerebral syphilis for the most part is accompanied by active spirochetes; hence the therapy is to be directed against this organism. Mercury, arsenic and iodine are the best available spirocheticidal drugs. Mercury and arsenic are active, iodine is very weakly toxic, but Neisser's most recent studies tend to show that it has actions other than that usually ascribed to it, namely, to promote the taking away of breaking-down syphilitic tissue or its product—its so-called resorption action, probably a result of increased sympathetic stimuli to the bloodvessels (see hyperthyroidisms). Neisser shows that iodides in large doses are toxic to spirochetes as well.

The treatment of nervous syphilis then should be an attempt to follow out a general antisyphilitic treatment with special attention to certain structural peculiarities of the nervous system. These peculiarities are of much importance. In the first place, small lesions in the central nervous system, by impinging on important centers, bring about disastrous results, optic atrophy, pontine, medullary hemorrhages, etc. The enormous importance of correct nervous and mental functioning in the struggle for existence is self-evident. The difficulties in the way of repair in nervous tissues are enormous—at times insuperable. Hence one's attack upon nervous syphilis should be prompt and complete. A complete sterilization is desirable—and this is often extremely difficult to bring about in nervous tissues.

It must always be borne in mind that cerebral syphilis is often present with a negative Wassermann reaction—this finding then should not deter one if there are clinical signs of diagnostic import. Not infrequently active antisyphilitic treatment (arsenical compounds) causes the appearance of a positive blood reaction—how often this occurs is not yet known.

Study of the cerebrospinal fluid is often a better guide to the correct appreciation of the situation as well as a reflection of the activity of the process. Lymphocytosis is often present months or years before any definite nervous signs. Lumbar puncture is too often neglected in cerebral syphilis. Many authors state that if the blood is negative to the Wassermann test there is little occasion for studying the cerebrospinal fluid. This leads to bad results. Vascular and meningo-vascular processes may progress for years in nervous tissues without giving rise to a positive Wassermann blood reaction. Here is an occasion in which this symptom fails to be present.

An energetic treatment should therefore be carried out if there are suggestive signs of nervous syphilis, even should there be a negative Wassermann test.

In certain patients rapid action seems imperative; in others the need for this rapidity is not so much in evidence. Granted a knowl-

[1] White and Jelliffe: Modern Treatment of Nervous and Mental Diseases, vol. 2. Lea & Febiger, Philadelphia, for more complete discussion. Leredde: Traitement du Tabes, Maloine, 1918.

edge of these requirements, the choice of remedies is not as simple as it might seem. Iodides given by the mouth in doses of 30 grains, 2 grams a day, show evidences of activity in about one week. Mercury by inunction shows results in about five days, while the newer arsenical preparations show reactive capacities in about forty-eight to seventy-two hours. None of these figures should be accepted as final, so far as curative action is concerned, nevertheless they are worth something. It is not apparent that iodides have a rapid toxic action; hence in lesions which are characteristic of the more florid aspects of spirochetal growth—basal meningeal types of acute onset particularly—they should not be chosen in the initial attack.

Inunction Method.—For years neurologists have taught—chiefly under the influence of Erb—that nervous syphilis is best attacked by the inunction method—combined with iodides. In those situations in which the time element is of less moment this attitude seems justifiable, especially for gummatous and exudative pathological formations and it may be for vascular localization of the spirochetes.

Oleate of Mercury.—The oleate of mercury is of value in that it is comparatively cleanly and produces results as rapidly as other mercurial preparations applied to the skin. A dram of the 10 per cent. oleate is to be used night and morning for four days. The patient then takes a vapor bath and the same dose is used once a day for four days more. If sponginess and soreness of the gums do not appear—with cleaned teeth and gums—the double dose may be continued; otherwise a single dose should be utilized. In using the oleate one usually employs a small piece of flannel in the rubbing—the first dose should be larger, as the flannel absorbs it, and the same piece of flannel should be used continuously.

The oleate may irritate the skin, but as it is absorbed fairly well from all parts of the body one can shift around more readily with it than with other mercurial ointments. Another object of using the oleate is on the ground of secrecy.

This line of treatment should continue at least six weeks; after the first week 10 grains (0.6 gram) of potassium iodide t. i. d. should be administered during the course of treatment. There is very little advantage in raising the amount of iodide above 60 grains (2 grams) a day. After six or eight weeks the treatment should be discontinued absolutely—to be renewed not later than three months after the termination of the last treatment. A third and fourth course is advisable, even imperative if a positive Wassermann test is present in the blood or lymphocytes above 10 to the cubic millimeter are obtained from the cerebrospinal fluid.

Unguentum Hydrargyri.—Unguentum hydrargyri is much used and widely recommended. It has the disadvantage of being dirty and of attracting attention. The latter may be partly obviated by adding some non-stainable coloring matter or some smelling compound such as balsam of Peru. Attention can thus be diverted from its

characteristic color. The ointment is used in daily doses of from ʒj (4 grams) to ʒij (8 grams) best rubbed in in the evening in a fairly definite manner and in places where the skin is more permeable. One uses the inner surfaces of the arm and forearm for the first rubbings, covering them with bandages, then the inside of the groin, then the popliteal space, then the abdomen and back. The fifth or sixth day the patient omits his rubbing—takes a Turkish bath and then starts over the same course. This course is kept up for thirty doses.

Other Details.—The care of the skin and of the mouth is naturally to be kept in mind. The blood Wassermann reaction should be tested at the end of the period, and if strongly positive, or if spinal puncture shows active lymphocytosis, or if clinical signs seem slow in responding, the inunctions should be continued at least two or three weeks longer.

Checking up by the Wassermann test and lumbar puncture three or four months later, or any increase in clinical signs should determine a repetition of the treatment along identical or more strenuous lines.

Iodides, 30 grains daily, are to be given throughout the course of the inunctions. All medication should cease at the end of the cure, unless there are definite indications for its continuance.

Other mercurial inunction masses may be used. Those of value are the hydrargyri vasenol, vasogen, mitin, resorbin, which have special indications which may render them particularly valuable.

Mercury Injection Treatment.—Injection treatment attempts an even more rapid and energetic attack upon the spirochete. Many battles have been fought among syphilographers as to the comparative merits of the insoluble or soluble salts of mercury. When so much diversity of opinion can be found, it usually indicates that the real differences are usually minimal. Hence ease of administration, safety, painlessness, etc., determine the choice of the remedy in each case.

Calomel, mercury salicylate and acetate of mercury are among the more favored insoluble salts. Calomel has occupied a high rank and can be utilized in the following forms:

℞—Hydrargyri chloridi mitis 5.0 gm.
Sodii chloridi 5.0 "
Aq. dest. 50.0 "
Mucilagio arabici 2.5 "

℞—Hydrargyri chloridi mitis.
Ol. sesami, 10 per cent.

Pravaz syringeful every second or third day, preferably into the muscles of the thigh or back, for 12 to 15 doses.

The hypodermic use of calomel is often accompanied by much pain. Abscess and necrosis is not uncommon, and lung emboli may occur—with care, however, calomel given by hypodermic is free from danger.

Creams of calomel, devised by Lambkin, have been extensively used, as they cause less pain and give rise to no complications if

bloodvessels are avoided. These creams should be sterile. The formulæ of some in use are as follows:

℞—Calomel	5 gm.
Creosote	20 "
Camphoric acid	20 "
Palmitin	100 "

Inject 10♏ of this cream once a week for one month, to be replaced by the following:

℞—Hydrargyri (metallic)	10 gm.
Creosote	20 "
Camphoric acid	20 "
Palmitin	100 "

This is injected in doses of 10♏ twice a week for three weeks.

After six doses have been given, stop for two months. Four injections of the metallic cream are then given at fortnightly intervals. Then a rest for four months. Then four injections as before and a rest for six months. Then a repetition of four fortnightly doses—an interval of one month and a final series of four metallic cream doses.

English syphilographers have found these creams admirable in army and navy work. They are adapted for early stages better than for nervous syphilis, but are worthy of more extended trial in nerve syphilis.

The use of the insoluble salts has the advantage of a much more prolonged action of the mercury. They also have the disadvantage—all mercurial salts share in this, however—of irritation of the kidneys. If albumin is found before the use of mercury one should look for a syphilitic nephritis. Tuberculosis, diabetes, alcoholism, marked cachexia are additional factors to be carefully dealt with. Gastro-intestinal disturbances are frequent, but it is extremely rare that mercury causes a neuritis.

Thorough cleansing of the mouth and the use of a chlorate of potash mouth wash is imperative.

The soluble salts in use are very numerous. They include the sozoiodolate, bichloride, lactate, succinamide, biniodide, benzoate and cyanide. Fournier lists about 30. They may be injected within the muscles or into the skin, superficially. All are somewhat painful and accidents are possible. In general the dosage is from $\frac{1}{4}$ to $\frac{1}{2}$ of a grain. The injections are given twice or three times a week.

General Scheme of Injection.—The following general scheme is suggested:

1. The site usually chosen is the posterior third of the buttock, to avoid the sciatic nerve and vessels.
2. The skin should be scrubbed with alcohol or ether.
3. The injection should be made deeply into the muscle, using each buttock alternately.
4. The syringe and piston should preferably be of glass, easily sterilized, and the needle of platinum iridium, about $1\frac{1}{2}$ inches in length and sterilized.
5. After insertion of the needle, the piston should be slightly withdrawn, and if any blood appears the needle should be reinserted in

order to avoid injection into a bloodvessel. The injection of the solution free from air bubbles can then take place.

Solutions.—Various solutions are in use. Only a few can be mentioned here.

℞—Hydrargyri sozoiodolate	gm.	0.2	gr. iij
Sodii iodidi	gm.	0.3	gr. v
Aq. dest.	gm.	10.0	ʒiiss

Dose—10 to 20 minims constitute the dosage.

℞—Hydrargyri lactatis	gm.	0.2	gr. iij
Aq. dest.	c.c.	18.0	ʒiv

Dose—10 to 25 minims.

℞—Hydrargyri succinamidi	gm.	0.2	gr. iij
Aq. dest.	c.c.	10.0	ʒiiss

Dose—10 to 25 minims.

℞—Hydrargyri chloridi corrosivum	gm.	0.5	gr. viij
Sodii chloridi	gm.	3.0	gr. xlv
Aq. dest.	gm.	100.0	ʒiij

Dose—1 to 2 c.c. daily or alternate days.

The use of corrosive sublimate—following Lewin—is usually very painful.

℞—Hydrargyri cyanidi	gm.	1.0	gr. xv
Cocain hydrochloridi	gm.	0.3	gr. v
Aq. dest. ad	gm.	100.0	ʒiij

Dose—1 to 2 c.c.

A useful variant of this combines the cyanide with arsenic and strychnine, as follows:

℞—Hydrargyri cyanidi,					
Strych. arsenatis āā	gm.	0.6	āā	gr. ix	
Cocain muriat.	gm.	0.3		gr. v	
Aq. dest.	gm.	60.0		ʒij	

Dose—5 to 10 minims every other day for 20 to 25 doses.

Cocaine may be added to any of the soluble salts. Its addition lessens the pain.

Fournier has always advocated the use of the biniodide dissolved either in sterilized oil or in water. It is, he claims, painless, sure and free from dangers. In 2457 injections only 9 produced pain. Such results, however, obtain only when the physician is very careful. Careless use with the biniodides will produce all of the accidents, pain, abscess, etc.

℞—Hydrargyri biniodide	gm.	0.4	gr. vj
Olive oil (sterilized)	gm.	10.0	ʒiiss

Dose—One Pravaz syringeful every other day.

℞—Hydrargyri biniodide	gm.	0.2	gr. iij
Sodii iodide	gm.	0.2	gr. iij
Aq. dest.	gm.	10.0	ʒiiss

Dose—1 to 2 c.c. daily or on alternate days, with gradual elevation of the dose if stomatitis or gastro-intestinal signs are not in evidence. Twenty to twenty-five injections constitute a course of treatment.

℞—Hydrargyri benzoatis	gm.	1.0	gr. xv
Sodii chloridi	gm.	2.5	gr. xlv
Aq. dest.	gm.	120.0	ʒiv

Dose—1 to 2 c.c. daily or on alternate days.

Combined arsenical and mercurial injections were very much in vogue before the introduction of the salvarsan preparations. One of the most popular of these has been the arsenical salicylate or enesol. This remedy has been used widely in nervous syphilis and often with surprisingly good results. Schaffer speaks very highly of it.

The combined use of the cacodylates and of mercury has been observed to give good results. The early reports of optic nerve disease apparently following the use of the cacodylates served to force these salts into the background. Inasmuch as such optic nerve changes apparently occurred in other than syphilitic patients, it would not appear that they are to be interpreted as instances of those neurorecidives which have been so actively discussed since salvarsan has been introduced.

Newer Arsenic Products.—Any attempt at an exhaustive summary of the various reports upon these remedies in the treatment of nervous syphilis would require a special volume. A simple enumeration of the bibliography alone—best obtained in brief in Lewandowsky's *Handbuch der Neurologie,* articles by Forster and Schaffer and others —in Nonne's discussion, already referred to would require dozens of pages. Only the present (1928) drift of opinion will here be expressed. Since the great disturbance in the chemical industries due to the Great War there have been many new arsenical compounds made, either as replicas or imitations or as substitutes for those originally introduced by Ehrlich and the German chemists. Not enough time has elapsed to permit an adequate appraisal of these remedies. The reader is warned that many pirate combinations are on the market and that fatal results have been more frequent in recent war times from the use of arsphenamine products. Great care must be taken in the selection of the product used.

In the exudative, hyperplastic, gummatous and arterial forms arsenical compounds, arsphenamine, related to salvarsan and neosalvarsan, are by far the most efficient remedies possessed at the present time. One form needs to be excepted, that of the large gummata, for which surgery alone is adequate. It would also appear that much larger doses of salvarsan are required for nervous syphilis than were used in the earlier stages of its administration. Since the use of adequate dosage the so-called neurorecidives have almost entirely disappeared. It is the present trend of opinion that mercury and arsphenamine combined gives the best results. Whether or not arsenic and mercury, both active spirochetal drugs, supplement each other in this combined use is not certain, but the results obtained have in many instances been very satisfactory.

At the same time it needs to be observed that a number of patients have relapsed and the final results of therapy in nervous syphilis have been far from being as hopeful as had first appeared. Because this has been so is no reason why it should remain so. The most obvious reason that stands out in many of the recent discussions that have taken place relative to this point is that the patients have

been insufficiently treated. Finally, arsphenamine has not been long enough, as compared with mercury, in use to warrant anything but as yet tentative conclusions. The general attitude is that the arsenical compounds, while extremely valuable, have been much overrated.

With this short summary of conclusions, a few words may be said as to its application and dosage. Whether neoarsphenamine is to replace arsphenamine or not cannot yet be determined. The newer silver-salvarsan is much in vogue, 1920–1922, while newer compounds as novarseno-billon, novarsenobenzol and sulfarsenol are claiming recognition. Sulfarsenol is highly praised as a spirochetocidal agent. Bismuth salts have of recent years been experimentally introduced. There is not enough experience in neurosyphilis to appraise their value. The intravenous administration of arsphenamine is the best method of giving it. It should not be given unless the patient is under some sort of supervision—in a hospital or remaining in bed—and minute attention to the technic is absolutely necessary to avoid certain dangers. It is highly important that fresh, distilled water be employed if arsphenamine is to be used intravenously.

To kill spirochetes in the nervous system, however, is one thing, and to overcome the results of tissue changes is quite a different one, and this above all is the stumbling-block in the treatment of nervous tissue syphilis. Nevertheless, if nerve tissues have not been extensively destroyed, one can hope for excellent results by a proper combination of arsphenamine therapy with mercury.

Arsphenamine must be used in much larger quantities, however, than was at first practised. At the end of this section the outline of an energetic combined therapy is given and reference may be made to those pages for the general indications of such a course of treatment. Modifications to a less active mercurial salt than calomel may have to be made. Every patient needs individual treatment.

Notwithstanding the very evident fact that salvarsan and neosalvarsan are active spirocheticidal drugs, it is still an important problem how to reach the spirochete in the nervous system. Careful chemical investigation of the cerebrospinal fluid has heretofore failed to obtain any trace of arsenic when salvarsan has been given in the usual manner. One may infer that the arsenic has become fixed in some chemical combination which fails to react to the usual chemical tests. It is not yet fully comprehensible why nervous syphilis is so resistant to treatment and why the hopes aroused by the striking results of salvarsan therapy in general syphilis seem not to have been borne out in nervous syphilis.

It has already been pointed out that the lymph spaces being blocked by the syphilitic process constitutes a great barrier to the introduction of drugs. When this difficulty is overcome and better penetration obtained then the treatment of the parenchymatous or central types may give better results.

Swift and Ellis have attempted to place a spirocheticidal solution directly into the cerebrospinal fluid. Salvarsan and neosalvarsan

were employed by direct injection into the spinal canal through the Quincke lumbar puncture. This method they found was to be condemned. It failed to give any beneficial results, and, moreover, caused marked pains. It is probably a dangerous procedure, as animal experimentation has shown.

An attempt was then made to introduce into the patient's cerebrospinal fluid some of his own blood serum which had previously been mixed with the arsphenamine by intravenous infusion. This procedure is carried out in the usual manner. After a certain length of time—one hour was found to give the most active serum—blood was withdrawn, separated from its corpuscles, after twenty-four hours, diluted with 40 per cent. of normal saline and then heated to 56° C. for thirty minutes. A lumbar puncture is then made and from 5 to 15 c.c. of cerebrospinal fluid is withdrawn, *i. e.*, until the pressure falls to 30 mm. of mercury. Thirty cubic millimeters of the warmed serum is then injected into the subarachnoid spaces. The patient must lie quiet, the foot of the bed usually being raised. After ten days to two weeks the injections, which are usually well borne are to be repeated.

They thus obtained very striking results in the action upon the serobiological factors known to accompany cerebral syphilis. Then a more crucial experiment was planned. This consists in the introduction into the subarachnoid spaces of serum taken from another individual, usually a secondary syphilitic under treatment, the technic being that just outlined. In the treatment of tabes by a heterologous serum most excellent results have been obtained in some cases. In others they have been *nil* or positively dangerous. Why the striking character of the results? for it is at once evident that the amount of spirocheticidal substance in a few cubic centimeters of serum taken from the body of another patient who had received the usual intravenous salvarsan therapy must be very small indeed, *i. e.*, reckoned as arsenic. If other factors than the salvarsan itself enter into the situation these are as yet unknown. One significant fact, however, would tend to indicate that other forces are operative. Extensive experiments carried on by Swift and Ellis with the heated and unheated serums show that the heated sera are three times as spirocheticidal to Spirocheta duttonii in mice.

In the treatment of tabes the intraspinal method is often very efficacious. Again it is disadvantageous. The advantages outweigh the disadvantages. Excessive pain is sometimes the first reaction.

In the treatment of paresis intracranial injections of salvarsanized serum offer increasing evidences of amelioration. These injections may be intraventricular or intradural. The indications are crystallizing. The general trend is to make a rapid attack by intravenous methods, which if unavailing can be followed up by the intracranial route.

The most striking, suggestive result is an almost immediate diminution in the number of pathological cells in the cerebrospinal fluid. The globulin reaction diminishes, positive Wassermanns of the cerebro-

spinal fluid with small quantities of fluid require larger quantities to show positive or become negative and the amelioration of the symptoms has in a few cases been rapid.

Mercury by the Mouth.—Mercury by the mouth will always remain one of the simplest, and yet at the same time, least efficient methods of treating syphilis of the nervous system. Here again one has a rich choice of remedies. Those most in use are: protoiodide grain $\frac{1}{8}$ to $\frac{1}{3}$, sublimate grain $\frac{1}{20}$, calomel 2 to 5 grains. Various vehicles are used.

The disadvantages of treatment by means of the intestinal canal are many—chief of which is the slow and weak action of the remedies employed. Moreover, the gastro-intestinal tract suffers.

The chief advantage is that mercury may be combined with the iodides. Furthermore, conveniences of medication must often constrain one to use this mode of giving antisyphilitic remedies, but only as a necessary choice.

In nervous syphilis it would appear that mercury medication by mouth is not radical enough. One may use it after an energetic treatment, by the methods outlined, has been employed, but oral administration is rarely a method of certain value and hence is not advisable, save under particular circumstances.

Among the newer mercury preparations which future experience may prove to be of value are: Mercury dicarboxylate, two forms of which are on the market, with pronounced toxic action on spirochetes in rabbits. It is claimed to be twenty times as toxic to spirochetes as corrosive sublimate and yet shows no action on the body. Its dosage has not yet been worked out.

Iodides.—Sodium and potassium iodide have been used in the treatment of syphilis of the nervous system for years and often with good results. According to Neisser the iodides are weak spirochetal poisons. Our belief in its resorptive powers is justified on empirical, if not on pharmacological grounds. Personal experience does not confirm the belief in the efficiency of specially large doses, although that is the American preference.

The use of the iodides in doses of from 10 to 30 grains t. i. d. combined with mercury is particularly valuable in the gummatous type of cerebral syphilis. It is folly, however, to try to do away with large gummata by means of massive doses of iodides.

The dosage of the iodides will depend upon the individual. There are many idiosyncrasies to be borne in mind. At times small doses cause marked disturbances and cannot be borne. Here one may employ other combinations than those of sodium or potassium. Hence strontium, rubidium and organic iodine preparations have come into use. Iodopin, sajodin, iodoglidin, iodoval, iodocitin, iodostarin are among the newer of these combinations.

Iodopin may be injected as well as administered by mouth. In the former case it is used in quantities of 10 c.c. on alternate days, or smaller doses 1 to 3 c.c. at more frequent intervals. In giving it by hypodermic both the syringe and the remedy should be slightly

warmed, the needle should have an ample bore and the drug be introduced slowly. It is also given by the mouth in 3j doses. In the form of iodopin, large quantities of iodine may be introduced without toxic effect. Its action on nervous syphilis has not been extensively studied.

Iodoval and iodocitin, the latter a lecithin-albumin compound, have been found to be borne well in the course of salvarsan-mercurial treatment. The former is given in doses of about 5 grains t. i. d. throughout an energetic salvarsan-mercury treatment—the latter in about the same doses.

Plan of Intensive Treatment.—Nervous syphilis is treated too gingerly by most practitioners. It is difficult to kill the syphilis organism, hence an energetic course of treatment is here outlined:

First day	0.03 calomel (or other mercurial) hypodermically.
Third day	0.05 calomel (or other mercurial) hypodermically.
Fifth day	0.4 salvarsan (or other arsenic preparation) intravenously.
Seventh day	0.5 salvarsan (or other arsenic preparation) intravenously.
Ninth day	0.05 calomel (or other mercurial) hypodermically.
Eleventh day . . .	0.05 calomel (or other mercurial) hypodermically.
Thirteenth day . . .	0.4 salvarsan (or other arsenic preparation) intravenously.
Fifteenth day	0.5 salvarsan (or other arsenic preparation) intravenously.
Seventeenth day . . .	0.05 calomel (or other mercurial) hypodermically.

This should be continued for six weeks, or until at least 5 grams of salvarsan are administered. The whole course can be compressed into three weeks if a soluble mercury salt is given and at least 5 grams of salvarsan can be administered in that time. The patient should be watched very carefully, especially with reference to the kidneys. Furthermore, there are patients who do not bear calomel well. Vagotonic individuals react excessively to mercury, especially to minute doses. The treatment should be carefully followed by the serobiological tests: (Compare article on Syphilis of the Nervous System in *Modern Treatment of Nervous and Mental Diseases,* White and Jelliffe, Vol. II.)

Paresis Treatment.—Up to the present time the treatment of the parenchymatous types of neurosyphilis has seemed unavailing. Here and there a paretic has been held for some years in a remission, but on the whole the treatment has been far from satisfactory.

This has induced a wide survey of newer forms of therapy in this usually fatal disorder.

It has been thought good practice to treat a suspected paretic by the intensive method here outlined. If the serobiological changes with the clinical signs have not shown that a cure has been arrived at further therapy is necessary.

Intraspinal therapy has not been of much service in paresis. Intraventricular therapy has helped a few cases that intravenous and intraspinal cases have left untouched. But here again certain forms

are resistant even to all mercurial, iodine or arsenical preparations. The inavailability of metallic toxic agents has caused certain observers to look to other means and Wagner v. Jauregg[1] of Vienna has tried out a number of agents, such as lutein, nucleins, various vaccines, tuberculin and of late has reported excellent results from the use of the tertian malarial parasite. His early experiments were interrupted by the war, since resumed and more or less widely corroborated, by Weygandt, and Nonne of Hamburg, Delgado of Lima, Peru, and others.

The technic is simple. Inoculation can be either intravenous or subcutaneous with about 4 cc. of infectious human blood. In subcutaneous inoculation, the skin should also be scarified with the needle after the injection and a drop of blood left on the scratches; then covered with a little bandage. Recent experience with malaria strains have shown that there are biological changes in time and that certain different tricks in connection with inoculation can help keep up the potency of the strain (American and German lit. in Gerstmann). In general, the intravenous injection works more quickly, but it seems that the rigors are then more severe. If the fever fails to develop, if the malaria does not take, it can be provoked by injection of typhoid vaccine intravenously (50 millions), by an intramuscular injection of sod. nucleinate, or both together, or a reinjection of the malaria. The patient should fever at least 8 to 10 times and not less than 102° F. After the full course of fevers, the patient gets 1.0 gram of quinine for four days in succession and then 0.5 gram for four more days in succession. Immediately after the fevers and quinine one begins with neosalvarsan treatment, the patient getting a total of about 5 gram. If the results of the treatment are not satisfactory one can inoculate again as soon as the salvarsan treatment is over. The patient should be kept under observation for control for many months afterward. If the spinal-fluid findings are not very normal after six months, the treatment is to be repeated.

If the fever gets too high and the patient gets a bad pulse or is otherwise weak, one can take either one of two courses. (1) Interrupt by sufficient quantities of quinine (say, after four attacks), administer salvarsan and then reinoculate for another course of four attacks after the salvarsan. (2) If the patient can stand the fevers, but must have them a little lower, one can give 0.1 to 0.2 gram of quinine, which will not stop the infection, but will put a damper on the severity of the attacks. A modern method is also the inoculation of malaria subcutaneously with a subsequent treatment of minute doses of quinine every day of the incubation period until the fever begins.

Every half year the spinal fluid of the patient should be examined until it is found normal. If it is not normal, the cure is repeated.

Some patients fail to take the malaria, especially at the second inoculation. In such cases, relapsing fever is inoculated, either from the blood of culture mice or from another patient. The treatment with

[1] Jauregg: Treatment of Paresis by Malaria, Jour. Nerv. and Ment. Dis., May, 1922. Delgado: Ibid. Gerstmann. Springer, 1926.

relapsing fever cannot be repeated because of an immunity. Relapsing fever can also be used to wake up an obstinate case of refraction to malaria. In such a case, the malarial attacks usually come between the relapsing fever periods. In the event that the relapsing fever plus the malaria begins then to put too great a strain on the patient, the malaria can be toned down by means of any one of the above mentioned quinine methods. But *not* with salvarsan, as this would stop the relapsing fever. This fever runs out of itself and does not recur.

The end-results are still to be reported on. The latest summaries (1928) all seem to indicate that the malarial treatment of paresis constitutes a definitely valuable addition to therapy. The theory of its possible efficacy is based upon two factors: (1) The influence of high temperature upon the spirochete; (2) the production of certain biological protective products by an organism zoölogically related to the spirochete.[1]

[1] Kirschbaum, W.: Ztschr. f. d. ges. Neurol. u. Psychiat., 1922, **75**, 635. Gerstmann, Kirby and a host of others. See Weichbrodt: Ztschr. f. d. ges. Neurol. u. Psychiat., 1926, **109**, 599, for a good summary: Pfeiffer: Ztschr. f. d. ges. Neurol. u. Psychiat., 1926, **105**, 224. Riva: Riv. sper. di fren., 1926, **50**, 13. Walther: Arch. Psychiat., 1926, **78**, 281. Trossarelli: Policlinico, 1927, **34**, 94. For a general review: Mingazzini: Bull. e atti. ac. Roma, 1927, **53**, 60. Freeman: Jour. Am. Med. Assn., 1927, **88**, 1064. Ferraro: Jour. Nerv. and Ment. Dis., 1927, **65**, 225. Vonkennel: Die Malaria behandlung der Frühlues, Karger, 1927. Relapsing Fever Treatment: See Jahnel: München. med. Wchnschr., 1926, **73**, 2015. See enormous literature in Quarterly Cumulative Index Medicus.

PART III.

PSYCHICAL OR SYMBOLIC SYSTEMS.

NEUROSES, PSYCHONEUROSES, PSYCHOSES.

Introduction.—The twentieth century sees psychiatry fast loosing its bonds to the old time formulations; with concepts, even when bearing the same name, broader and deeper; and with new points of view that have required either new terms to express them or a new use of the old terms. It was almost altogether in the descriptive stages, struggling with those fundamental formulations, mania, melancholia, dementia, which had come down through the centuries. The close of the century, however, found these concepts gradually yielding and the process of their dismemberment well started. Paresis had been pretty well differentiated clinically though the full appreciation of its significance had to wait upon the proof of its luetic origin. Paranoia had been separated out but was largely thought of as a disorder of the intellectual processes and as often secondary to chronic states of mania and melancholia. The various degrees of idiocy and imbecility were recognized as were also the toxic deliria, the senile and various organic deteriorations, and certain symptomatic states such as the puerperal psychoses, that later were to be otherwise accounted for. There remained the great mass of more or less undifferentiated excitements and depressions out of which group were recognized certain adolescent and periodic types. Kraepelin, by applying the criteria of course and outcome formulated from this mass the two outstanding concepts of the relatively benign manic-depressive group and the relatively malignant dementia precox group, the most notable of contributions of descriptive psychiatry. Already the French school had begun a study of psychological mechanisms, particularly in hysteria, which was later reformulated and extended by the psychoanalytic school. The close of the century, therefore, found descriptive psychiatry at the peak of its development as represented by the Kraepelinian school of Munich and the beginnings of an interpretative psychiatry.

Kraepelin's method was a distinct advance upon what had gone before. It approached the problem of mental disease by the natural history method of studying its life history, but in so doing still remained at the descriptive level. Psychiatry in this period of its development

was occupied largely with questions of classification and dominated by a psychology which was also in its descriptive stage of development and handicapped by metaphysical speculations on the nature of the psyche and its relations to the body.

The present-day trends and concepts of psychiatry had their beginnings in a larger knowledge of the nervous system, particularly of its development and of the functions of the vegetative neuromusculoglandular apparatus; in the growth of a genetic psychology (psychology of primitive man and child psychology); in the results of the studies of borderland states (particularly the neuroses and psychoneuroses); in the growth of a behavioristic psychology largely founded upon animal psychology; and finally in the rise of the psychoanalytic school.

The older psychology, both normal and morbid, was not only descriptive but largely a refined physiology of the sense organs. In this psychology the *sensation* was considered the unit of psychological experience while in the new approach to psychological problems the *wish* is coming to replace the sensation as the unit. The wish is the form in which the psyche registers the result of the somatic trends, (action patterns, engrams), as finally integrated and expressed in the psyche. Psychology thus becomes the study of the tendencies of the whole individual as expressed in the psyche, the functions of the individual, in distinction from physiology which deals only with the functions of the several parts, the organs.

The Individual as a Whole.—Inasmuch as each organism is so integrated as to have tendencies which express it as a whole to which its several parts minister as subordinate to the larger ends the psyche has a history which is equal in length, contemporaneous, to the history of the soma. To speak only of man, this history is the history of his cultural development and is comprised of a past of over one million (according to the best recent calculations) years. Just as the history of the body has been laid down in structures, the organs, which represent its final answer to the several problems of adjustment in terms of physiological function, the history of nutrition, for example, as laid down in the structure and functions of the gastro-intestinal tract, etc., so the history of the psyche is laid down in ways of thinking of which ideas and feelings are the manifest evidence, and as the meanings of the various organs can only be understood by an analysis of their past (embryology and comparative anatomy), so the meanings of given ideas and feelings can only be understood by tracing their growth and development. The historical past of the psyche is the region of the *unconscious*, and this region can be reconstructed only by an analysis, an uncovering of its contents much as the history of the earth's crust can only be known through excavation and the discovery of the fossil remains in the several strata. The uncovering of the contents of the past in the unconscious may, by analogy, be termed paleopsychology. This concept is important because in mental disease we constantly see ideas, feelings, ways of thinking outcropping which can only be understood if we relate them to like ideas, feelings, and ways of thinking in the

developmental history of the psyche. Patients constantly display psychological symptoms which can thus be at once related to the psychological characteristics of primitive man and of the child.

In dealing with the individual then at the psychological level it becomes necessary, for the first time, to use terms which refer to the individual as a whole. In fact that is only another way of designating what psychology is. At the physico-chemical and the sensorimotor levels it was possible to deal with isolated phenomena so far only as related to the particular and immediate end of the function under consideration. For example: tapping the patella tendon produces an extension of the leg on the thigh, which, if average in extent, indicates a normal motor pathway to the quadriceps. At the psychological level, however, it is no longer possible to deal with parts of the individual in this way, but the whole individual comes at once under consideration. Thus it may be a question of desire, of failure, of regret, of inefficiencies of all sorts, but it is always a desire, failure, regret, or what not, of the individual. The individual as such has failed in effecting an adequate adjustment. The failure, it is true, may have been conditioned by bad vessels, by visceral disease of all sorts, but the consideration of these factors is a consideration at the physiological level. As soon, however, as the problem is expressed at the psychological level it is of necessity expressed in terms that refer to the individual as a whole, as a social unit.

In this scheme instinct occupies a position midway between the organic and the functional or better, for our purposes, between the somatic and the mental and its expressed cravings at the psychological level are therefore mental representatives of organic or somatic tendencies.

A few paragraphs devoted to an account of the development of these tendencies of the whole individual before taking up a consideration of the disorders at this level will be useful.

Omnipotence versus Dependency.—The baby in its mother's uterus has no conscious desires; it has to do nothing consciously for itself, not even to breathe; it rests quietly, far removed from sources of outside stimulation and irritation, its functions being in large part performed for it by the mother. After the baby is born this condition of affairs still continues, or at least an effort is made for it still to continue. The baby, to be sure, has to begin to breathe for itself, to eat for itself, to perform the functions of digestion and elimination for itself, but on the other hand, there stand about the army of the household, not satisfied to wait upon desire, but with every heartstring of emotion tense to forestall it. He is waited upon hand and foot by all; he remains, in the sense that every desire is satisfied, relatively omnipotent.

As the days go by and development proceeds apace, as the sense organs register more accurately, the muscular adjustments become more refined, the baby's contact with the world becomes progressively and increasingly complex; and try as they will the loving attendants cannot forestall all of his desires, and so there come times when food

is not offered at the instant it is needed, when sleepiness overtakes the baby but he cannot woo it if he is in a bright and noisy street or on a clattering car far from his soft bed. And so there arises insidiously but necessarily the mental state of desire, things wished for because they are not had.

Still even in this stage of development the discrepancy between desire and attainment is not great. Attainment, in fact, is usually very near at hand, the hunger is not permitted to last long, the baby does not have to be kept awake by noises, except for a brief period, while in the matter of other desires, such, for instance, as the desire to empty the bladder, that is indulged in forthwith without any further consideration of the matter. As development progresses, however, desires become more and more numerous, because the baby touches reality at more numerous points, and each one of these points offers a new possibility for a frustrated or delayed desire, while with such matters as emptying the bladder and particularly the bowel there soon steps into the situation the social repressions represented by the prohibitions of the mother.

Thus growing up in the life of the baby, beginning even in the earliest days, there is an ever-increasing discrepancy between desire and attainment and as the years go on it will be seen, without the necessity for further illustrations, that the amoral, egocentric baby must gradually more and more take into consideration the world about him. He is forced to lay his conduct along certain lines which imply a putting off of the satisfaction of desire into an ever-receding future. Later in life, when he is hungry and wishes to eat, he can only satisfy this desire provided he has worked and earned the wherewithal to buy food, and if he endeavors to satisfy it otherwise by taking any food that may be at hand he offends the social usuages and becomes a thief. If he wishes to empty his bladder he has to wait until he gets to an appropriate place; it cannot be done anywhere and at any time. He has to adjust himself to the requirements of society or run serious risks if he fails. As he becomes progressively more complex, as his desires become more and more difficult of fulfilment, as he demands more and more of the world, the individual finds that he has to put off fulfilment further and further into the future and be satisfied to struggle perhaps for years to attain some specific end.

Conflict is therefore at the very basis, the very root of mental life, the adjustment of the individual to the world of reality is by no means a passive molding by external forces, but the individual is constantly and actively, in his mind at least, reaching out and trying to mold the world to suit himself. Conflict is even more universal than this statement would indicate. It is not only at the basis of mental life but of all life. The change from unicellularity to multicellularity is a change brought about, so to speak, by the necessity for a finer adjustment to functional problems effected by the specialization of the functions of groups of cells—organs. These groups of cells have, therefore, to relinquish certain of their tendencies in favor of a greater

specificity of response which is calculated better to serve the individual —biological unit—as a whole. In the mental sphere the analogy is as between the wishes of the individual as such and the welfare of the social group. These two groups of impulses constantly cross and as a result the individual has constantly to renounce his personal, selfish ends to the welfare of the group and to substitute—sublimate—his personal satisfactions by the reward of social esteem. Just as groups of cells get out of hand when they are not adequately integrated in the general scheme of the whole individual and produce tumor formations so the individual who is not adequately integrated as a member of the social group becomes asocial and a group of such individuals becomes the nucleus of a distinctive social movement.

An important corollary to this concept is that the conflict is always between diametrically opposed tendencies. In general the great tendencies are self-preservative and race-preservative (creative), symbolized at the symbolic (psychological) level in various ways as between death and life, bad and good, left and right, down and up, etc. This is the basis of the *ambivalency* of the symbol.

It is from this basal fact of conflict that there take origin two forms of thinking, an understanding of which is of great importance for the comprehension of the psychoneuroses, in fact of all behavior, sick or well. Thinking which is dominated by the reality motive, the thinking which is a conscious intentional effort at efficient relation with reality, is the thinking to which the word thinking is usually applied. But there is another kind of thinking, the thinking by fantasy formation, which is of great importance. In this form of thinking it is not the reality motive that dominates, but the pleasure-pain motive. The other horn of the conflict is here represented, and in moments of quiescence when the real world slips away from our vision and we settle back within ourselves, our thoughts flow without reference to this outside world, they come and go without critique on our part. We are dreaming, perhaps in sleep or perhaps in waking, and the fancies which come at these moments of rumination are all wish-fulfilling fancies controlled by the pleasure motive and represent the satisfaction of desires which are either put off or rendered incapable of fulfilment in the real world. These thoughts are not only the thoughts that dreams are made of, but the thoughts which the psychoneuroses are made of, and are therefore of immense importance for their understanding.

From the very first the immediate satisfaction of desire is frustrated, to be technical, it is repressed and some other form of activity has to be substituted, for example in later life, to use our same illustration, instead of maintaining the immediate relationship between hunger and food, there is introduced another series of factors, represented by work and compensation for work in the shape of money, which money may be exchanged for food. And so, instead of the immediate relationship that obtains in infancy a more remote relationship is maintained, and the activities instead of going straight

to their goal take a more or less circuitous and involved path. The original relationship therefore tends to be lost sight of, and the more involved and complicated one takes its place. There are, therefore, gradually throughout the period of development, all sorts of desires being repressed which, thus put out of consciousness, are replaced by other forms of activity. The desires which belong to infancy and which thus are early repressed and superseded by other forms of activity, constitute the material out of which the *unconscious* is formed and the material from which come the activating moments for fantasy formation. The discrepancy between desire and fulfilment, then, is compensated in later life by the wish-fulfilling fantasies that have their origin in the repressed material of infancy and occupy the realm of the unconscious.

Between this realm of the unconscious, which contains relatively infantile material only, and the realm of the clearly conscious, there lies the realm of the so-called foreconscious, which contains the material of recent experience, material which is quite easily made conscious. In other words, it is just out of mind and it is not difficult to bring back into the focus of attention when the individual so desires.

The region of the unconscious is of very great importance for an understanding of the pyschoneuroses, because it represents the region of the deepest repressions, of the thoughts that are least like the present conscious thoughts, and which, therefore, when they break through into consciousness, produce symptoms that are so grotesque and strange appearing upon the surface and non-understandable, not only to the onlooker, but to the patient himself. It is therefore desirable to know something of the nature of the unconscious and of its content. To do this certain features in the development of the child, particularly those, of course, that are at a later date repressed and superseded by other activities, will have to be described.

Before doing this, however, it is necessary to point out that all the activities of the individual lead in one of two directions, viz., in the direction of self-preservation, the nutritive activities, or in the direction of race-preservation, the sexual activities. The energy which drives toward these goals may be called the *libido*[1] and so it may be spoken of as the nutritive or the sexual libido, not as different kinds of energy but as different directions, different pathways along which the energy is being used. Now each organ in the body tends to preserve itself but, on the other hand must give some of its energies to the preservation of the whole individual just in the same way that an individual member of society while he strives to take care of himself must give something, in the way of taxes, for the general weal.

Self-preservation and race-preservation are seen thus to be fundamentally opposed to one another, the former implying getting and

[1] The word libido as indicated by its origin and as used by Freud implies and has a distinct erotic (libidinous) connotation. It is used in this sense in the text, but, because there is no other more satisfactory term it is also used in the more general sense of psychic energy, interest. The necessity for this double use is unfortunate but it is believed that the context will make it clear in each case which meaning is intended.

keeping, the latter giving. One who keeps that which he should give is called selfish.

From the few words already devoted to the evolution of the individual from the early condition of relative omnipotence it will be seen how the change has to be one of progressive socialization. For example, from the infantile immediate relationship hunger—food there is developed the more remote relationship hunger—work—money—food in conformity with the social requirements. The libido has to be socialized or to use the more frequent term, sublimated. The instincts of the individual remain the same but cultural advance depends upon a projection of these instinctive tendencies upon ends that yield less immediate concrete satisfactions and are, with the progress of social evolution, progressively more socially useful, as in the example of the instinct of hunger just cited.

In that proportion in which the individual is unable to effectually sublimate his libido, to break away from dependence upon earlier, infantile ways of pleasure seeking to higher social forms of behavior he is crippled in his capacity to live at his best, and this crippling in its milder manifestations we call the neuroses and the psychoneuroses, in its severer forms the psychoses, and in its most severe forms the various grades of defect extending to the depths of imbecility and idiocy.

It is quite obvious that the idiot and imbecile require the same sort of solicitous care as the normal infant. In the higher grades of personality defect, however, the necessity is clothed in symbols which distort and obscure the meanings in accordance with the mechanisms already described as at work in dreams (*q. v.*). For example, a patient develops the necessity of a particular dietary which can only be provided in the home and every effort to go out from the home results in a gastro-intestinal upset because of the inability to obtain it. By this symbolic mechanism the patient is therefore permitted, under the guise of invalidism, to remain, like a little child in the home situation, cared for and protected from the world of reality by the parents. In such a case the general result pointed is not only attained but analysis will show that there was an early fixation upon the gastro-intestinal ways of pleasure seeking (a so-called partial libido trend) which determines the particular form of the later symptoms.

Projection of Erotic Interests.—Then as regards the more distinctly race-preservative, or sexual libido: in early infancy the child's love is very naturally given out to the only people to all intents and purposes who constitute his milieu, namely the members of the immediate family, the father, the mother, the brother, sister, and perhaps nurse. This love, contrary to the usual way of thinking of it, is very definite in its direction, and from a very early date presents certain sexual, erotic characteristics. Of these sexual characteristics jealousy of a younger brother or sister who comes into the family and deflects a certain amount of affection which the child would otherwise enjoy is within the observation of most people, while the fact that the love of the child is given out to the members of the family, characteristically

the parent of the opposite sex, is not only a matter of common observation, but a matter of great importance psychologically. As the child develops these loves are repressed and covered into that all-inclusive amnesia for the infantile period, and when adulthood comes along and the child has grown to manhood or womanhood and finds its mate, the love which had before been spent upon the members of the family now finds its true object.

These erotic interests in the members of its family or in their surrogates are but the milestones in the pathway along which the libido has to travel to complete emancipation. The projection of the infant's erotic interests is first upon the parents, then upon brothers and sisters who are most like the parents, and progressively upon love objects less and less like the parents but in an unbroken line that can always be traced back to its parental source but which leads further and further away from any outward semblance to them until a love object is finally reached which embodies the truly emancipated ideals. All along the way there is the constant play, back and forth, of love and hate, the desire for independence and for dependency, the conflict between demands of the self and the creative demands of the race. The issue is a complex of adjustments which depends for its effectiveness upon the ease with which the fixations along the way have been dissolved and the libido has been free to move forward to higher planes of sublimation.

It is this infantile erotic fixation for the members of the family that is the root for so many of the incest phantasies of the psychoneuroses and the psychoses. This love for the parent of the opposite sex, for example, if it breaks through into the clear realm of consciousness becomes a horrid thing incompatible with the individual's peace of mind. Such things are quite common. For example, a patient marries a man who unfortunately presented a number of very close resemblances to her father. These resemblances served to stir into activity the unconscious love for the father, and she therefore, in her feelings toward her husband, is outraged beyond all endurance, for it is as if she were married to her father. Life with her husband is quite unendurable. She is constantly flying into passions, assaulting him, upbraiding him, etc.

If this psychology is the usual psychology, why is it that all people are not in danger from such sources? Perhaps they are to a limited degree, but it is necessary to bear certain things in mind to understand how the unconscious becomes mixed up, as it were, in the daily life of the individual, as in the case just cited. Individuals with such unconscious father complexes will get along in life perhaps quite well until they meet some difficulty. The difficulty drives them back within themselves, it prevents the outward flow of interest into reality, makes them egocentric, introspective, they are unable to make an efficient reaction, and they therefore are driven back to fantasy formation where things come true and the difficulties are all removed. The reason why this driving back of the psychophysical energy within

the individual under conditions of stress; the reason why this should stir up a particular complex, is because in the life of the individual there has been an undue fixation at that point in the course of development. The patient just cited had never been able to emancipate herself as she should have from the necessity for the loving care and tenderness and protection of the father and to go out into the world and, so to speak, stand upon her own feet, and when difficulties arose in her life and she was thrown back upon herself, she went back to that point at which there had been an infantile fixation, where she had previously gained satisfaction.

From the few words of description of this patient's condition, who had symbolically married her father, it will be seen how important it becomes to know the content of the fantasies, and this is best determined by a study of the dreams, and without going into the principles of dream analysis, which are out of place here, it is well to remember that the neurosis or the psychoneurosis, like the dream, is not only a compromise between desire and fulfilment, but it is a wish-fulfilling mechanism that brings to pass the fulfilment both of the wish in the foreconscious, the wish with reference to the difficulty that caused the introversion in the first place, and also the wish in the unconscious, the wish at the fixation-point, which serves as a pull-back once the introversion has started. (See Psychoanalysis in Chapter II.)

Organ Eroticism.—Previous to the period when the infant begins to project its erotic interests upon those about it, they are confined to itself and the sensations which accompany the functioning of its several organs. Pleasure and pain are derived from the skin surface as it is subjected to different degrees of temperature, as it is bruised or swathed in soft wraps; from the healthy or cramp-like contractions of the striped or the visceral muscles; from the functioning of the excretory organs; from the presence of hunger or its satiation; from terrifying or soothing sights and sounds. All of these sources of pleasure are, in the normal course of events, finally dominated by the peculiar quality and degree of that pleasure which is derived from the genital zone, and fall into their proper relationship in degree to it. If, however, in the course of development, one of these zones, these partial tendencies have been too strongly stimulated, or the pleasures derived from it have been prolonged over too long a time, there arises the danger that it will play too prominent a part in the whole group and tend to dominate unduly the situation. In so doing it not only puts its stamp upon the resulting character, which can thus be designated as dominantly oral, anal, muscle, skin, eye erotic in nature but tends also to prevent that free flow of libido to ever new and more desirable objects of interest. Such a fixation tends to keep the erotic interests self centered and to prevent their satisfactory projection upon love objects outside the self. On the other hand the concentration of erotic interests on the genital segment drains the eroticism from other segments and organs and leaves them free, as it were, to perform their allotted physiological tasks unhampered.

Bearing these facts in mind it will be easy to understand that the child's first sexual feelings have reference to its own body, more particularly to parts, segments, of its body, it is organ-erotic; that next its sexual feelings are transferred upon itself as an individual love object (the auto-erotic, narcissistic stage). Next it seeks outside itself for a love object; first seeking in its immediate surroundings and also seeking someone most like itself (the homosexual stage). It seeks, in other words, outside of itself, but still an object as much like itself as possible. And finally, the period of object love, when fulfilment is had in an entirely different individual and of a different sex (heretosexual stage).

In addition to the above facts the child not only passes through these various stages of psychosexual development mentioned, but in its earliest infantile state it is susceptible, theoretically at least, of deflection in any direction. So, for example, at the period when the love is given out to those in the immediate surroundings it not infrequently is given out to a member of the same sex; differences in sex are not appreciated in these early days and come only with later development. Other differences are equally indefinite. The erogenous zones of which the genital organs are only one, and the anus and the lips constitute the most important additional ones, are still more or less indefinite, and sexual erethism may be predominantly focalized in any one of them. And so the roots of the various so-called perversions are founded in these early fixations. The determining factor in the early fixations, the mechanisms that have brought them about, in short, their uncovering, can only be accomplished by fathoming the unconscious. This is the work of psychoanalysis and the most prominent means at its disposal at the present time is by the analysis of the dreams. (See Chapter II on Mental Examinations.)

The analysis of the symptoms not only uncovers such individual material along the lines indicated but, going deeper, brings out still more fundamental traits which find normal expression in the ways of thinking of primitive man. This is the so-called archaic material.

Bearing in mind the concept of the individual as an integrated whole and the psyche as expressing the tendencies of the whole we are prepared to find that instinctive tendencies, requiring as they do the functioning of the several organs to bring about their satisfaction, should, when thwarted (repressed), express themselves in states of physiological tension in those organs which would be called upon to function in the event the tendencies could find expression. These states of physiological tension are expressed as visceral tonicities (pylorospasm, cardiospasm, spastic constipation, increased blood-pressure, etc.) and postural tensions (bodily attitudes, peculiarities of using the limbs, facial expressions, characteristic poses, mannerisms, etc.) and the energy used in effecting the repression can be conceived as bound up in these tensions.[1]

[1] Kempf, E. J.: The Autonomic Functions and the Personality, Nervous and Mental Disease Monograph Series No. 28.

These are the mechanisms which recent studies of the autonomic apparatus and the emotions have disclosed and which serve to fill that long existing hiatus between mind and body and do away finally with the need of considering them as in any way separate and distinct. They are the mechanisms that explain that host of somatic conditions which have long been known to be of psychogenic origin and to suggest an explanation for many others whose etiology at present is obscure.

Symbolism.—All of the various difficulties of the individual—his conflicts—express themselves at the psychological level or the symbolic level as we have designated it in this book, by ideas and feelings—symbols. Just as the original tendencies of the child are relatively few so his later and more elaborated activities must be the outgrowth from these simple beginnings. A highly developed curiosity may result in a scientist, a child of marked sadistic traits may develop into a butcher or a surgeon, the child who was inordinately fond of making mud pies may later develop as an engineer or architect and these later developments can be seen to be outgrowths of the earlier tendencies and to symbolize them. We see this particularly even in those individuals whose activities are plainly attempts at compensation for obvious defects. The cryptorchid chooses a highly virile occupation—cowboy—or becomes dictatorial and overbearing; the individual with a pronounced father hate leans toward anarchistic social views; the impotent man becomes a prude or perhaps spends his time and money in trying to invent a big gun or perfect a perpetual motion machine.

One can interpret the symbol in two ways, namely, from the phylogenetic standpoint, as exhibiting the cultural stage in evolution and development exemplified, or from the ontogenetic standpoint as traceable to concrete individual experiences. For example, a person who shows his extreme mother attachment by his choice of food, living largely on milk and soft foods, shows that in that region of his conduct he is still acting like the primitive man who has faith in magic—by using the symbols of the infantile dependence upon his mother he tries to continue that dependence in existence and so get the feeling of security it gives. Such an individual, however, might, theoretically at least, have used many other symbols other than food for the same purpose. For example, he might have used the symbol of some particular style of clothing, or have retained his infantile style of handwriting, or copied a peculiar form of speech, such as a lisp or stammer, from his mother or in innumerable other ways symbolized his dependence. Which of the myriad available means he uses depends upon what his personal experience has been.

The first, the phylogenetic, or race interpretation of symbols gives us the material for the foundation of an interpretative psychology. The second, the ontogenetic or individual interpretation leads us to the actual experiences in the lives of individuals which have led to the choice of the symbol and is the method of maximum advantage in the approach to the concrete problems of the psychosis as it occurs in a particular individual.

Classification.—Although we have already dealt with the principles underlying a classification of diseases of the nervous system in the Introduction of this work, it would seem that an additional short discussion of classification as applied to diseases of the mind is warranted at this point if for no other reason than because classifications have occupied the attention of psychiatrists so greatly in the past and have been the bugbear of the student of this branch of medicine.

The concept of disease as being something that invaded the individual and possessed him like the medieval devil, has perhaps lingered here longer than in other departments of medicine, where there has been greater success in running down the etiological factor or in defining the pathological processes. But even in such a definite group as tuberculosis it is at once recognized that the organism may not only be invaded in any of its parts, but that of two organisms similarly invaded the symptomatic picture may be widely different. In other words, the picture of the so-called disease in any particular instance is dependent upon the nature of the disintegrating factor, the nature of the invaded host, and the interaction between the two. The resulting picture is but the expression of this conflict as shadowed forth in the symptoms. This is not only true in a narrow sense, but it is true in the broad sense that it is the nature of the host that in the first instance makes possible the invasion and, too, accounts for the location of that invasion. The "shut in" type of personality which manifests itself in a tendency to withdraw from the social environment and lead a cloistered existence may tend at the psychological level in the direction of precox, and at the physiological level in the direction of pulmonary tuberculosis. Some day, not far distant, we shall be able to correlate this type of personality with underlying physiological conditions (organ inferiority perhaps) which will enable us to express this relation in much more definite terms. In any case the implication is apparent that the organism, in its various reacting tendencies, expresses itself, not alone at the physiological level, but of necessity also, and undoubtedly quite as definitely, at the psychological level. The organism is a biological unit and whatever affects it in any of its parts has its psychological reverberation. Disease is the resulting picture of a process which is disintegrating in its tendencies and mental disease is that portion of the picture which is visible at the psychological level. The organism as a mechanism "goes off" as a whole and the resulting symptoms of its reaction are manifested at every reacting level and perhaps should be as accurately read at one level as another. All levels, however, need to be interrogated to get an adequate understanding of all that is going on.

Classifications in the past have only grouped those symptoms which seemed to stick together for some reason and have had little else to recommend them, founded as they were upon a purely descriptive psychiatry. The growth of an interpretative psychiatry, however, has resulted from a deeper search for causative factors and the defini-

tion of the mental mechanisms involved in the production of the symptoms and the day is rapidly approaching when mental disorders will, as a result, be classified by describing and defining the mechanisms involved and showing how, in each instance, the symptoms have come to pass as a result of their operation upon the material at hand. Finally, these mechanisms will be correlated with the organism's reactions at other than the psychological level and the reaction of the organism as a whole will be understood in terms of all the factors involved. This is the goal of present-day psychiatry and rapid strides are being made in bringing it within the range of a practical vision. It is one of the distinct efforts of the authors of this work to indicate how this is coming about by injecting all through the discussions of the disorders predominantly manifested at the different reacting levels (physicochemical, sensorimotor and symbolic) indications of how the given reactions under discussion may manifest themselves at the other levels.[1]

The various mental disorders will be considered under the usual conventional captions, but in the interpretative section the mechanisms involved will be defined and discussed in a manner that will pave the way for this larger approach to the problems of psychopathology.

It is the desire of the authors to keep the student's mind open for this broader vision, because it is appreciated that in the past, with its emphasis on classification, too much effort was expended in placing a given psychosis at a certain point in some scheme of classification, in other words, giving it a name, and only too often interest stopped when the appropriate label had been appended. Only by understanding the processes that are involved can an intelligent therapy be developed. A classificatory scheme which does this will remain sufficiently fluid to be useful rather than tending, as do the older schemes, to become static and fixed beyond the point of capacity to let in new concepts or adjust to new hypotheses.

Such a scheme of classification is that proposed by Kempf.[2] The psychoses as a group are first divided into benign and pernicious not, however, on the basis with which we are already familiar, namely, that the outcome in recovery or chronicity shall be a factor in determining the diagnosis. In such a scheme the patient whose psychosis had been difficult to diagnose was put in the manic-depressive group if he recovered but in the precox group if he did not. It is similar to the situation before the days of bacteriological diagnosis. A child with sore-throat might appear to have diphtheria but if it got well it was tonsillitis. A fatal outcome was necessary to clinch the diagnosis of diphtheria. In Kempf's scheme the qualifications benign, pernicious do not refer to the outcome alone in this way but indicate the seriousness of the psychosis as dependent upon the patient's attitude toward his difficulties.

[1] White, Wm. A.: Foundations of Psychiatry, Nerv. and Ment. Dis., Pub. Co., Washington, 1921.

[2] The Mechanistic Classification of Neuroses and Psychoses Produced by Distortion of Autonomic-Affective Functions. Jour. Nerv. and Ment. Dis., August, 1919.

FUNCTIONAL CLASSIFICATION OF NEUROSES AND PSYCHOSES.

Diagnostic classification.		Mechanistic differences.	Common symptoms.		Common causes.	Old diagnostic terms.
PERIODIC — BENIGN: tendency to accept the personal source of the wishes or cravings which cause the distress or psychosis	Suppression neuroses	Clear to vague consciousness of the nature and effect of the ungratifiable affective cravings	Distressing hypertensions or hypotensions of autonomic (visceral) segments (mild to severe)	Decrease of power to coördinate; persistent thoughts; preoccupation; unpleasant dreams; insomnia; errors; accidents; scalp pains; headache; dizziness; stiffness or weakness of external muscles of eyes, back of neck, limbs, back, tongue, pharynx; increased or decreased secretion of glands of mouth and stomach; dyspnea; tachycardia; high blood-pressure; loss of or freakishness of appetite; hyperchlorhydria; diarrhea; constipation; dysmenorrhea; amenorrhea; sexual impotence; pollakiuria; hyperirritability of diseased structures; decrease of energy, or efficiency, or ability to learn.	Fear of responsibility or liability for having or seeking the relief of vigorous cravings; fear of competition revealing functional or organic inferiorities; fear of pain, injury, loss of money, honor; fear of violating sacred traditions or transfers; love of unobtainable, unresponsive or perverse object; hate, shame, disgust for unavoidable object	Pyschasthenia (?); neurasthenia; hysteria (?); anxiety neuroses; manic-depressive types—mild; psychoneuroses; situation and accident neuroses.
	Repression neuroses	Vague consciousness to total unconsciousness of the nature and influence of the ungratifiable affective cravings	The above symptoms plus functional distortions of the projicient apparatus and sense organs	Amnesias (specific); anesthesias: specific, localized, general; hyperesthesias; paresthesias; postural tensions: spastic, flaccid; simulations of postures: functions; convulsions without loss of consciousness; elimination of segments or functions; recurring incoördinations: errors; accidents; misinterpretations: misrepresentations; fixed preferences: aversions, phobias, compulsions, obsessions (acceptable to the *ego*), mannerisms, attitudes, fetiches, symbols, rituals, habits, sexual reactions; cravings for certain stimuli: esthetic, sexual.	Same as above, but usually occurring under more acute critical, physiological or environmental conditions necessitating immediate relief or forgetting as unexpected exposure of secret vices, wishes, indulgences, scandals, asocial cravings, failures, inferiorities	Psychoneuroses; psychasthenia; hysteria; compulsions, obsessions, phobias; epileptoid tendencies; dementia precox types—incipient; war neuroses (shell shock); some types of prison neuroses.
		Persistent striving to develop potent functions and win social	Usually some of the above symptoms plus vigorous com-	Usually eccentric personal, vocational, professional, religious, artistic, mechanical, commercial, philosophical strivings; sexual	Fear of loss of sexual potency and domination by perverse crav-	Manic types; paranoid types; paraphrenia; compensa-

Acute or Chronic or	or	Compensatory neuroses	esteem initiated by fear of impotence or loss of control of asocial cravings	pulsions or inspirations to strive directly or indirectly for specific environmental social conditions and potent functions	strivings (pimps, prostitutes, seducers); penitent acts, reforms, reconstructions; solicitous interests, confessions, hyperconscientiousness; obsessions, divine inspirations, occult powers; increased muscle tensions, high blood-pressure, tachycardia, exophthalmic tensions; exhibitionistic dress, voice, manners; heedless spending, grand unsubstantiated claims; eccentric modesty, curiosity, etc.	ings; fear of failure; fear of fear; fear of loss of love object; fear of inferiority, censure, ridicule; fear of organic failure; hyperactive glands: thyroid, adrenals, sex; glycohemia; glycosuria	tory strivings in paresis, alcoholic and drug neuroses, mental defectives; psychopathic personalities; pathological lying; specific manias; compulsions; obsessions.
	Pernicious: tendency to oppose or refuse to accept the personal source of the wish or craving, to hate those who would attribute a personal source, to blame an external or impersonal cause	Regression neuroses	Failure to compensate but regression to a preceding more comfortable, irresponsible level, permitting wish-fulfilling fancies, and postures and indulgences	Distressing visceral tensions rare; persistent maintenance of affective attitudes characteristic of (See next space)	Evasion of responsibility; general inefficiency; wandering; hoboism; homesickness; indifference; apathy; childish day-dreams; lowered muscle tonus, no capacity to compensate to win esteem; muteness, amotility, indifference to cleanliness, order, system, coöperation, failure; hopeless depression, suicide; preadolescent, infantile, prenatal attitudes; indulgence in excretory erotic play, fancies, childish destructiveness	Infantile love fixations; loss of irreplaceable love object; permanent, insurmountable obstacles to the love cravings; blotting out of disastrous or shameful experiences or the adolescent development of perverse cravings, particularly autoerotic	Depressive types; involutional melancholia; "shut-in" personality; allied dementia precox types; epileptoid types.
		Dissociation neuroses	The uncontrollable cravings dominate the personality despite the efforts of the ego to prevent it	Symptoms of distressing visceral tensions, with or without functional distortions with or without eccentric defenses; compensations with (See next space)	Sensory derangements: delusions, hallucinations, environmental and social disorientation, uncontrollable preoccupation, confusion, delirium, stupor; anxiety, apprehension, panic at loss of self-control; wild or systematic compensatory striving, bluffing, raging as a defense against the uncontrollable cravings; defensive compulsion to systematize signs, remarks, proofs of exogenous influences; uncontrollable obsessions, phobias, mannerisms, impulses, postures, rituals, fancies (distressing to the *ego*); heedless, yielding to excretory erotic cravings, fancies of preadolescent or infantile nature	Irrepressible, uncontrollable asocial or perverse segmental cravings; inability to prevent repressed, disguised cravings from breaking through the ego's resistance because of fatigue, discouragement, depressing domination by others, seduction, toxemia, physical shock, unsuitable means of sublimating, metabolic dystrophies	Hallucinations, deliria due to toxemias, etc.; functional epilepsy (hallucinated); hysterias, manic-depressives (hallucinated); dementia precox (hallucinated); paranoid, catatonic, hebephrenic, simple, mixed.

The psychoses are then divided into suppression, repression, compensatory, regression and dissociation types. The meanings of these terms are sufficiently set forth in the table. They represent a scale in the seriousness of the psychological difficulties. It is important to understand, in using these terms, that they are not mutually exclusive; that dissociation, for example, is a result of severe regression, and that a patient may begin with a compensation psychosis but gradually lose ground and finally develop a dissociation, or, on the other hand, come under observation as a dissociation psychosis but get better and present the features of regression and suppression types on the way to recovery. In the psychiatry of the nineteenth century a simple melancholia today might be a hallucinated melancholia tomorrow, a change in name with no adequate corresponding appreciation of what the change in symptoms might mean. In this example, for instance, the breaking through of the repressed material and its resymbolization as the hallucinations. In this scheme, however, it is not the apparent symptoms which are used as a basis of classification but the mechanisms which produce them.

Southard[1] has made a very practical descriptive grouping of the great classes of the psychoses under eleven heads as follows:

1. Syphilitic	Syphilopsychoses
2. Feeble-minded	Hypophrenoses
3. Epileptic	Epileptoses
4. Alcoholic, drug, poison	Pharmacopsychoses
5. Focal brain ("organic," arteriosclerotic)	Encephalopsychoses
6. Bodily disease ("symptomatic")	Somatopsychoses
7. Senescent, senile	Geriopsychoses
8. Dementia precox, paraphrenic	Schizophrenoses
9. Manic-depressive, cyclothymic	Cyclothymoses
10. Hysteric, psycho-, neurasthenic	Psychoneuroses
11. Psychopathic, paranoiac, et al.	Psychopathoses

In the columns which follow giving the principal symptoms of these conditions, those physiological disturbances of function are set forth with regard to which there is no serious controversy. In the future development of psychiatry there will undoubtedly be an enormous advance of our knowledge of the specific correlation of psychic states, and physiological states along the lines indicated here and elsewhere throughout this book. At least it must be apparent that for an accurate sizing-up of an individual it is as important to adequately evaluate the meanings of his bodily attitudes (visceral, postural) as it is to evaluate his attitudes of mind.

[1] A Key to the Practical Grouping of Mental Diseases, Jour. Nerv. and Ment. Dis., January, 1918.

CHAPTER XV.

THE ACTUAL NEUROSES AND PSYCHONEUROSES.

The field of the neuroses and the psychoneuroses is not only the broadest field in psychiatry, but perhaps the broadest field in all medicine. Not only is the field an extensive one in point of the actual number of persons who suffer from these afflictions, but it is a field of very great importance for the understanding of mental phenomena both in the realm of disease and in the realm of the healthy. It is in the manifestations of these disorders, which have been well termed borderland states, that the early departures from the normal can be found and studied, which, in much more aggravated form, appear in the psychoses.

Then again problems that lend themselves much more satisfactorily to therapeutic attack are to be found here, conditions which, although they may represent practically any degree of departure from the normal, are, as a rule, capable of material alleviation, if not actual cure by therapeutic measures. When the immense number of people who are affected by neuroses or psychoneuroses is considered, the great amount of suffering that these diseases entail, the impaired efficiency in which they result, and then consider that they are, for the most part, susceptible of great improvement, if not actual cure by therapeutic endeavor, it will be seen that this department of medicine is not only the most attractive, but is one which perhaps offers most in the way of results.

The number of people actually afflicted with these conditions is difficult to estimate. The frank cases of the psychoneuroses and the actual neuroses are very numerous, as are also more or less larvated conditions, while on the other hand, every specialist in medicine is dealing constantly with manifestations of these conditions as they appear upon the physical side. Perhaps these physical manifestations are best known to the gastro-enterologist, the gynecologist, and the genito-urinary surgeon, but the ophthalmologist, the laryngologist, the internist, and in fact every specialist has his share. From the viewpoint of the organism as a whole it must be at once evident that every individual who is sufficiently sick to have his general efficiency appreciably impaired must present mental symptoms, must be also mentally ill, but the authors also believes that the proportion of patients who present mental symptoms of etiological significance in their illness is vastly larger than is generally believed.

In the following chapters the disorders at the symbolic (psychological) level will be discussed. It is a fundamental tenet of this book, however, that the three levels treated herein, viz., the vegetative, the sensorimotor, and the symbolic are not mutually distinct but only

different aspects of the strivings of the individual and the component parts thereof as expressed by the various forms of solutions and compromises made in the processes of integration and adjustment. (See introduction.) Not only are the different levels not distinct, but the same symptom may arise as a result of disturbance at any one of the levels. For example constipation may be due to a disturbance at the vegetative level (vagotonic spastic constipation), at the sensorimotor level, due to injury or disease of the cord, or at the symbolic level, a purely psychogenic symptom. Further than this, purely emotional causes if severe or of long duration may produce structural changes from which recovery is very slow or impossible or may precipitate, by affording favorable conditions, severe, even fatal infections. In all of these conditions the individual, as such, is more or less severely crippled but the understanding of the symptoms and their proper treatment must depend upon a comprehension of the mechanisms involved in their production.

THE ACTUAL NEUROSES. (NEURASTHENIA AND ANXIETY NEUROSIS, HYPOCHONDRIA AND TIC.)

Neurasthenia.—The term neurasthenia, since it first came into use only a little over a generation ago, has been applied to almost every conceivable condition. Almost all illnesses are accompanied by a certain amount of easy fatigability, emotional instability, and a general out-of-sorts feeling. All combinations of this kind which cannot be specifically diagnosed and placed under some well-known caption are easily dropped into the miscellaneous group of neurasthenia. Not only have all sorts of conditions, therefore, been included under this term, but the most varied symptoms have been thereby designated as neurasthenic. Not only have the more pronounced physical conditions been included, such as general arteriosclerosis, but it is not uncommon for some of the more serious psychoses, especially in their milder manifestations, as the cyclothymias, to be diagnosed as neurasthenia. It is highly desirable, therefore, to limit the application of the term to a definite condition.

This condition is known as a primary fatigue neurosis and has certain quite characteristic and constant symptoms which are in the main a feeling of pressure on the top of the head, more or less insomnia, spinal irritation, with perhaps pain in the back, certain paresthesias, and gastro-intestinal symptoms, easy fatigability, difficulty in fixing the attention and in application to work, emotional irritability, and some depression. This condition is commonly seen following or accompanying exhausting experiences or severe illnesses. It is seen, for example, after typhoid and influenza and as a sequel to prolonged mental strain and overwork. In such instances, however, it is secondary in origin to these more or less obvious factors.

Despite outward evidence to the contrary it has been traced in many instances where careful analysis of the symptoms has been made, and

when there are no such obvious etiological factors as those just mentioned, to a specific sexual etiology, namely, to excessive masturbation or frequent pollutions or to their psychological equivalents, namely, auto-erotic phantasies. It is the auto-erotic fixation, the inability to get beyond that period of development in which pleasure is sought in self, that is the serious matter rather than the concrete evidences of this fixation in any particular acts. In contrast to the etiology of the anxiety neurosis, which, as will be seen, is dependent upon an excessive excitation but inadequate utilization and incomplete discharge of the energy of the sexual act in the psychic sphere, in neurasthenia the specific etiology is dependent upon a deficient external stimulation and excessive though inadequate discharge in the physical sphere.

One has to think in addition to the specific etiology of the fact that in most instances where masturbation is practised into adulthood there is a serious moral conflict. The individual feels ashamed, chagrined, humiliated by having yielded to the physical demand, but perhaps deeper down are tendencies to perversion and incestuous acts. This, of course, adds to the difficulty by increasing the amount of energy dissipated. In addition to this the moral conflict is usually very greatly enhanced either by being told or reading of the awful results of this habit. This is especially so when these results are told to the child in order to frighten him into desisting and are accompanied by threats of cutting off the organ and the like. From this point of view the term neurasthenia is used in a very restricted sense. The obvious question raised by these considerations is whether this specific etiology must be present in all cases? Are other causes, such as severe illness and overwork, only capable of producing neurasthenic symptoms upon such a background? Whether or not this is so it is obvious that the secondary neurasthenias disappear when the primary factors can be dissipated. Rest following over work is usually adequate treatment. It would only be when this has failed that other factors should be sought.

It must not be lost sight of in dealing with this class of patients that a moderate amount of masturbation during infancy, about the third or fourth year, at the end of what Freud calls the first latency period, is normal and probably has as its function the focalization of the sexual erethism upon the sex organs. It will be remembered that before this time the various erogenous zones of the body such as the sex organs, the lips, the anus, are of practically equal significance. For the function of reproduction the sex organs must emerge with a preponderant erethism, otherwise some one of the perversions will take the place of normal sexuality. It seems, therefore, the function of masturbation to help produce this result.

When masturbation, however, is indulged in about the period of puberty and later on into adult life it has certain dangers in addition to those which are more nearly at the physiological level and which are productive of neurasthenia. The individual in his psychosexual development passes through an auto-erotic period when he finds his

sexual interests in himself, then through a period in which his sexual interests are transferred to the immediate members of the family the period of narcissism in which at first he is most interested in those members of the family most like himself, namely of the same sex. Passing through these auto-erotic homosexual and narcissistic periods he finally reaches, after having passed the period of puberty, to the possibility of giving his love out to someone else, not only besides himself, but someone removed from the family circle and someone of the opposite sex. He becomes normally heterosexual and attains the period of object love. Now one of the serious dangers of masturbation is the danger it has of fixing the individual at some intermediate point in his psychosexual development. The principal danger is, of course fixation at the infantile auto-erotic period, which is naturally the particular quality of sexuality that masturbation ministers to. This fixation not only prevents the proper psychosexual development, but drags the whole personality back upon itself and prevents that open, free, and outward manifestation which is essential to success in life, to a finding of one's place in the world. These people are too thoroughly occupied with themselves to be able to deal with the outside world of reality with any degree of efficiency. An important aspect of the world of reality is the social aspect. Sublimation tends always toward socialization so that a fixation which interferes with attaining a position of social independence as a useful and respected member of the herd withholds the premium of social esteem and the indulger in auto-erotic pleasures feels, in consequence, that he has lost the respect of his fellows.

In the act of masturbation the individual is both the subject and the object. He has to supply the energies from both sources, not only the energies from within, but all of the energies and stimuli which normally would come from without from the person of another. The term masturbation, from these considerations, it will be seen, must be considerably broadened in its meaning. Masturbation is an essentially auto-erotic phenomenon. From this point of view, sexual intercourse, which has only the meaning of self-indulgence, is masturbation. Intercourse only reaches its full biological significance when, in addition to affording individual gratification, it gives something which is of social benefit. Concretely, of course, this is the case when its object is the production of a child. Equally is this true when it is an expression of love which serves to deepen the union and mutual understanding between two persons so that they become of greater service to each other and thus, by their combined and reinforced efforts, to the race. During his indulgences the masturbator develops all manner of fantasies and these fantasies often throw a considerably light upon the nature of his psychosexual development, quite similarly as do the dreams in those patients who suffer from frequent pollutions. Menzies[1] has indicated the complex origin of these

[1] Auto-erotic Phenomena in Adolescence. An Analytical Study of the Psychology and Psychopathology of Onanism, Paul B. Hoeber, New York.

phantasies in his analysis of the psychology of masturbation. He thinks they can be included as reactions to a infantile coprophilia (self-disgust, masturbation apperceived as "dirty"); (*b*) the castration complex (guilty dread from disobedience of parental prohibitions. Masturbation apperceived as dangerous; (*c*) the œdipus complex (associations with the proscriptions of the "incest barrier"—masturbation apperceived as "immoral" or "indecent"). The sources of the masturbatic impulse he traces to: (1) Fixation in infantilism; (2) revolt against parental authority and incestuous fixation; (3) sexual tension.

It will be seen, therefore, that in neurasthenia there is a condition that is by no means simple and that requires careful analytical study in order to be able to deal with it intelligently.

A final word as to the causes of neurasthenia: The alleged causes of this disorder have been as multiform as the conditions which have been ranged under it. There are a large group of cases which are supposed to be dependent upon injuries, traumatic neurasthenia, and another large group that is supposed to be dependent upon overwork. Although it is not quite possible to speak dogmatically with regard to the traumatic group at this time, still from analogy, as the result of cases studied in the group supposed to be dependent upon overwork, it will be seen that the same reasoning applies to both and it is again the same reasoning that may apply to anxiety neurosis, or in fact to any of the conditions described in this chapter, but more particularly perhaps to the actual neuroses. The traumatism or the overwork, as the case may be, or any other apparent assigned cause can be said not to be the true cause of the neurosis, but only its occasion. The traumatism or the overwork could not produce the neurosis in the absence of the specific etiology. It is quite understandable that a given individual may stand a series of sexual traumatisms over a considerable period of time, but be strong enough to resist the development of a neurosis. On the occasion, however, of having his resistance reduced as the result of an injury, or as the result of long-continued overwork the neurosis crops out. This is the explanation for many of these conditions, and it is the reason why a banal cause may develop a result that is out of all proportion in both *quantity and quality*.

Anxiety Neurosis.—The anxiety neurosis was separated from the general group of actual neuroses and psychoneuroses by Freud. The name anxiety neurosis indicates that the symptoms all group themselves about the cardinal symptom of anxiety, and it is significant of this anxiety that, while it is a psychic fact, it is still not of psychic but of somatic origin. The anxiety, therefore, is not susceptible of being analyzed into psychic components, but its source can only be found at the physiological level. This anxiety arising at the physiological level and manifesting itself in the psychic sphere then becomes a "free-floating anxiety" which may attach itself to any idea and therefore appear to be of psychic origin. On the other hand, it may express itself simply as anxiety without ideational content.

Anxiety may thus be seen to be the correlative of fear. While fear is the emotion which correponds to a danger threatening the organism from outside, anxiety corresponds to a danger which threatens the organism from within.

Anxiety is thus seen to be very different from fear which is an affect related very definitely to an external object which can be attacked or run away from. Anxiety, with its unconscious and somatic roots is much more difficult for the organism to deal with as it has no ready made mechanism for fight or flight. One reason it becomes attached to external objects is to enable these fight and flight mechanisms to come into play.

It will help to make understandable what has to be said about the anxiety neurosis if it is understood at the start that the whole sex relationship which is consummated by the sexual act consists of two parts, a somatic and a psychic. In contrast to neurasthenia, which results when the discharge of energy is inadequate upon the somatic side, anxiety neurosis occurs whenever the discharge is inadequate in the psychic sphere.

Symptoms.—The following is the description of the symptomatology of anxiety neurosis as given by Freud:[1]

"1. *General Irritability.*—This is a frequent nervous symptom, common as such to many nervous states. It is mentioned here because it constantly occurs in the anxiety neurosis and is of theoretical significance. Increased irritability always points to an accumulation of excitement or to an inability to bear accumulation, hence to an absolute or relative accumulation of excitement. The expression of this increased irritability through an auditory hyperesthesia is especially worth mentioning; it is an oversensitiveness for noises, which symptom is certainly to be explained by the congenital intimate relationship between auditory impressions and fright. Auditory hyperesthesia is frequently found as a cause of insomnia, of which more than one form belongs to anxiety neurosis.

"2. *Anxious Expectation.*—I cannot better explain the condition that I have in mind than by this name and by some appended examples. A woman, for example, who suffers from anxious expectation thinks of influenza-pneumonia whenever her husband, who is afflicted with a catarrhal condition, has a coughing spell; and in her mind she sees a passing funeral procession. If on her way home she sees two persons standing together in front of her house she cannot refrain from the thought that one of her children fell out of the window; if she hears the bell ring she thinks that someone is bringing her mournful tidings, etc., yet in none of these cases is there any special reason for exaggerating a mere possibility.

"The anxious expectation naturally reflects itself constantly in the normal, and embraces all that is designated as 'uneasiness and a tendency to a pessimistic conception of things,' but as often as

[1] Loc. cit.

possible it goes beyond such a plausible uneasiness, and it is frequently recognized as a part of constraint even by the patient himself. For one form of anxious expectation, namely, that which refers to one's own health, we can reserve the old name of hypochondria. Hypochondria does not always run parallel with the height of the general anxious expectation; as a preliminary stipulation it requires the existence of paresthesias and annoying somatic sensations. Hypochondria is thus the form preferred by the genuine neurasthenics whenever they merge into the anxiety neurosis, a thing which frequently happens.

"As a further manifestation of anxious expectation we may mention the frequent tendency observed in morally sensitive persons to pangs of conscience, scrupulosity, and pedantry, which varies, as it were, from the normal to its aggravation as doubting mania.

"Anxious expectation is the most essential symptom of the neurosis; it also clearly shows a part of its theory. It can perhaps be said that we have here a quantum of freely floating anxiety which controls the choice of ideas by expectation and is forever ready to unite itself with any suitable ideation.

"3. This is not the only way in which the anxiousness, usually latent but constantly lurking in consciousness, can manifest itself. On the contrary it can also suddenly break into consciousness without being aroused by the issue of an idea, and thus provoke an attack of anxiety. Such an attack of anxiety consists of either the anxious feeling alone without any associated idea, or of the nearest interpretation of the termination of life, such as the idea of 'sudden death' or threatening insanity; or the feeling of anxiety becomes mixed with some paresthesia (similar to the hysterical aura); or finally the anxious feeling may be combined with a disturbance of one or many somatic functions such as respiration, cardiac activity, the vasomotor innervation, and the glandular activity. From this combination the patient renders especially prominent now this and now the other moment. He complains of 'heart spasms,' 'heavy breathing,' 'profuse perspiration,' 'inordinate appetite,' etc., and in his description the feeling of anxiety is put to the background or it is rather vaguely described as 'feeling badly,' 'uncomfortably,' etc.

"4. What is interesting and of diagnostic significance is the fact that the amount of admixture of these elements in the attack of anxiety varies extraordinarily, and that almost any accompanying symptom can alone constitute the attack as well as the anxiety itself. Accordingly there are rudimentary attacks of anxiety, and equivalents for the attack of anxiety, probably all of equal significance in showing a profuse and hitherto little appreciated richness in forms. A more thorough study of these larvated states of anxiety (Hecker) and their diagnostic division from other attacks ought soon to become the necessary work for the neuropathologist.

"I now add a list of those forms of attacks of anxiety with which I am acquainted. There are attacks:

"(*a*) With disturbances of heart action, such as palpitation with transitory arrhythmia, with longer-continued tachycardia up to grave states of heart weakness, the differentiation of which from organic heart affection is not always easy; among such we have the pseudo-angina pectoris, a delicate diagnostic sphere.

"(*b*) With disturbances of respiration, many forms of nervous dyspnea, asthma-like attacks, etc. I assert that even these attacks are not always accompanied by conscious anxiety.

"(*c*) Of profuse perspiration, often nocturnal.

"(*d*) Of trembling and shaking which may readily be mistaken for hysterical attacks.

"(*e*) Of inordinate appetite, often combined with dizziness.

"(*f*) Of attack-like appearing diarrhea.

"(*g*) Of locomotor dizziness.

"(*h*) Of so-called congestions, embracing all that was called vaso-motor neurasthenia.

"(*i*) Of paresthesias (these are seldom without anxiety or a similar discomfort).

"5. Very frequently the nocturnal frights (pavor nocturnus of adults) usually combined with anxiety, dyspnea, perspiration, etc., is nothing other than a variety of the attack of anxiety. This disturbance determines a second form of insomnia in the sphere of their anxiety neurosis. Moreover, I became convinced that even the pavor nocturnus of children evinces a form belonging to the anxiety neurosis. The hysterical tinge and the connection of the fear with the reproduction of appropriate experience or dream, makes the pavor nocturnus of children appear as something peculiar, but it also occurs alone without a dream or a recurring hallucination.

"6. '*Vertigo.*'—This, in its lightest forms better designated as 'dizziness,' assumes a prominent place in the group of symptoms of anxiety neurosis. In its severer forms the 'attack of vertigo,' with or without fear, belongs to the gravest symptoms of the neuroses. The vertigo of the anxiety neurosis is neither a rotatory dizziness nor is it confined to certain planes or lines like Ménière's vertigo. It belongs to the locomotor or coördinating vertigo, like the vertigo in paralysis of the ocular muscles; it consists in a specific feeling of discomfort which is accompanied by sensations of a heaving ground, sinking legs, of the impossibility to continue in an upright position, and at the same time there is a feeling that the legs are as heavy as lead, they shake, or give way. This vertigo never leads to falling. On the other hand, I would like to state that such an attack of vertigo may also be substituted by a profound attack of syncope. Other fainting-like states in the anxiety neurosis seem to depend on a cardiac collapse.

"The vertigo attack is frequently accompanied by the worst kind of anxiety and is often combined with cardiac and respiratory disturbances. Vertigo of elevations, mountains and precipices, can also be frequently observed in anxiety neurosis; moreover, I do not know whether we are still justified in recognizing a vertigo of stomachic origin.

"7. On the basis of the chronic anxiousness (anxious expectation) on the one hand, and the tendency to vertiginous attacks of anxiety on the other, there develop two groups of typical phobias; the first refers to the general physiological menaces, while the second refers to locomotion. To the first group belong the fear for snakes, thunderstorms, darkness, vermin, etc., as well as the typical moral overscrupulousness, and the forms of doubting mania. Here the available fear is merely used to strengthen those aversions which are instinctively implanted in every man. But usually a compulsively acting phobia is formed only after a reminiscence is added to an experience in which this fear could manifest itself; as, for example, after the patient has experienced a storm in the open air. To attempt to explain such cases as mere continuations of strong impressions is incorrect. What makes these experiences significant and their reminiscences durable is after all only the fear which could at that time appear and can also appear today. In other words, such impressions remain forceful only in persons with 'anxious expectations.'

"The other group contains agoraphobia with all its accessory forms, all of which are characterized by their relation to locomotion. As a determination of the phobia we frequently find a precedent attack of vertigo; I do not think that it can always be postulated. Occasionally, after a first attack of vertigo without fear, we see that though locomotion is always accompanied by the sensation of vertigo, it remains possible without any restrictions, but as soon as fear attaches itself to the attack of vertigo, locomotion fails, under the conditions of being alone, narrow streets, etc.

"The relation of these phobias to the phobias of obsessions, which mechanism I discussed above,[1] is as follows: The agreement lies in the fact that here as there, an idea becomes obsessive through its connection with an available affect. The mechanism of transposition of the affect therefore holds true for both kinds of phobias. But in phobias of the anxiety neurosis this affect is (1) a monotonous one, it is always one of anxiety; (2) it does not originate from a repressed idea, and on psychological analysis it proves itself not further reducible nor can it be attacked through psychotherapy. The mechanism of substitution does not therefore hold true for the phobias of anxiety neurosis.

"Both kinds of phobias (or obsessions) often occur side by side, though the atypical phobias which depend on obsessions need not necessarily develop on the basis of anxiety neurosis. A very frequent ostensibly complicated mechanism appears if the content of an original simple phobia of anxiety neurosis is supplanted by another idea, the substitution is then subsequently added to the phobia. The 'protective measures' originally employed in combating the phobia are most frequently used as substitutions. Thus, for example, from the effort to provide one's self with counter-evidence that one is not crazy,

[1] Die Abwehr-Neuropsychosen, Neurol. Centralbl., 1894, Nos. 10 & 11.

contrary to the assertion of the hypochondriacal phobia, there results a reasoning mania. The hesitations, doubts, and the many repetitions of the *folie du doute* originate from the justified doubt concerning the certainty of one's own stream of thoughts, for, through the compulsive-like idea one is surely conscious of so obstinate a disturbance, etc. It may therefore be claimed that many syndromes of compulsion neurosis, like *folie du doute* and similar ones, can clinically, if not notionally, be attributed to anxiety neurosis.[1]

"8. The digestive functions in anxiety neurosis are subject to very few but characteristic disturbances. Sensations like nausea and sickly feeling are not rare, and the symptom of inordinate appetite alone or with other congestions, may serve as a rudimentary attack of anxiety. As a chronic alteration analogous to the anxious expectations one finds a tendency to diarrhea which has occasioned the queerest diagnostic mistakes. If I am not mistaken it is this diarrhea to which Möbius[2] has recently called attention in a small article. I believe, moreover, that Peyer's[3] reflex diarrhea which he attributes to a disease of the prostate is nothing other than the diarrhea of anxiety neurosis. The deceptive reflex relation is due to the fact that the same factors which are active in the origin of such prostatic affections also come into play in the etiology of anxiety neurosis.

"The behavior of the gastro-intestinal function in anxiety neurosis shows a sharp contrast to the influence of this same function in neurasthenia. Mixed cases often show the familiar 'fluctuations between diarrhea and constipation.' The desire to urinate in anxiety neurosis is analogous to the diarrhea.

"9. The paresthesias which accompany the attack of vertigo or anxiety are interesting because they associate themselves into a firm sequence, similar to the sensations of the hysterical aura. But in contrast to the hysterical aura I find these associated sensations atypical and changeable. Another similarity to hysteria is shown by the fact that in anxiety neurosis a kind of conversion[4] into bodily sensations, as, for example, into rheumatic muscles, takes place which otherwise can be overlooked at one's pleasure. A large number of so-called rheumatics, who are moreover demonstrable as such, really suffer from an anxiety neurosis. Besides this aggravation of the sensation of pain I have observed in a number of cases of anxiety neurosis a tendency toward hallucinations which could not be explained as hysterical.

"10. Many of the so-called symptoms which accompany or supersede the attack of anxiety also appear in a chronic manner. They are then still less discernible, for the anxious feeling accompanying them appears more indistinct than in the attack of anxiety. This especially holds true for the diarrhea, vertigo, and paresthesias. Just

[1] Obsessions et phobies, Révue neurologique, 1895.
[2] Neuropathologische Beiträge, 1894. 2. Heft.
[3] Die nervösen Affektionen des Darmes, Wiener Klinik, 1893.
[4] Freud: Abwehr-Neuropsychosen.

as the attack of vertigo can be superseded by an attack of syncope, so can the chronic vertigo be superseded by the continuous feeling of feebleness, lassitude, etc."

Of great importance is the somatic symptomatology. The patient characteristically stresses the somatic symptoms and these cases are often treated as suffering from some obscure organic disease. What has already been said (Introduction) about the relation of visceral tonicities, postural tensions and organ eroticism to emotional states will serve to explain the somatic symptoms.

The Etiology and Occurrence of Anxiety Neurosis.—The following remarks on the etiology and occurrences of anxiety neurosis are quoted from Freud's original paper:[1]

"In some cases of anxiety neurosis no etiology can readily be ascertained. It is noteworthy that in such cases it is seldom difficult to demonstrate a marked hereditary taint.

"Where we have reason to assume that the neurosis is acquired we can find by careful and laborious examination that the etiologically effective moments are based on a series of injuries and influences from the sexual life. These at first appear to be of a varied nature but easily display the common character which explains their homogeneous effect on the nervous system. They are found either alone or with other banal injuries to which a reinforcing effect can be attributed. This sexual etiology of anxiety neurosis can be demonstrated so preponderantly often that I venture for the purpose of this brief communication to set aside all cases of a doubtful or different etiology.

"For the more precise description of the etiological determinations under which anxiety neurosis occurs, it will be advisable to treat separately those occurring in men and those occurring in women. Anxiety neurosis appears in women—disregarding their predisposition—in the following cases:

"(*a*) As virginal fear or anxiety in adults. A number of unequivocal observations showed me that an anxiety neurosis, which is almost typically combined with hysteria, can be evoked in maturing girls, at the first encounter with the sexual problem, that is, at the sudden revelation of the things hitherto veiled, by either seeing the sexual act, or by hearing or reading something of that nature.

"(*b*) As fear in the newly married. Young women who remain anesthetic during the first cohabitation not seldom merge into an anxiety neurosis which disappears after the anesthesia is displaced by the normal sensation. As most young women remain undisturbed through such a beginning anesthesia, the production of this fear requires determinants which I will mention.

"(*c*) As fear in women whose husbands suffer from ejaculatio precox or from diminished potency.

"(*d*) In those whose husbands practise coitus interruptus or reservatus. These cases go together, for on analyzing a large number of

[1] Loc. cit.

examples one can easily be convinced that they only depend on whether the woman attained gratification during coitus or not. In the latter case one finds the determinant for the origin of anxiety neurosis. On the other hand, the woman is spared from the neurosis if the husband afflicted by ejaculatio precox can repeat the congress with better results immediately thereafter. The congressu reservatus by means of the condom is not injurious to the woman if she is quickly excited and the husband is very potent; in other cases the noxiousness of this kind of preventive measure is not inferior to the others. Coitus interruptus is almost regularly injurious; but for the woman it is injurious only if the husband practises it regardless, that is, if he interrupts coitus as soon as he comes near ejaculating without concerning himself about the determination of the excitement of his wife. On the other hand, if the husband waits until his wife is gratified, the coitus has the same significance for the latter as a normal one; but then the husband becomes afflicted with an anxiety neurosis. I have collected and analyzed a number of cases which furnished the material for the above statements.

"(*e*) As fear in widows and intentional abstainers, not seldom in typical combination with obsessions.

"(*f*) As fear in the climacterium during the last marked enhancement of the sexual desire.

"The cases (*c*), (*d*), and (*e*) contain the determinants under which the anxiety neurosis originates in the female sex most frequently and most independently, of hereditary predisposition. I will endeavor to demonstrate in these—curable, acquired—cases of anxiety neurosis that the discovered sexual injuries really represent the etiological moments of the neurosis. But before proceeding I will mention the sexual determinants of anxiety neurosis in men. I would like to formulate the following groups, every one of which finds its analogy in women:

"(*a*) Fear of the intentional abstainers; this is frequently combined with symptoms of defense (obsessions, hysteria). The motives which are decisive for intentional abstinence carry along with them the fact that a number of hereditarily burdened eccentrics, etc., belong to this category.

"(*b*) Fear in men with frustrated excitement (during the engagement period), persons who out of fear for the consequences of sexual relations satisfy themselves with handling or looking at the woman. This group of determinants which can moreover be transferred to the other sex—engagement periods, relations with sexual forbearance —furnish the purest cases of neurosis.

"(*c*) Fear in men who practise coitus interruptus. As observed above, coitus interruptus injures the woman if it is practised regardless of the woman's gratification; it becomes injurious to the man if in order to bring about the gratification in the woman he voluntarily controls the coitus by delaying the ejaculation. In this manner we can understand why it is that in couples who practise coitus interruptus

it is usually only one of them who becomes afflicted. Moreover, the coitus interruptus only rarely produces in man a pure anxiety neurosis, usually it is a mixture of the same with neurasthenia.

"(*d*) Fear in men in the senium. There are men who show a climacterium like women, and merge into an anxiety neurosis at the time when their potency diminishes and their libido increases.

"Finally I must add two more cases holding true for both sexes:

"(*e*) Neurasthenics merge into anxiety neurosis in consequence of masturbation as soon as they refrain from this manner of sexual gratification. These persons have especially made themselves unfit to bear abstinence.

"What is important for the understanding of the anxiety neurosis is the fact that any noteworthy development of the same occurs only in men who remain potent, and in non-anesthetic women. In neurasthenics, who on account of masturbation have markedly injured their potency, anxiety neurosis as a result of abstinence occurs but rarely and limits itself usually to hypochondria and light chronic dizziness. The majority of women are really to be considered as 'potent;' a real impotent, that is, a real anesthetic woman, is also inaccessible to anxiety neurosis, and bears strikingly well the injuries cited.

"How far we are perhaps justified in assuming constant relations between individual etiological moments and individual symptoms from the complex of anxiety neurosis, I do not care to discuss here.

"(*f*) The last of the etiological determinants to be mentioned seems, in the first place, really not to be of a sexual nature. Anxiety neurosis originates in both sexes through overwork, exhaustive exertion, as, for instance, after sleepless nights, nursing the sick, and even after serious illnesses."

Hypochondria.—In hypochondria there is an abnormal sensitiveness to the functioning of the internal organs of the body in contrast to the sensitiveness of the external parts in fixation hysteria. The gastro-intestinal tract is more frequently selected and so these patients occupy themselves a great deal of the time with a consideration of the functions of digestion and the elaboration of dietary schedules. The fixation here is at the level of what has been defined as organ eroticism (Introduction).

Tic.[1]—This rather obscure symptomatological group is still perhaps best briefly described in the words of Trousseau's classic definition.[2] "Painless tic consists of momentary twitching of lightning rapidity confined, as a rule, to a small group of muscles, usually those of the face, though the muscles of the neck, the trunk, the limbs can also be affected. . . . With one patient there may occur blinking of the lids, a twitch of the cheeks, the nostrils, or the lips, that makes one think he is pulling faces; with another the head nods or there is a sudden

[1] Meige, H., and Feindel, E.: Les Tics et Leur Traitement, Paris, Masson and Co., 1902. Ferenczi, S.: Psychoanalytical Observations on Tic, Internat. Jour. of Psychoanalysis, March, 1921.

[2] Trousseau: Clinique méd. de l'Hotel-Dieu, 1873, vol. 2.

and repeated twist of the neck, with a third a shrug of the shoulders, a spasmodic movement of the abdominal muscles or of the diaphragm; in short it is an unceasing series of bizarre movements that defy description. . . . In some cases the tic is accompanied by a cry or by a more or less loud vocal sound. The tic may consist entirely of this very characteristic laryngeal or diaphragmatic chorea. There also occurs a very strange propensity to reiterate a cry or a word; the patient will even utter words in a loud voice which he would rather keep back. . . ."

From this description it can be appreciated how this manifestation, which has been various designated as "convulsive tic," "maladie des tics," and when accompanied by echolalia and coprolalia, "maladie de Gilles de la Tourette,[1] is a very complex series of phenomena which, while they may occur apart and by themselves as it were, and constitute a very special and separate problem, may also occur symptomatically in the course of neuro-psychic disorders.

The work of Ferenczi[2] is most suggestive as indicating the probable mechanisms involved and clearing up the nosological difficulties. He sees in tic a symptom that can only be adequately understood in terms of narcissistic libido. Tic is a defensive mechanism developed to ward off threatened injury to the body. This "narcissistic disease" produces a fixation of the libido upon single organs under these conditions: (*a*) Danger to life or the menace of a trauma; (*b*) injury of a part of the body already heavily charged with libido (an erotogenic zone); (*c*) constitutional narcissism when the smallest injury to a part of the body strikes the whole ego. There are various ways in which the patient may ward off suffering. The simplest way is to withdraw from the stimulus. Corresponding to this method are a series of tics that Ferenczi would call flight reflexes. The climax of this form of reaction is reached in the general negativism of catatonia. A more complicated tic repeats active defense movements against a disturbing stimulus. A third form is directed against the person of the patient. Here are found various tics of which the scratching tics is the type. The culmination of this type of reaction is reached in the self-mutilations seen so frequently in schizophrenia.

Nosologically it would seem that tic is of narcissistic-organic nature. There is a "damming up of organ libido," an undue occupation of the interest with the patients own body, a hypersensitiveness with resulting inability to endure a physical stimulus without a defense reaction. Tic is thus allied with fixation hysteria, hypochondria, and dementia precox and with such symptoms as occupation cramp, catatonia, mannerisms, stereotypies, habit spasm, coprolalia, echolalia, grimacing, negativism, rigidity, mutism, phonator tics, and perhaps in many other motor manifestations the tic mechanism may be a component even if it is not adequate as a complete explanation.

[1] Gilles de la Tourette: Études sur une affection nerveuse, caractérisée par l'incoordination motrice, et accompagnée d'echolalie et coprolalie, Arch. de Neurologie, 1885.

[2] Loc. cit.

As might be expected, from its organ-fixation nature, tic is notoriously resistive to treatment. When not complicating severe psychosis (schizophrenia) the prognosis, while not so good for recovery, is usually not bad so far as the progress of the symptoms is concerned. The treatment is eventually psychotherapeutic with which may be combined various motor and reëducational exercises.

THE PSYCHONEUROSES. (HYSTERIA, COMPULSION NEUROSIS AND "SHELL SHOCK.")

Conversion Hysteria.—Historical.—To write the history of hysteria would mean practically to write the history of medicine, for hysteria stands throughout the ages as the type of functional disease which, protean in its manifestations, is found associated with all great therapeutic movements in medicine. Whether it be the therapeutics of religious conversion, of Perkin's tractors, or hypnotism, or more recently of persuasion, a considerable proportion of the patients who recover and thereby become largely responsible for the vogue of the particular therapeutic measure involved, belong to the great clinical group of hysteria.

Hysterical manifestations have been prominent in mental epidemics that have swept over whole continents, while the more specific and the more grotesque symptoms have always been observed and described.

The modern period in the history of hysteria might be said to have begun with Charcot. This period is still so recent as to be within the memory of many, and the influence which the Charcot school exerted is still all too dominant in certain quarters. The picture of hysteria as Charcot drew it, particularly of the *grande hysterie* with its regular march of histrionic attitudinizing, as set forth in the world-renowned pictures of Richer, is familiar.

For many years following Charcot the most brilliant work in elucidating the hysteria problem was done in France, and many illustrious names are crowded into a few years. All sorts of explanations were formulated, theories that were physiological, that were psychological, and that were biological, with numerous variants of each. The most illuminating worker in this field for many years, the one whose theories produced the greatest influence in the study of this disease was Pierre Janet, of Paris. His was a theory of dissociation, and he believed hysteria to be purely a mental malady. It was due to a poor synthesis of the personality which enabled certain groups of ideas to drop away from effective association with the main portion of the personality and occupy a region which Janet termed the subconscious, and there existing more or less independently, produce their results irrespective of corrections from the rest of the personality. The hysterical manifestations, then, were the manifestations of these split-off parts of the personality. Janet's views were a great advance upon the current concepts of hysteria, but although they rendered possible a deeper insight into the nature of the disease and the disease processes, they

were still largely descriptive, though, of course, the description was much refined from that of Charcot.

Various kinds of dissociation theories have been built up by investigators since Janet, and the dissociation theory was variously elaborated, particularly in this country by Sidis, White, Prince and others. It remained, however, for a Viennese physician, Sigmund Freud, to get beyond the point of description into a true interpretative attitude toward the disease.

Freud showed that the reason for the dissociation was that the dissociated ideas were out of harmony with the rest of the personality, that they represented ideas and feelings that were in conflict with the conscious tendencies of the individual, and that they were therefore repressed. Repression became with Freud, then, a fundamental factor at the basis of hysterical manifestations, an active, not a passive mental factor which tended to put out of mind certain inacceptable groups of ideas, and was therefore the cause of the dissociation. Added to this capacity for repression is an overemphasis of the ability to transfer the libido to objects, an increase in the capacity for object cathexsis as it is called. Hysteria is a transference neurosis.

The Mechanism of Conversion Hysteria.—Starting with dissociation as the most fundamental descriptive term applicable to the hysterical state —the doubling of the personality, in the sense of Janet—it has been shown that there is at the basis of this process of dissociation an active process called repression, which has as its function the splitting off of inacceptable idea-constellations—complexes—from the main body of the personality, and thus, so to speak, putting them out of mind. It has also been intimated that these split-off complexes because of being split off, do not therefore cease to act. As a matter of fact they go on functioning, but the functioning is independent, more or less, of the balance of the personality.

This process of repression and dissociation, following upon conflict, is a very general one and is found in divers mental states and is in fact a normal process. It is not these processes or mechanisms which are characteristic of any particular mental disorder, but it is the *way in which the split-off complexes manifest themselves* that produces the different types of mental disorders.

From what has been said it will be seen that if the individual, or more specially, the psyche, be considered as being a complex of adaptive mechanisms which is always making an effort to come into closer adaptation with the environment, then the meaning of a conflict is that there enter into this mechanism certain factors to which it cannot make efficient adaptation. This results in repression and splitting, but the whole tendency of the machine is to readjust effectively by bringing about in some way a new state of affairs. In the conflict there are two groups of tendencies in the psyche which are diametrically opposed one to the other. No solution of the conflict can possibly be brought about by a fulfilment of one of these groups, because manifestly the conflict would still remain. Therefore any readjustment

that takes place must in some way bring to pass the tendencies of both groups. Inasmuch as these groups are opposed to each other, such a result cannot actually be brought to pass in the world of reality at the level of the conflict. Therefore, unless an adequate adjustment can be brought about by an all-inclusive synthesis at a higher level an artificial world which is not governed by the strict laws of reality has to be brought into existence wherein these opposing forces can both, as it were, allegorically find their ends attained. This is well shown in the following dream: The patient said *she saw herself dead, lying in a coffin, with a red rose in her hand.* The red rose symbolized her sweetheart because of the frequent presents of red roses which he had made to her. Being dead in a coffin probably has several meanings, but among others has the meaning of a regression. The coffin is the matrix, it symbolizes a going back to the protection of the mother, and so the dream symbolizes the two opposing desires, one infantile, the desire for union with the mother, the other adult and recent, the desire for union with her sweetheart.

In the different mental disorders this end is brought about in different ways. The hysterical mechanism is different from the other mechanisms inasmuch as, while it is a general rule that the painful affect of the split-off complexes is drafted off by various channels and thus finds expression, and while it is a general rule that this expression is not consciously associated with the idea content of the complexes themselves so that the patient is saved from a realization of their true nature, is thus conserved from an appreciation of the pain that would result if they were understood at their true value, in hysteria the painful affect is drafted off into bodily innervation, thus producing the somatic phenomena of hysteria. This is the process of *conversion* and is characteristic of the familiar forms of hysteria, hysteria which is therefor called *conversion hysteria.* Freud believes this conversion to depend upon a special somatic predisposition. The so-to-speak strangulated, unreacted-to emotion of the split-off complexes manifests itself as the physical symptoms of the psychoneurosis and in this way the strong affect of the split-off complex is weakened. The complex is robbed of its affect, which is the real object of conversion and hence its value to the individual.

This is perhaps a somewhat involved statement but a simple example will make it clear. A patient ought to make a call upon a recently bereaved friend. This is recognized as a distinct obligation but the patient's infantile necessity of escaping reality and seeking pleasure makes the duty seem a very onerous affair. Thus arises a conflict between duty, born of conscious appreciation of the social obligation, and desire, born of the childish inability to make the necessary sacrifice of personal comfort. As a result the patient develops a headache, and so, being ill does not have to go. The selfish desire is thus gained and at the same time the social demands are satisfied by the illness which offers an acceptable excuse, while the painful recognition of the patient's own selfishness is converted into the pain in the head. The affect is

thus displaced, the whole situation symbolically distorted and the two opposing tendencies, conscious and unconscious both satisfied. Incidentally the pain in the head is a self-punishment for not obeying the socially useful and unselfish demand and has as a function the tendency to drive the individual along the path of development, for only by following this path can the pain be avoided. An adequate adjustment by a synthesis at a higher level would result in making the call, and wanting to make it, and deriving pleasure and satisfaction from having comforted the bereaved person.

This is such an example as everyone has constantly presented to him by all manner of persons placed in disagreeable circumstances. The tendency to develop some minor physical ill as an excuse and an escape from a recognized duty is used very widely, one is tempted to say, at times by almost everyone. It is the fundamental hysterical (conversion) mechanism which throws upon the body, makes it the scapegoat, the responsibility for our moral failures. And yet more than this. It produces suffering and pain, which here as elsewhere, point the way of relief by making the wrong path as unattractive as possible.

Symptoms.—The symptomatology of hysteria is naturally a very complex one, but from what has been said it will be seen that it tends to group itself more especially about disturbances of motion and of sensation. In addition to this it also tends to manifest itself in certain crises.

All forms of paralyses and anesthesias may manifest themselves. Paralyses of the limbs, either singly or hemiplegia with or without contracture, are common, while anesthesias may be distributed in almost any way, involving the superficies or the special senses. As a rule, of course, the distribution of these various phenomena do not follow the anatomical areas of nerve supply. They show a symbolic grouping.

The *disturbances of sensibility* are of many forms. Very characteristic are the glove and stocking anesthesias, involving the extremities of the limbs, hands, a lower part of forearm and feet, and lower portion of legs. Patches of anesthesia may be found upon any portion of the cutaneous surface and they may be widely distributed and often not constant in location but varying with different examinations. Hemianesthesia, especially of the left side of the body, crossed and alternating forms are found. Light touch is more often involved, frequently deep pain also, while insensitiveness to heat and cold also occurs.

A characteristic form of anesthesia which is very frequently found is concentric limitation of the field of vision.

These anesthesias do not follow anatomical areas and experiments will readily determine that they are psychological. This distribution, which is plainly not anatomical is as plainly psychological, that is, the anesthesia affects the hands, arms, etc., as they are thought of by the patient. If for example an anesthetic area is stimulated, the patient will say he feels nothing, but if asked to guess the nature of the stimulus, will show a surprising number of correct replies.

Hypoesthesias, hyperesthesias, various forms of neuralgia, especially visceral, and headache are also frequently hysterical.

The disturbances of motility are largely various forms of paralysis. Hemiplegia, monoplegia and paraplegia, with or without contractures, are the commoner forms. Astasia abasia is a characteristic hysterical condition.

Spasms of various parts are not infrequent. Spasms of the tongue, of the face and of the extremities when not demonstrably of organic origin are hysterical as a rule. Tremors, myasthenic types of reaction, and easy fatigability are frequent. Choreiform movements, tics, and certain occupation spasms are often hysterical.

The speech is involved frequently. Hysterical aphonia is well known and usually a diagnosis is warranted if the patient can only whisper replies to questions and an examination discloses healthy vocal cords. Stuttering is frequently hysterical and an analysis will show that the words with which there is difficulty have especial significance for the patient. Other respiratory disturbances of an asthmatic character may also be hysterical.

The distribution and characteristics of these motor disturbances are psychological, that is, disturbances develop, not as they would result from actual organic disease, but as the patient thinks they should result.

Visceral disturbances, especially of the gastro-intestinal tract, many of the false gastropathies with gastric crisis of vomiting and diarrhea, are quite frequent. There may also be hysterical attacks simulating renal or hepatic colic, gastric ulcer, etc.

Vasomotor disturbances, localized edemas, disturbed reflexes, fever, secretory and trophic disorders have all been described.

The visceral disturbances are also psychological in origin but in detail often dependent upon deeper disturbances of integration. Disgust, for example, gives rise to vomiting and the analysis will show, back of the symptom (vomiting) the feeling of disgust attached to some sort of experience in the individual's past (an actual experience or an experience only thought of) which is thought of as disgusting.

Symptoms which cannot be accounted for on anatomical and pathological grounds should always lead to an analytic examination of the psyche. Even marked disturbances may have originated in the psyche and continued so long as to produce organic changes, as for example, muscular atrophy from prolonged disuse of a limb, the paralysis of which was of psychogenic origin.

Among the *episodic phonomena* are found disturbances of emotion, either exaltation or depression, which can be understood because of the displacement of the affect. There are various types of delirium which may or may not be associated with convulsive seizures, producing, especially, when long drawn out, the so-called somnambulisms, during which all sorts of ideas may be manifested and the patient be quite disoriented. Dream states not infrequently occupy the field and lead by development to all sorts and degrees of double

personality, which is simply a more elaborate expression of the split-off complexes, indicating that they form a relatively large part of the personality. In fact, these split-off systems, provided recovery is not possible, tend to gather to themselves more and more of the personality and thereby to lead a more and more independent and broader existence.

Amnesias of course are frequent in the symptomatology. Any portion of the personality which is active may be amnesic for any portion of the personality which is opposed to it in the conflict.

In the analysis of hysterical symptoms it is relatively easy to account for them logically. While the symptoms, on the surface, often resemble dementia precox, one is not so often brought face to face with the crude outcropping of the unconscious. It is more frequently found that the symptoms lead directly back to actual situations, as in the case of[1] Lucy R., published by Breuer and Freud. This patient was disturbed by a subjective sensation of smell, which was traced back to a smell of burning pastry in a perfectly well-recollected scene where the children had forgotten the pastry and it had become burnt. Why the smell of burning pastry should be chosen for hysterical conversion was again traced to the young woman's love for the children for whom she was governess and the repressed wish that she might take the mother's place as the result of her love for her master. And in the case of Freud's of[2] Elizabeth, who, while engaged in nursing her sick father spends one evening away from home at the solicitation of her family. Upon this occasion she meets a young man and on her walk home with him gives herself up to the happiness of the situation. On the return, however, finding her father much worse, she bitterly reproaches herself for forgetting him in her own pleasure. This thought, however, is repressed. In the course of her caretaking she had each morning to change the dressings on her father's swollen leg. To do this she took his leg upon her right thigh. The suppressed complex seized upon the feeling of weight and pain of her father's leg upon her thigh as an efficient avenue of expression for her repressed wish which thus comes into consciousness under the disguise of a painful area on the right thigh corresponding in extent and location to the place upon which the father's leg rested.

From these examples it will be seen that the hysteric is the victim of the spontaneous and aberrant activity of repressed and split-off complexes that have to do with past events in the patient's life (actual or psychological) and that the expression of these complexes produces the symptoms of the psychoneurosis, and that so far as the hysterical manifestations are concerned the hysteric may be said to live in the past, for each access of symptoms is but a reanimation of past experiences. Past thoughts and feelings must not be put down as of no account, they are just as much facts as actual experiences, they are psychological facts.

[1] Freud, S.: Selected Papers on Hysteria and Other Psychoneuroses, Nervous and Mental Disease Monograph Series, No. 4.

[2] Loc. cit.

These split-off groups of ideas and feelings which tend to independent existence as secondary states which are reanimated in the hysterical somnambulisms are groups of ideas and feelings at relatively high levels. That is they hang together quite as do ideas and feelings in consciousness as we know it. They are logically related and tend to reproduce actual experiences or series of experiences. In other words they constitute minor personalities which maintain a sort of parasitic existence within the larger personality whole. The splitting in hysteria tends to be massive or molar in type rather than molecular as it does not tend to involve the partial tendencies to the extent of disintegrating the personality by involving those constituent elements which go to make up conduct as we ordinarily know it. The deeper splits in the personality which involve the more deep-seated factors and produce those grotesque symptoms which seem unpsychological are characteristic of those more grave disturbances which are found in the dementia precox group of psychoses.

Like all psychoneurotics the hysteric is infantile. In other words, there is a certain defect in psychosexual development, and the difficulties which they meet in life tend to drive them back upon themselves, to cause an introversion of the libido, that is, to remove their interest from the actual world of reality and to center it back again in themselves. As already explained, this introversion process tends to reanimate progressively lower psychosexual levels, and with a patient who is already infantile the tendency to reanimate, for example, the autoerotic level is easily manifest. This is well shown in certain symbolic masturbatory acts which recur during the hysterical seizures of which probably involuntary micturition is one.

The symptomatology of hysteria is then the symptomatology of the activity of the split-off dissociated idea constellations or complexes and their manifestation by the mechanism of conversion thereby producing symptoms of physical disorder.

These split-off complexes tend always to become dynamic and manifest themselves episodically in the hysterical seizures. The process of dissociation or splitting, once begun, tends to continue and new material tends constantly to be added to these split-off elements by further cleavage, and thus this new portion of the personality continues to grow at the expense of the total personality. Energy accumulates in these split-off systems, and when it becomes sufficient in amount it breaks through, so to speak, and produces the attacks. These attacks are made up characteristically of a living over again of those experiences which constituted the etiological moments of the psychoneurosis. In conversion hysteria, as has been pointed out, the breaking through of the energy from the split-off complexes manifests itself in bodily innervation—the symptoms of the disease are physical.

Aside from these episodic manifestations, there are the crises or paroxysms of the disease, the so-called *interparoxysmal symptoms*, which harking back to a middle age demonology are still termed *stigmata*. These are most characteristically various anesthesias, anesthesias

which are rarely complained of by the patient, often entirely unknown to him, being only brought out upon examination. It is because of this latter fact that Babinski has been led into the error of supposing that they were entirely the result of the examination, a position the erroneousness of which one can demonstrate to one's own satisfaction. Even though it were absolutely true, the fundamental fact, the why of the symptoms, the reason why certain patients react in such a way to an examination, remains unexplained by this renowned French neurologist.

An analysis of the stigmata shows also, and usually without much difficulty, a logical connection with preceding experiences, as for example, the smell in the case of Lucy R., or the anesthesia of the thigh in the case of Elizabeth, already cited.

There is another group of symptoms which follow of necessity as a result of the splitting of the personality. It can be easily seen from this dynamic conception of the nature of the disease that a person who is not at one with himself has not at any one time the full quota of his energies available, and therefore it is found that the general efficiency of this class of patients, particularly in the psychic sphere, is very greatly reduced; it is reduced in proportion to the amount of the personality which is represented by these split-off complexes. These patients, therefore, are not equal to the tasks they once could do. They are nervous and irritable, they tire easily, they lack capacity for consecutive application, and forget readily. These are general symptoms of the condition, secondary symptoms, the result of any splitting, and which are added to the primary symptoms which are expressions of the actual conflict.

A connecting link between the two portions of the personality is seen in the hysterical fantasies and also, of course, in the dreams which themselves belong to the realm of fantasy formation, fantasies which are thoughts that come without being bidden at moments of mental abstraction, thoughts that flow along without volitional choice, that replace one another without the exercise of critique, in other words, day-dreaming or night-dreaming as the case may be. These fantasies represent the activity of the submerged complexes as they break through and manifest themselves in the upper consciousness. They are of great importance in discovering the nature of the conflict and are very common features of the hysteric, although usually the patient does not realize it until his attention is addressed to these vagrant mental manifestations, because they not only come unbidden, but when they go they leave no tell-tale traces in the conscious memory. A further connection between the unconscious and conscious are the conscious fantasies. These are fantasy formations which apparently lie in clear consciousness and are not repressed. Fantasies of this sort are permitted in clear consciousness only because they are not understood at their true value. They really represent chiefly repressed material.

The peculiar characteristic symptom of hysteria, *conversion,* is of

great importance to understand. In paranoia the unconscious affects which are striving for expression are projected upon outside circumstances and persons—the mechanism of projection—(See paranoia). In hysteria the conversion mechanism is responsible for the indictment of the bodily organs. Errors of diagnosis are therefore favored and the patient seeks, so to speak, to delude the doctor to believe in some disease of an organ that can be treated. Such treatment the patient hopes will relieve the symptoms and the responsibiility is transferred in this way to the doctor the patient assuming no responsibility for them. Many complex chronic conversions cause physical disease.[1] The results may be tragic as in cases where important organs are removed on this hypothesis. There have undoubtedly been many ovaries removed in years past to cure a psychic conflict. This cutting out of the psychosis has, however, a very disastrous result for the patient who is thus projected still further along the path of chronic invalidism.

To resume conversion, hysteria is the result of a splitting of the personality in which certain split-off complexes are psychosexually determined; and leading an existence more or less independent of the total personality, express themselves by the mechanism of conversion in bodily innervation. The hysterical symptoms, then, become the representation through conversion of the unconscious fantasies which originate in the repressed complexes, while the structure of the hysterical attack is in every way similar to that of a dream. The attack is the breaking through of the energy of the repressed systems and manifests itself by a wish-fulfilling delirium, the elements of which may be overdetermined, displaced, and inverted for purposes of disguise, as are the elements of a dream.

Fixation Hysteria.—In fixation hysteria, while there is the same tendency to replace a mental by a physical manifestation, there seems to be an accentuation of the somatic predisposition with reference to a particular part of the body and that an external part as distinguished from the increased sensitiveness of the internal organs is hypochondria. Excellent examples of this fixation hysteria were seen during the war in those cases in which the localization of the hysterical symptoms was determined by a wound.

Anxiety Hysteria.—Anxiety hysteria, as the name indicates, occupies a midposition between conversion hysteria on the one hand and anxiety neurosis on the other. There is, so to speak, a combination of the two conditions, although this is not quite the situation. In conversion hysteria the affect of the repressed complexes is drafted into bodily innervation and produces the physical symptoms of the disease. In anxiety hysteria the affect remains in the mental sphere, producing there various phobias. In anxiety neurosis as already pointed out, the anxiety has its origin not at the psychical, but at the physiological level and is a representation in the psychic sphere of a

[1] See Jelliffe: Psoriasis as a Hysterical Conversion Product. Groddeck: Psychical Causation of Physical Disease, 1917.

disturbance in the somatic. In anxiety hysteria, the anxiety is also produced at the physiological level, but it is a secondary symptom and is the result of the physiological accompaniments of the emotions which go with the phobias, such as difficulty of breathing, cardiac palpitation, etc. The anxiety or the phobia at the conscious level represents a desire, a wish in the unconscious, but is attached not to the original but to a related object. A fear of having acquired syphilis, for example, is quite characteristically motived by a wish for potency. The old maid's fear of a man under the bed is almost obviously conditioned by a wish that there might be, while the fear of being assaulted and robbed is really a fear (desire) for sexual assault. Such a fear in a man, for instance, is quite typical of homosexual cravings.

Anxiety hysteria is one of the most widely distributed diseases. It is particularly the disease which manifests itself in childhood and from which most of the so-called nervous children suffer. It is much more easy of approach therapeutically than the compulsion neurosis, its accessibility being comparable to that of hysteria, and so offers greater opportunities for treatment. Probably many cases of this disorder are included under Janet's psychasthenia.

Compulsion Neurosis.—Compulsion neurosis contains probably the majority of that complex group to which Janet gave the name of psychasthenia. Janet's group, however, contained not only the compulsion neuroses, but a number of other things, particularly the anxiety neuroses, probably many anxiety hysterias, perhaps some neurasthenias, conversion hysterias and even schizophrenias.

The characteristics of the compulsion neurosis are certain compulsive tendencies to act or to think in certain ways. The patient is forced, apparently against his will and without reason, to think certain ideas or certain thoughts or to do certain things. The compulsion increases until it is yielded to, then a period of calm follows which may be of variable length, until, so to speak, the energy has again accumulated, when the compulsion again manifests itself and must again be relieved by yielding. The patient has perfect insight into the matter, knows the whole business is foolish, but he cannot help it.

The *symptoms* group themselves about the motor, sensory, ideational and affective aspects of the psyche. So we find compulsive acts, compulsive sensations and hullucinations or pseudo-hallucinations, compulsive ideas, and compulsive feelings, such as fears and doubts.

Mechanism of Compulsion Neurosis.—In hysteria the repressed material manifests itself by *conversion*. The accumulated affect of the split-off complexes is drained off through bodily innervation. The physical symptom is made the scapegoat to save the more important mental adjustment. The highly affect-laden complexes are thus deprived of their emotion. The hysterical attack is a wish-fulfilling delirium which brings to pass in a sort of allegorical dramatization the fulfilment of both elements in the conflict.

In contradistinction to these characteristics of hysteria, in the compulsion neurosis there is no conversion. The affect of the repressed

complexes is drained off, not through bodily innervation, but by attachment to otherwise indifferent ideas. The affect is displaced to a substitute. This substitution, quite as in the conversion of hysteria, is a distortion mechanism and serves equally with it to disguise from the patient the real source of the affect.

Then again, while in the hysterical attack both elements in the conflict come to contemporaneous fulfilment, such unification through the symptoms is less evident in the compulsion neurosis, although the attempt is made to bring it about. What occurs on the surface, at least, is a constant alternation between the ascendency of the two factors in the conflict, which two factors in their ultimate analysis resolve themselves, perhaps always, into love and hate. The symptoms of the neurosis are due to the constant conflict between the unconscious wish and the repressing forces which try to keep it out of consciousness but do not quite succeed, as it gains expression under symbolic disguise. This constant back and forth play between tendencies which are of opposite sign (ambivalent opposites) and in which neither one is able to maintain ascendency, is the cause for the frequency of feelings of doubt and indecision. As will be seen from the following quotation Freud believes these symptoms represent the return in disguised form, of self-reproaches dating back to childhood. The period in childhood from which these symptoms date is that period in the sexual life of the child before the primacy of the genitals is established—the *pregenital* period—and to the portion of that period when as yet the distinctions of masculine and feminine play no part, but their forerunners, activity and passivity, are beginning to come into play. It is previous to the organization of sexual polarity. In this period it is the partial tendencies, sadistic and anal, that are prominent and he therefore calls it the sadistic-anal phase of libido development.[1]

The compulsion neurosis is a true defense neurosis and its symptoms, at least the compulsive acts, which develop late in the course of the disorder are of the nature of ceremonials, which not only serve to disguise the true situation from the patient, but so to speak, atone for evil. The symptoms therefore not only permit the forbidden to come to pass under adequate disguise but also punish the patient by the sufferings of his neurosis for yielding to his lesser self, for offending his ego-ideal.

Freud has very well said that it is much more correct to speak of obsessive thinking than of obsessive ideas. It is the obsessive element, the compulsion, the so-called Zwang of the Germans which is the essential thing in this neurosis, and which may express itself in all possible ways.

The mechanism of the production of the symptoms of the compulsion neurosis is a quite complicated one. It cannot be expressed better than by quoting the language of Freud.[2]

[1] Freud, S.: A General Introduction to Psychoanalysis, Boni and Liveright, New York, 1920.

[2] Hitschmann: Freud's Theories of the Neuroses, Nervous and Mental Disease Monograph Series, New York.

The extracts from Freud, which are taken from his original formulation of the compulsion neurosis concept, are somewhat involved and difficult to understand so the plan will be followed of interspersing them with comments, explanations, and illustrations for the purpose of simplification and classification.

"Sexual experiences of early childhood have the same significance in the etiology of the compulsion neurosis as in hysteria, still we no longer deal here with sexual passivity but with pleasurably accomplished aggressions, and with pleasurably experienced participation in sexual acts, that is, we deal here with sexual activity. It is due to this difference in the etiological relations that the masculine sex seems to be preferred in the compulsion neurosis.

"The compulsion neurosis is developed in its full-blown form as a type of expiatory ceremonial for acts guiltily participated in, *i. e.*, acts in which the patient took an active part if only by acquiescence and for which he therefore holds himself responsible.

"In all my cases of compulsion neurosis I have found, besides, a subsoil of hysterical symptoms which could be traced to a pleasurable action of sexual passivity from a precedent scene. I presume that this coincidence is a lawful one and that premature sexual aggression always presupposes an experience of seduction. But I am unable to present as yet a complete description of the etiology of the compulsion neurosis. I only believe that the final determination as to whether a hysteria or compulsion neurosis should originate on the basis of infantile traumas depends on the temporal relation of the development of the libido.

"The essence of the compulsion neurosis may be expressed in the following simple formula: Obsessions are always transformed *reproaches* returning from the repression, which always refer to a pleasurably accomplished sexual action of childhood. In order to elucidate this sentence it will be necessary to describe the typical course of compulsion neurosis.

"The compulsive ways of thinking and acting are only substitutes for the reproaches which are symbolical distortions in order to prevent a recognition on the part of the patient of the real meaning, in order to keep from his consciousness a realization of the actual circumstances of his guilty conduct.

"In a first period—period of childish immorality—the events containing the seeds of the later neurosis take place. In the earliest childhood there appear at first the experiences of sexual seduction which later makes the repression possible, and this is followed by the actions of sexual aggressions against the other sex which later manifest themselves as actions of reproach."

The original experience here referred to as one of "sexual seduction" must be understood in a much broader sense than is usually given these terms. The term sexual, for example, refers to the whole realm of the race-preservative libido as previously outlined. The listening to "nasty" stories might therefore be such an original form of "sexual

seduction" which easily has its wrongfulness emphasized by the child's failing to tell the mother all the things the boys at school talked to him about, although warned by her to do so for the very obvious reason of preventing this very form of auto-erotic indulgence. Later on the very acts of exchanging confidences and having secrets of a sexual nature with other boys becomes a matter for future reproaches. This is, of course, but one of innumerable types of experience which may bring about similar mechanisms of defense.

"This period is brought to an end by the appearance of the—often self-ripened—sexual 'maturity.' A reproach then attaches itself to the memory of that pleasurable action, and the connection with the initial experience of passivity makes it possible—often only after conscious and recollected effort—to repress it and replace it by a primary symptom of defense. The third period, that of apparent healthiness but really of successful defense, begins with the symptoms of scrupulousness, shame and diffidence.

"The next period, the disease is characterized by the return of the repressed reminiscences, hence by the failure of the defense; but it remains undecided whether the awakening of the same is more frequently accidental and spontaneous, or whether it appears in consequence of actual sexual disturbances, that is, as additional influences of the same. But the revived reminiscences and the reproaches formed from them never enter into consciousness unchanged, but what become conscious as an obsession and obsessive affect and displace the pathogenic memory in the conscious life are compromise formations between the repressed and the repressing ideas."

An example will make this clear. A young man, when a very young boy, was subjected to a homosexual assault. The affair occured in a portion of a room the floor of which was covered with a white bear-skin rug. Following this episode shame and diffidence became marked character traits. Later on he indulged in the habit of masturbating and for these occasions would select a dark room. While engaged in this forbidden practice he would fear discovery and imagine he could see the bright eyes of his accusers looking at him. The whites of the eyes were their worst feature. Here we find the later return of the repressed reminiscences symbolically expressed by the fear of white in the fancied eyes of the discoverer of his habit. Still later this fear became generalized into a dislike and fear of white objects in general, especially the whites of eggs. This phobia is therefore not only a symbolic expression of the repressed reminiscences but a defense against the pain of their recognition. It is also a mechanism for turning the individual from his auto-erotic practices upon the healthy path of psychosexual development.

"In order to describe clearly and probably convincingly the processes of repression, the return of the repression, and the formation of the pathological ideas of compromise, we would have to decide upon very definite hypotheses concerning the substratum of the psychic occurrence and consciousness. As long as we wish to avoid it we will

have to rest content with the following rather figuratively understood observations. Depending on whether the memory content of the reproachful action alone forces an entrance into consciousness or whether it takes with it the accompanying reproachful affect, we have two forms of compulsion neurosis. The first represents the typical obsessions, the content of which attracts the patient's attention; only an indefinite displeasure is perceived as an affect, whereas for the content of the obsession the only suitable affect would be one of reproach. The content of the obsession is doubly distorted when compared to the content of the infantile compulsive act. First, something actual replaces the past experience, and second, the sexual is superseded by an analogous non-sexual experience. These two changes are the results of the constant tendency to the repression still in force which we will attribute to the 'ego.' The influence of the revived pathogenic memory is shown by the fact that the content of the obsession is still partially identical with the repressed or can be traced to it by a correct stream of thought. If, with the help of the psychoanalytic method, we reconstruct the origin of one individual obsession we find that one actual impression instigated two diverse streams of thought, and that the one which passed over the repressed memory, though incapable of consciousness and correction, proves to be just as correctly formed logically as the other. If the results of the two psychic operations disagree, the contradiction between the two may never be brought to logical adjustment, but as a compromise between the resistance and the pathological result of thought an apparently absurd obsession enters into consciousness beside the normal result of the thought. If both streams of thought yield the same result, they reinforce each other so that the normally gained result of thought now behaves psychically like an obsession. Wherever neurotic compulsion manifests itself psychically it originates from repression. The obsessions have, as it were, a psychical course of compulsion which is due, not to their own validity, but to the source from which they originate, or to the source which furnishes a part of their validity.

"A second form of compulsion neurosis results if the repressed reproach and not the repressed content of memory forces a replacement in the conscious psychic life. Through a psychic admixture, the affect of the reproach can change itself into any other affect of displeasure, and if this occurs there is nothing to hinder the substituting affect from becoming conscious. Thus the reproach (of having performed in childhood some sexual actions) may be easily transformed into shame (if someone else becomes aware of it), into hypochondriacal anxiety (because of the physically harmful consequences of those reproachful acts), into social anxiety (fearing punishment from others), into religious anxiety, into delusions of observation (fear of betraying those actions to others), into fear of temptations (justified distrust in one's own moral ability of resistance), etc. Besides, the memory content of the reproachful action may also be represented in conscious-

ness, or it may be altogether concealed, which makes the diagnosis very difficult. Many cases which on superficial examination are taken as ordinary (neurasthenic) hypochondria often belong to this group of compulsive affects; the very frequently so-called 'periodic neurasthenia' or 'periodic melancholia' especially seem to be explained by compulsive affects or obsessions, a recognition not unimportant therapeutically.

"Besides these compromise symptoms which signify the return of the repression and hence a failure of the originally achieved defense, the compulsion neurosis forms a series of other symptoms of a totally different origin. The ego really tries to defend itself against those descendants of the initial repressed reminiscence, and in this conflict of defense it produces symptoms which may be comprehended as 'secondary defense.' These are throughout 'protective measures' which have performed good service in the struggle carried on against the obsessions and the obsessing affects. If these helps in the conflict of the defense really succeed in repressing anew the symptoms of return obtruding themselves on the ego, the compulsion then transmits itself on the protective measures themselves and produces a third form of the 'compulsion neurosis,' the compulsive action. These are never primary, they never contain anything else but a defense, never an aggression. Psychic analysis shows that despite their peculiarity they can always be fully explained by reduction to the compulsive reminiscence which they oppose.

"One example instead of many: An eleven-year-old boy has obsessively arranged for himself the following ceremonial before going to bed: He could not fall asleep unless he related to his mother most minutely all experiences of the day; not the smallest scrap of paper or any other rubbish was allowed in the evening on the carpet of his bedroom. The bed had to be moved close to the wall, three chairs had to stand in front of it, and the pillows had to lie in just such a particular position. In order to fall asleep he had to kick with both legs a number of times, and then had to lie on the side. This was explained as follows: Years before, while putting this pretty boy to sleep, the servant girl made use of this opportunity to lie over him and assault him sexually. When this reminiscence was later awakened by a recent experience it made itself known to consciousness by the compulsion in the above-mentioned ceremonial which sense could really be surmised and the details verified by psychoanalysis. The chairs before the bed which was close to the wall—so that no one could have access to it; the arrangement of the pillows in a definite manner—so that they should be differently arranged than they were on that evening· the motion with the legs—to kick away the person lying on him; sleeping on the side—because during that scene he lay on his back; the detailed confession to his mother—because in consequence of the prohibition of his seductress he concealed from his mother this and other sexual experiences; finally, keeping the floor of his bedroom clean—because this was the main reproach which he had to hear from his mother up to that time.

"The secondary defense of the obsessions can be brought about by a forcible deviation to other thoughts of possibly contrary content; hence in case of success there is a compulsive reasoning regularly, concerning abstract and transcendental subjects, because the repressed ideas always occupied themselves with the sensuous. Or the patient tries to become master of every compulsive idea through logical labor and by appealing to his conscious memory; this leads to compulsive thinking and examination to doubting mania. The priority of the perception before the memory in these examinations at first induce and then force the patient to collect and preserve all objects with which he comes in contact. The secondary defense against the compulsive affects results in a greater number of defensive measures which are capable of being transformed into compulsive actions. These can be grouped according to their tendency. We may have measures of penitence (irksome ceremonial and observation of numbers), of prevention (diverse phobias, superstition, pedantry, aggravation of the primary symptom of scrupulousness), measures of fear of betrayal (collecting papers and shyness), and measures of becoming unconscious (dipsomania). Among these compulsive acts and impulses the phobias play the greatest part as limitations of the patient's existence."

That a line of secondary defenses, so to speak, becomes necessary means that the original, the primary defenses, were not sufficient. These primary defenses were broken down and a more vigorous effort has had to be made by the psyche to protect itself. The psyche, in other words, adopts mechanisms, which, so far as possible, keep the libido from resorting to infantile ways of pleasure seeking and force it along the path of social usefulness.

The phobias keep spreading out as in the case cited in which the fear of white originally symbolically attached to the whites of the eyes became a fear of all things white, and tend to more and more limit the patient's activities by closing an ever-increasing number of paths of expression.

The ceremonials in the form of peculiar successions of movements, as in the case of the boy cited above by Freud, the various tics, the saying over of formulæ, of prayers, the special arrangements of furniture, of clothes, the ceremonials of the bath room and of eating, dressing, etc., are very numerous and infinitely varied. Among these the various cleansing ceremonials are common. One patient, because her thoughts were unclean was in constant fear that she would offend God by allowing some particle of secretion from her body to come between her and Him. Tears for instance might have been due to unclean thoughts and so the greatest care in washing had to be exercised before she spoke God's name or prayed. This necessity extended to all the secretions, and so a great deal of time was occupied in most detailed and painstaking processes of washing and avoiding all forms of pollution.

"There are cases in which we can observe how the compulsion

becomes transferred from the idea or affect to the measure, and other cases in which the compulsion oscillates between the returning symptoms of secondary defense. But there are also cases in which no obsessions are really formed, but the repressed reminiscence immediately becomes replaced by the apparent primary defensive measure. Here that stage is attained at a bound which otherwise ends the course of the compulsion neurosis only after the conflict of the defense. Grave cases of this affection end either with a fixation of ceremonial actions, general doubting mania, or in an existence of eccentricity conditioned by phobias.

"That the obsessions and everything derived from them are not believed is probably due to the fact that the defense symptom of scrupulousness was formed during the first repression and gained compulsive validity. The certainty of having lived morally throughout the whole period of the successful defense makes it impossible to give credence to the reproach which the obsession really involves. Only transitorily during the appearance of a new obsession, and now and then in melancholic exhaustive states of the ego do the morbid symptoms of the return also enforce the belief. The 'compulsion' of the psychic formations here described has in general nothing to do with the recognition through belief, and is not to be mistaken for that moment which is designated as 'strength' or 'intensity' of an idea. Its main characteristic lies in its inexplicableness through psychic activities of conscious ability, and this character undergoes no change whether the idea to which the compulsion is attached is stronger or weaker, more or less intensively 'elucidated,' 'supplied with energy,' etc.

"The reason for the unassailableness of the obsession or its derivative is due only to its connection with the repressed memory of early childhood, for as soon as we succeed in making it conscious, for which the psychotherapeutic methods already seem quite sufficient, the compulsion, too, becomes detached."

The mechanism of the compulsion neurosis is therefore seen to be an extremely complicated one and one which produces a great variety of symptoms, with all possible ramifications of meaning.

This mechanism, as set forth in this rather intricate statement by Freud, may be more simply stated by saying that, in distinction from hysteria, in which the disguise is brought about by a transfer of the repressed material into symbols of bodily ailment—conversion—in the compulsion neurosis the disfigurement is kept wholly within recognized psychological territory. The distortion is produced by displacement of the affect upon indifferent ideas (substitution) and the development of a purificatory ceremonial. Fear of animals (snakes, mice, etc.), may be the accepted conscious equivalent of fear of sexuality added to which is a type of ceremonial, and is well illustrated in the case of the eleven-year-old boy cited.

The extracts thus far cited from Freud were from his entire paper (1894–5–6). A more recent paper (1909)[1] carries the subject somewhat

[1] Abstracted in the Psychoanalytic Review, January, 1916, vol. 3, No. 1.

further. In this paper he discusses further the distortion by which the affect is displaced from the matter of real moment to a substitute of little importance and how through this mechanism the ceremonials finally grow up.

As a result of his further studies Freud believes that the fundamental elements that are opposed to one another in the compulsion neurosis are love and hate. This fundamental conflict arises very easily and is conditioned by the necessity on the part of the child. Thus the child's love is frequently replaced by hate for the parent who interferes with him and prohibits him from exercising some pleasurable activity. The constant alternations between love and hate produce the indecision which these patients so constantly show. They are in constant doubt as to the course of action they should pursue, their real doubt being their doubt of their power to love. Love and hate exist side by side, love never having fully succeeded in dominating but only in repressing the hate into the unconscious. This same doubt makes matters lead to uncertainty in the carrying out of the various protective and defensive measures and necessitates the endless repetition, a typical characteristic of the ceremonials.

The compulsion neurotics are, as a rule, superior persons who are striving with tremendous energy to attain to higher things. The mechanisms they use are used more or less by all, but for reasons which are as yet not fully worked out, lead, in some instances, to this peculiar form of illness. Freud suggests, provisionally, that there is a connection between the unconscious hate and the sadistic component of the sexual instinct which was exceptionally developed and was prematurely and too profoundly repressed. Very briefly then, what has happened to these patients is that some infantile way of gaining pleasure has received too much emphasis in the course of their psycho-sexual development so that, in this region they remain fixed to those ways of gaining pleasure which have proved satisfactory and are thus unable to proceed upon the path of development and leave them behind. The conscious ideals of the patient are such that actions conditioned by such desires cannot even be admitted to consciousness and so the neurosis arises as the expression of this conflict and, too, an expression of the essentially moral qualities of the patient, that is, those qualities which insist upon continuing the fight for further advance on the road of development. To be more specific: this course of events can be ascribed to a precocious ego development and the formation of the super-ego (ego-ideal) at the anal-sadistic stage, that is, the child worked out its Oedipus problems at the anal-sadistic stage of development instead of at the later phallic stage.[1] Everyone passes along the same road in development. It is when some phase which should be brief is overemphasized and prolonged that the basis of such a conflict is laid.

Symptoms.—The symptoms of the compulsion neurosis are very varied. They have to do with all types of obsessional thinking and

[1] Rickman, John: A Survey. The Development of the Psycho-analytical Theory of the Psychoses, British Jour. Med. Psy., 1926, vol. **6**, Pt. 4, 1927, vol. **7**, Pts. 1 and 2.

acting, that is, thinking and acting which takes place aside from the volition of the patient, which he cannot prevent but which he must yield to, as already described. This is the compulsion element which gives the name to the neurosis.

The symptoms have been variously divided and may be described under the form of motor symptoms, obsessive acts of various sorts; tics, spasmodic torticollis, even epileptic attacks; sensory symptoms, bsessive sensations, amounting at times to well-marked hallucinations; affective symptoms, obsessive emotions, more particularly those of doubt and fear; and ideational symptoms, obsessive ideas, such as continual questioning.

The commonest and best known of the obsessions are the *phobias* or fears which usually refer to some very specific object or set of conditions which acquire their quality of fear as the result of taking over an affect by displacement which is of deep though unconscious significance to the patient. Thus there are *misophobia* (fear of dirt or contamination), *metallophobia* (fear of metal, door-knobs, money, etc.), *agoraphobia* (fear of wide or open spaces), *claustrophobia* (fear of narrow or closed spaces), *pyrophobia* (fear of fire) and so on indefinitely.

The obsessions of *doubt—folie de doute*—are common and result in a state of mind in which the patient is torn between two courses of conduct and cannot choose, or having done something, such as turn out the gas before going to bed, is seized with doubt as to whether he really did do it or not and must get up and satisfy himself. Then doubts when they refer to religious or philosophical matters lead to continuing questionings and elaborate processes of reasoning from which the patient cannot free his mind.

Quite allied to the phobias and doubts are certain moral obsessions such as overconscientiousness and exaggerated scrupulosity.

Of the various obsessional activities the so-called *manias* are best known. Thus there are *kleptomania* (a compulsion to steal), *pyromania* (a compulsion to set something on fire), *dipsomania* (a compulsion to drink), etc.

In addition there are all sorts of less easily classified and more complex forms of obsessional ways of thinking, feeling and acting. There are complicated ceremonials, such as that of the eleven-year-old boy already cited, ways of arranging things that must be carried out. Strange, and to the patient inexplicable attractions and repulsions, dislikes for certain kinds of food, or persons with a particular color of hair, all manner of tics, habits, mannerisms, ceremonials, the necessity for touching things—*delire de toucher*—fixed ideas, hypochondrias, etc.

Such psychological phenomena must, of course, have a reason for their existence, and as the reason is not apparent it cannot be explained by the patient; it must be sought by psychoanalysis in the unconscious.

These obsessions produce a tremendous amount of mental unrest and suffering if they are not yielded to, and a sense of relief is expe-

rienced when they are yielded to, oftentimes, however, with a following sense of remorse for having yielded.

The compulsive ideas and acts represent compromise formations which permit the patient to obtain satisfactions in infantile ways, *i. e.*, to revert to old ways of gaining pleasure which were active and important in infancy when the erogenous zones were as yet not clearly differentiated. Here are found the explanation for urinary and fecal fantasies, for certain cutaneous, anal, and gastro-intestinal satisfactions which are used as ways of getting pleasure when driven away from reality. They become infantile ways of reacting to reality situations and so are inefficient, sick ways.

"Shell Shock."[1]—"Shell shock" is a popular term which has been applied to a multitude of conditions during the great war. While it is true that the explosion of large caliber shells may produce actual physical injury by setting masses of air and gas in violent motion or by creating a vacuum and so produce symptoms of concussion or of the nature of caisson disease, still the vast majority of the cases included in this group belong to the functional neuroses and psychoneuroses with, of course, a certain admixture of borderland conditions and mild psychoses. The largest number by far of the true functional "shell shock" cases are cases of true conversion hysteria or anxiety hysteria. The mechanisms of "shell shock" are therefore mechanisms with which we are already familiar. The term is an exceedingly unfortunate one, as it assists in deflecting the vision of both the patient and the public from the true state of affairs and thus of necessity makes it difficult to apply an effective therapy. "Shell shock" is really a term for both patient and relatives to hide behind. Not infrequently there is associated with the condition of "shell shock" an organic disturbance which may be masked by the functional symptoms and so overlooked.

MIXED NEUROSES.

Neurasthenia, anxiety neurosis, hypochondria, tic, hysteria, the compulsion neurosis, have been described. If the etiology and mechanisms of these six conditions be considered it will be seen that they do not of necessity mutually exclude one another and as a matter of fact not infrequently certain admixtures are found in clinical experience. Anxiety hysteria, for example, has taken a rather definite place among these conditions, while as will be readily seen from the nature of the etiological moments neurasthenia and anxiety neurosis are not infrequently found associated in various proportions, while, of course, it follows that the etiological moments of the actual neuroses are not excluded from operating in the same patient who may have a psychoneurosis. Such combinations are therefore of not uncommon occurrence.

Aside from the possibility of the mixture of the neuroses it should

[1] Southard, E. E.: Shell-shock and other Neuropsychiatric Problems, Boston, W. M. Leonard, 1919.

also not be lost sight of that the etiological moments of the neuroses may also operate in persons who are suffering from the more severe psychoses, as for example, manic-depressive psychosis and dementia precox. We quite commonly find hysterical symptoms in the precox, while neurasthenic and anxiety states are not infrequent in the depressions of manic-depressive psychosis. Other combinations, of course, might be mentioned. The important thing to bear in mind, however, is the nature of the etiological moment and the mechanism of the condition, and then these will be recognized when the individual patient is under analytic observation.

Finally, the purely psychic element is found more and more in association with the actual neuroses. This was intimated in the description of the etiology and mechanisms of neurasthenia and it has also been spoken of in the description of the anxiety neurosis. More will be said on this point under the head of Treatment.

TREATMENT OF THE NEUROSES AND PSYCHONEUROSES.

In the treatment of the actual neuroses the main thing, as indicated by the description which has been given, is to correct the sexual life of the patient. In dealing with neurasthenia the habit of masturbation (using this term in its broader significance, including the auto-erotic phantasies), if it be present, must, of course, be dealt with before anything definite can be accomplished, while, of course, with both neurasthenia and anxiety neurosis, matters of coitus interruptus, ejaculatio precox, abstinence, etc., must be carefully inquired into and the sexual life modified as indicated so that the evil effects which result from them may be remedied. These are the simple thing to do, and in many cases will produce marked betterment, if not recovery.

Of course in dealing with these conditions it is not meant simply that certain concrete physical ways of indulgence should simply be stopped. It is essential that the whole scheme of living should be raised to a higher plane based upon an understanding by the patient and his orientation toward healthier ideals.

Rest cures, hydrotherapy, massage, electricity, exercise, and all such therapeutic agents have their place in the treatment of the neuroses, particularly the actual neuroses, but their place is a secondary one. It has already been indicated that the precipitating factor in the outbreak of a neurosis may, for example, be overwork, but that the neurosis would not eventuate in the absence of the specific etiology. The explanation of this occurrence was that the patient was strong enough to stand up under the results of sexual traumata until his general resistance was reduced by overwork and then the neurosis appeared. It will be seen, therefore, that the usual methods of treatment very frequently bring about a cure, but not in the way in which they are supposed to. By changing the patient's sexual habits, removing him from his surroundings, sending him away to a sanitarium, oftentimes the sexual situation is more or less well solved

temporarily. Now, if during this period of residence in a sanitarium he is on careful diet, given regular exercise with baths and massage, it is perfectly understandable that his general resistance will be increased so that he may overcome the effects of the sexual traumata. In this way he may get well without any direct attack upon the factors of the specific etiology. This is the principle which is oftentimes seen in operation in the improvement and alleged recoveries of neurotics as a result of the rest cure. The old symptoms may return in full vigor following a rest-cure regimen as soon as the patient undertakes to resume his usual way of living.

Inasmuch, however, as even the actual neuroses may present some admixture of purely mental symptoms, either primary or secondary in origin, it may be necessary ultimately and before satisfactory results can be obtained to deal with these mental symptoms. If so, they must be dealt with in precisely the same way as the mental symptoms of the psychoneuroses, namely, by psychoanalysis.

The main principle involved in psychoanalysis may be said to be an analysis of the patient's mental condition sufficiently complete to thoroughly understand, to reconstruct the symptomatic manifestations of his malady. Before psychoanalytic methods were employed usually no explanation was sought for mental symptoms and apparently it rarely entered anyone's mind that they had any. The patient who had a phobia or a tic was simply looked upon as being nervous, perhaps having had some fright or bad impression earlier in life, and was usually treated by tonics or rest or travel or some other such means that was not addressed to the solution of the problem in any way. From what has already been said about the psychic development of the individual it will be readily appreciated that no mental fact can fail to have a thoroughly logical and understandable reason for its existence, and it is one of the objects of psychoanalysis to find this out.

Briefly, *the technic of psychoanalysis* is about as follows, being of course, modified in detail to some extent by the exigencies of the occasion and as the result of the special predilections of the physician practising it. When the patient cal s upon the physician the physician should let the patient, as far as possible, without interrupting him by questions or otherwise, detail to him his difficulties. This may take only a few minutes, or may be quite a lengthy recital, but it is usually worth while to listen carefully to the whole thing, perhaps occasionally by a suggestion, keeping the patient to the point if he tends to be too circumstantial. This original statement contains a description of the things from which the patient is suffering, and if careful attention is paid to it one may get many hints as to how to pursue the further inquiry. As a result of this conversation and perhaps another, if it is necessary, the physician makes up his mind whether the patient's illness is a suitable one for psychoanalytic treatment, and if it is he so states and then makes arrangements for regular consultations, preferably not less than three times per week, of an hour's duration each, and all other preliminaries are arranged too. Should it appear that the

patient has any symptoms of physical illness he should be sent to a competent physician skilled in the particular trouble that appears to be present, for the psychoanalyst should under no circumstances undertake to treat the physical condition. The reasons for this will appear later. It is preferable, too, that the physical condition be attended to fully, if possible, before the psychoanalytic treatment be taken up. In other words, it is undesirable to do a psychoanalysis while the patient is under the care of another physician.[1]

Having arranged all the preliminaries, the patient calls at the appointed hour and the psychoanalytic conversation proceeds about as follows: Bearing in mind the ultimate goal, the psychological explanation of the patient's symptoms, the patient may be approached by beginning the discussion of one of the symptoms, either simply asking about it or else pursuing some line of inquiry that was suggested in the original conversation. On the other hand, the method may be pursued of endeavoring to first get a clear understanding of the whole life of the patient, beginning from the earliest recollections and tracing the development to the present. It really makes very little difference how one starts, because in a very short time there will be all manner of suggestions to develop inquiries along various lines, and these will have to be followed out for an untangling of the situation.

During the course of the psychoanalytic conversations one will get very shortly to a point from which progress seems to be impossible, for it does not take long to exhaust the conscious material of the patient. One then has to penetrate the foreconscious, which is relatively easy, and the unconscious, which is relatively difficult. The method of procedure here is the method of free association. Perhaps a point has been attained in the conversation, a situation has been unfolded, which has no apparent explanation. The patient cannot give any reasons which adequately account for it. Under these circumstances the patient is asked to, so to speak, take the situation as a starting-point, and then relaxing into a condition of perfect passivity observe the thoughts that come to his mind and recount them as fast as they appear. In other words, he is asked to place himself in a mental state favorable to fantasy formation, he is asked to relinquish his grasp upon his mental life, to permit his ideas to flow untrammelled and unselected by his volition and critique. He is asked, as it were, to become the observer of his own ideas, to sit as if he were sitting in the window of a moving train, recounting aloud the objects as they passed by. So he is asked to observe his ideas and to tell them as they come. This sounds like rather a simple procedure, but it is a very difficult one for the patient to learn, and in fact when the patient can do it and do it easily he is approaching the end of his treatment.

Experience shows that when patients are instructed in this way they take the situation as directed as a starting-point, and with the intention of telling the ideas that come to their mind they will relax

[1] Jelliffe: Technique of Psychoanalysis, Nervous and Mental Disease Monograph Series, No. 26, 1919, 2d Edit.

into a condition of passivity. Perhaps then for a considerable time they do not speak, and if they are asked why they do not tell what comes to their mind they will say that nothing comes. This is, of course, the interference of the repressed complexes; it is the resistance which they have to coming into consciousness which is manifesting itself in this way. The whole thing, then, has to be gone over again with the patient. It has to be explained to them that their mind cannot be an absolute blank, and they have to be warned especially not to exercise choice as to what ideas they shall tell and what they shall not tell, that it makes no difference how absurd or inconsequential the idea is that comes to their mind they must tell it; even if the idea is extremely disagreeable they must tell it, for no matter how little connection it may appear to have with their trouble, if the starting-point has been from some problem in the case these ideas that come must have some connection with that problem. It must be explained to them that, of course, they cannot see the connection, but that they must tell the ideas so that the physician may have them and that he will be able to see what bearing they have in the situation. Of course he may not be able to see at once, but it is so much material which, if it does not come in for utilization today, can be used perhaps tomorrow, or the day after.

This is the process of *free association*, one which is very difficult for the patient to learn and one which requires much skill and no little art on the part of the physician. The physician must be ever on the alert. It requires the most intense application to the question in hand, for everything must be watched with the utmost care. Every little detail must be observed as containing perhaps a hidden meaning behind it. The hesitations, the stammerings, the mistakes, the slips of the tongue, all have their significance. For example, in talking to a young man, who told about his previous illness, he said that the physician had prescribed four quarter-grain tablets of "quinin" for him. He had no sooner mentioned the name of the drug than he immediately corrected himself and said "calomel." An inquiry into the meaning of quinin to him brought out a most important event in his life, an event undoubtedly of significance in his neurosis. The repressed complex was struggling as it always is, for expression. A favorable opportunity presented itself. Calomel and quinin are easily mixed in the pronunciation, and the repression slipped its moorings for a moment and found expression. It remained for the observer to be sufficiently keen to see the possibility of meaning in such a mistake and find out that meaning.

The most important single aid in determining the content of the unconscious is the dream. The split-off complexes are, in accordance with the theory set forth, actively repressed by the individual, they are not permitted to come to expression if he can help it. They therefore can only express themselves in symbolic form, in which form they are disguised not only to others, but, of more importance, to the patient himself. In this disguised form they are not recognized for

what they are, and therefore the painfulness of their recall to consciousness is avoided. A previous moral delinquency may thus come to the surface under a complete disguise without causing any particular distress, whereas it would be quite unbearable if it came forth in its true colors. It can be seen from this why it is that obstacles are so quickly reached in the psychoanalytic procedure. The dream is the best and most useful avenue for overcoming this type of obstacle. Here the repressed complexes come upon the stage in all their paraphernalia of symbolic disguise and find an opportunity for expression. The patient, not understanding what the dream means, will pretty generally recount it in all its details, a thing which he would absolutely refuse to do, in many instances at least, if he had the slightest suspicion of what it could mean. The physician is therefore, so to speak, in a position to come up on the blind side of the patient, to see the play of his unconscious fantasies, to be let behind the scenes, as it were. Now if he can penetrate these disguises then he not only is capable of determining the meaning of the dream, but also he learns in this way the nature of the repressed complexes, and is therefore in a position to begin to read meaning into the symptoms of the neurosis. The whole matter of dream interpretation constitutes a special chapter in psychology and is hardly discussable in a text-book of this sort with its necessary limitations.

In addition to the dream analyses one should also inquire into the fantasy formations of the patient, fantasies which are formed in the daytime. They are interpreted on the same principles as the dream.

The events uncovered by the method of free association must be dealt with as facts. It makes little difference whether they ever did happen in reality or not, they are nevertheless *psychological facts;* they represent the way the patient thinks and so have just as much value as if they represented real occurrences.

It will be seen, therefore, that psychoanalysis is a lengthy, painstaking, detailed dissection of the mind of the patient sufficient for the reconstruction of the symptoms. This dissection starts at the surface and may go to practically any depth. Bearing in mind the principles already elucidated it will be seen why it is possible to effect an apparent cure at various levels; why sometimes a single conversation may apparently produce the miracle of a cure, while in other patients a half dozen will produce the same effect, and in still others months of careful work are required. The individual has been thrown out of adjustment by causes which, in their last analysis, have been operative the greater part of his life. Up to a certain point, however, up to a certain difficulty, he has been able to get along. Now, when this difficulty comes he breaks and the neurosis makes its appearance. If he can be patched up, so to speak, rehabilitated, back to the point where the break occurred, an apparent cure results. For these apparent cures it will be seen that it is only necessary to carry the patients back to a point at which they are capable of making adjustment. This

is what very often happens, especially in methods of treatment other than the psychoanalytic. For a real cure of the patient, however, something very much more radical than this is required: the analyst has to proceed to ever and ever deeper levels until he has sought out and found the final stronghold of the neurosis. Anything short of this can only serve to effect a compromise.

This seems to be the most advantageous point to answer the question which is constantly being asked and which it seems impossible to make many people understand, namely, the question of how the analysis, the unravelling of the symptoms, produces a cure. In order to understand that it is necessary to recur to the statement that the illness is due to a conflict and that one element of the conflict is unconscious to the patient; he does not know what it is that he is fighting. He therefore cannot deal with it frankly, openly, intelligently. One is reminded of the story of a certain king who propounded the question to the wise men as to why a bowl of water was not increased in weight when a live fish was put into it, while it was increased in weight when a dead fish was put into it. This created a tremendous disturbance. All sorts of arguments and reasons were propounded, heated discussions arose, and the wise men were quite generally out of tune with one another. Finally, it occurred to some one to try the experiment and see what the facts were. As soon as the experiment was tried and the facts were determined there was no longer any cause for argument. The conflict subsided. This is quite comparable to the position in which the patient finds himself, fighting something of which he is unconscious and about which he knows nothing. The facts in the case in regard to the fish in the bowl of water were unknown, and as long as they were unknown nothing but chaos reigned among the wise men. As soon as the facts were brought to light, however, by proper experimentation, then there was no longer anything to fight about. In addition to these reasons for the subsidence of the conflict there are others that are of more or less importance in different cases; among them is a large element of reëducation to which the patient is subjected throughout the period of psychoanalysis. The cause of his neurosis implies that he is somewhat infantile, somewhat undeveloped. The physician, who should be a man of wide learning, whose business it is to deal with the problem of right living, cannot help but infuse into the patient in the many hours of conversation a philosophy of life which is helpful, and this undoubtedly happens as one of the most important elements in the reëducation, development and rehabilitation of the patient.

While the simple uncovering of the meaning of a symptom is often enough to make it disappear, for the real rehabilitation of the patient the whole meaning of infantile ways of reacting must be finally understood and the patient must be willing to forego this means of obtaining pleasure in order to advance to a higher level of adjustment, to solve the difficulties by attaining a higher level, which means at once renunciation and fulfilment.

And finally, about the matter of transference. A considerable number of the neurotics and the psychoneurotics and of persons who require psychotherapeutic treatment, are excessively introverted, that is, their interests are turned within, upon themselves, and they cannot be made over into efficient people capable of dealing with the outside world of reality until their interests can be made to flow outside of themselves, until they can become interested in persons and things and events. In the course of psychoanalysis, if it is to proceed successfully, one of the earliest things that happens is that the interest of the patient begins to flow upon the physician. It is transferred to him. This is a matter of utmost importance. It is a matter which should be watched with the greatest care, for it is the barometer of the relationship between physician and patient. It is the factor in the personal equation which plays such a great part and which was supposed in the old days to play practically the only part. As soon as the transfer begins to take place then the patient will begin to bring dreams for analysis, and in other ways to show, so to speak, every desire to please the physician by doing as he wishes, and before long one will generally find that the dreams are occupied with the idea of the physician, they are transfer dreams. Now in these dreams one may find exactly how the physician is held in the mind of the patient. In symbolic form the transfer dream may indicate that the physician is held in high regard and that he is respected, and this is of course as it should be. On the other hand, he may have failed to deal with a certain situation adequately, and the dream will show that the patient is disappointed or that perhaps some idea that the physician suggested the patient thinks is ridiculous and silly, or the physician may make the mistake of talking over the head of the patient so that the patient cannot understand, and the dream will say what the patient could not say himself, that it is all too deep for him, that he cannot follow, and that the physician is altogether beyond him in the whole matter, and it all seems quite hopeless. The transfer dream therefore becomes a very important matter and requires the physician constantly to look within and to exercise his self-critique, for he must always realize that if he fails at a certain point the trouble is not with the patient, but with himself, and sometimes the dream will indicate what the trouble is. The meaning of the transfer is, of course, that the patient cannot unburden his very soul, cannot stand unclothed in all the nakedness of his real self, cannot, in other words, completely confess himself to an indifferent person. There must be something in the physician which commands the patient's affection, respect and confidence, and therefore the psychoanalytic work, while it makes great demands upon the patient also makes great demands upon the physician.

Sometimes the transfer is of such a character and sufficiently intense of itself to interfere with the progress of the analysis. Under these circumstances it must be discussed with the patient, sufficiently discussed to do away with the resistances it has raised, and finally at the completion of the analysis the whole matter of the transfer must

be freely gone into and analyzed, so that there is no misunderstanding as to what its real meaning might be. The patient is to be placed in full and complete possession of all of the facts, which means a full and complete possession of himself. Nothing must be hidden from him, the mirror must be held up so that he can see himself in it in every detail.

It is because of the necessity of transfer to successful psychoanalytic handling of a case that it is undesirable to have another physician treating the patient at the same time. The other physician might acquire the transfer, and this would prevent the psychoanalyst from accomplishing anything. This is especially apt to be the case where the other physician has to do with the physical condition of the patient and has to come into personal contact with him in making examinations and the like.

As already said, the transfer is the result of the beginning flowing outward of the patient's interests into the world of reality. The physician naturally is the one toward whom this interest first flows. He therefore, so to speak, puts himself in a position to be utilized by the patient; he becomes a bridge by which the patient is able to get back into the world and in his progress in the analysis he is utilized, as it were, by the patient, as the receptacle for all the affects that have been aroused by the emotionally important persons in his life. The physician by his relatively impersonal relations to the patient (sitting behind him during the analytic hour) invites this type of emotional catharsis which at the same time serves to further the analysis by uncovering significant material. At the completion of the treatment, when the transfer has been fully analyzed and the patient understands what it means, then the physician steps aside. Having served the purpose of a bridge, having gotten the patient back into reality, he steps aside and leaves the patient there to stand upon his own feet.

Transference is not an isolated nor an unusual phenomenon nor is it one confined to the psychoanalytic situation. It is a universal psychic way of progress, the way of interest, which takes the individual from lower to higher levels in his ways of thinking and acting. It is seen easily in the little boy's desire to emulate his father and later to take some great man as his model for imitation. It is the basis of all interest in reality and is the constant tool used for affecting adequate adjustments with the outside world. Life is a constant play of transfers of interests, that is of love, and in proportion to the capacity to love, to give of ourselves to some goal, some ideal, are we capable of living our lives at our best.

The transfer in psychoanalysis is therefore a tremendously powerful influence for good and also for evil if used ignorantly or for venal ends. It is used in this latter way by those who do not know what they are doing and sometimes by a certain type of practitioner who uses it solely to keep the patient coming to him and paying fees. Psychoanalysis thus demands a high type of conduct on the part of the physician. It demands that he devote himself unselfishly and

unstintingly to the sole object of the patient's welfare. It is only when he is able to do this that he becomes a worthy object of emulation and so places himself in an attitude toward the patient, to utilize to its fullest extent for the patient's good, the great power he possesses in the transfer.

The great obstacle to the treatment of the psychoneuroses is the general obstacle that the patient in a certain real sense does not want to get well. There are two contrary trends struggling for supremacy in his psyche. He has two sets of desires each trying to gain fulfilment and each diametrically opposed to the other. Now the symptoms of his neurosis constitute a compromise, and in his then state of mind the only compromise, the only solution of the problem possible to him. He therefore, although he wants to get away from the suffering of his neurosis, still is unwilling to give up the symptoms which compensate him, even though that compensation be inadequate. This is illustrated in many ways. For example, the patient insists upon leading the physician back to infantile occurrences in order to escape a frank discussion and facing of present problems. Again, by the free associations the patient will lead the physician up all sorts of blind alleys for the same purpose. The patient always wants to avoid his task. It is the function of the physician to hold him to it. To this end it is important that the physician should have a wide knowledge of the meanings of symptoms and symbols and be able fairly clearly to see at once their general significance otherwise he will be led into interminable and futile discussions. It is only in the final stages of the analysis, when the patient is made whole, at one with himself, that he can understand why it is that these things have come about and how it is that he no longer needs his illness, but can dispense with it. So for a long time the physician has to contend against an innate desire on the patient's part to retain the symptoms of his illness. This is particularly well seen in the compulsion neurotic. Here the patient has built up an elaborate structure which he considers quite as does the artist his work of art and he, equally with the artist, resents all attempts to tear it down.

In addition to the strict analysis of the patient it must not be lost sight of, particularly in severe neuroses and more especially in the more malignant psychoses, that the patient is the center of certain dynamic, affective forces in his personal environment (the family in particular) and that it will often be necessary to modify those forces, to change their nature, direction or strength, by dealing with the particular individuals, in order to make the way possible for the patient to get well.

Various accessory forms of treatment, such as baths, massage, sanitarium treatment, travel, etc., have the same place here as with the actual neuroses mentioned before. They should never be considered primary, but only as secondary. If they are utilized without a thorough analytic understanding of the patient they are quite as apt to do harm as good, because by no possibility can it be foreseen what the results

will be unless the matter which has to be dealt with is known beforehand.

At the present writing it would seem that one of the most useful adjuncts to the psychoanalytic treatment of the neuroses might prove to be a carefully selected organotherapy. The endocrine imbalances represent the organic handicap of the patient and there is evidence accumulating that a judicious effort at restoring the lost balance will be of value in tiding the patient over a period of reconstruction that he might be unable to negotiate without such assistance. Having passed the critical period the organotherapy may perhaps be dispensed with for the imbalance may well often be the result of a prolonged conflict and when the vicious circle is once broken the balance returns.

It must be borne in mind that the symptoms of a neurosis or a psychoneurosis may cover and conceal a true psychosis. This will appear in the course of the analysis, but cannot always be foreseen as the result of the first examination. It is a possibility that should be borne in mind so that the physician may be guarded in what he says in regard to the possibilities of treatment and the ultimate outcome of the case.

CHAPTER XVI.

MANIC-DEPRESSIVE PSYCHOSES.

The general concept of the manic-depressive psychoses has been an extremely difficult one for some reason or other for many people to adequately grasp. From the earliest times the marked cases of melancholia and of maniacal excitement have of necessity been observed and in many instances ably described, and at one period of time the manias and the melancholias constituted by far the larger portion of the types of mental disorder. At that time in the history of psychiatry, when the diagnostic hair-splitting was at its height, innumerable varieties of mania and melancholia were described and given specific names. They were differentiated on the basis of whether hallucinations were present or not, whether delusions were present or not, and upon like matters of what seems now superficial observation. It was observed also that there were a certain few cases in which states of excitement alternated with states of depression. These so-called circular types have been recognized for a long time. It goes without saying that during this period, when psychiatry was in a purely descriptive stage, a stage from which it has not yet by any means fully emerged, when the excitements and the depressions were the symptoms in evidence, that excitements and depressions belonging to all sorts of conditions, dementia precox, general paresis, arteriosclerosis, toxic and infectious psychoses were included in the broad concepts of mania and melancholia that were prevalent and that the formulation of the manic-depressive group has been the result of a gradual weeding out from all of these various sources of the things that belong together and the elimination of those which further analysis showed were not similar.

The manic-depressive concept, however, was not definitely formulated until Kraepelin, by a study of life histories, described the condition as a disease of affect fluctuations which might at one time manifest itself by the profoundest depression and at another time by the highest grade of excitement. Even after this formulation many thought that the term manic-depressive psychosis applied only to the so-called cases of "circular insanity" and failed to appreciate that there were inherent and fundamental relations between the two extremes of affect disturbance. It was difficult to understand how there could be any relationship which bound together cases of such unlike outward appearances and they failed to see that a patient who manifested a single attack either of depression or excitement could be diagnosed as a manic-depressive solely because the depres-

sion or the excitement, as the case might be, presented the symptomatology of the depression or the excitement as found in other patients who presented a series of attacks of both kinds.

The present-day concept of the manic-depressive group considers these as mental disorders essentially of affect fluctuations manifesting themselves sometimes by depression, sometimes by excitement, sometimes by a mixture of the two and finally the concept has grown to include, on the one hand, a manic-depressive character which tends to react on the basis of a labile affectivity, and on the other, various atypical manifestations which present secondary symptoms that tend to mask the fundamental affective ones. And so the concept emerges that finds the root of the psychosis in certain character traits that never amount to serious pathological manifestations and certain very mild affect fluctuations, the cyclothymias, and including certain atypical varieties with secondary symptoms of delusion formation and disorders of the sensorium that are of greater practical significance than the disturbances of affect.

Here also, as elsewhere, it is seen that the manic-depressive type of reaction merges into other types so that reactions that closely resemble the manic-depressive are seen in certain phases of precox, whereas various of the depressions and excitements from other causes also closely parallel the manic-depressive types in their symptomatology. With this concept of reaction types in mind, these mergings into adjacent territories are understandable. With the concept of a disease entity, one which looks upon disease as a definite something back of the symptoms and which produces them, it is impossible to understand the meanings of these attenuated and border states.[1]

Bodily Type.—From the Greeks to the present day there have been repeated efforts to define types of individuals. A four-fold type was developed to serve military ends and worked out, more especially by the French during the World War. The four types are the digestive, respiratory, muscular, and nervous. More recently Kretschmer[2] has described a type of bodily configuration that seems to be closely correlated with the cycloid make-up: It is characterized briefly as follows:

Pyknic Type.—Characterized by the pronounced peripheral development of the body cavities (head, breast and stomach), a tendency to a distribution of fat about the trunk, and a graceful construction of the motor apparatus (shoulders and extremities). In general the man is of middle height, rounded figure, with a soft broad face on a short massive neck sitting between the shoulders. The magnificent fat paunch protrudes from a deep vaulted chest which broadens out below. The limbs are soft and rounded with little muscle or bone relief, often quite delicate. The joints of the hand in particular are often slim and almost elegantly formed. The shoulders are not broad but rounded, high, and pushed forward together and often set down

[1] For literature from 1906 to 1910 see Referate by Hamburger. Ztschr. f. d. gesamte Neurol. u. Psych., Band **2**, Heft 9.

[2] Körperbau und Charakter, 1921.

against the breast with a characteristically sharp depression on the inner deltoid curve. The shoulders seem to be slipping downward and inward over the swelling chest, the neck seems to disappear and the upper spine is slightly kyphotic. The point of the chin is directly joined with the upper forehead without any definite bends by a sloping line. The breast-shoulder-neck proportions, the shape of the head and face, and the disposition of the fat are characteristic. The face is, from the front, of a smooth five-cornered or shield-shaped type. The skull is large, round, broad and deep but not very high.

Etiology.—In the first place there are certain hereditary factors to deal with in this class of cases as there are in the precox group. There are certain families which show a preponderance of the manic-depressive psychosis, as there are families that show a preponderance of the precox type of reaction. A recent study of Riebeth[1] would indicate that this statement only applies to a given generation. While manic-depressive and dementia precox psychoses seem not to be found in the same generation of a given family, the two psychoses may occur in different generations, in which case dementia precox is found in the descendants of manic-depressives, but the reverse relation seems not to occur.

In harmony with the hereditary tendencies which appear to be present in this group of cases it is found that the group may be widely differentiated into two extremes, the one in which the constitutional factors appear to be predominantly in evidence and in which the various attacks appear to originate either without any cause at all or at least without a determinable or apparent cause that is sufficient, the *endogenous type;* and the other which appears to be more or less largely determined by causes which are apparent, such as the inability of the individual to adjust to certain conditions of life and repeated breakdowns with the return of these conditions, the *reactive type.* It is important to recognize these two groups of cases, between which of course every intermediate variety may be found, because of the significant bearing which the type of etiological factor has upon the probable outcome of the therapeutic attack.

In describing these types of mental disturbance the two principal phases—the manic and the depressive—will first be described and then the various forms of periodic psychoses of the manic-depressive group will receive consideration, while finally certain less common combinations of symptoms known as the mixed states will receive attention.

Manic Phase.—The cardinal symptoms of the manic phase of a manic-depressive psychosis are three in number, namely (1) *flight of ideas,* (2) *psychomotor hyperactivity,* (3) *emotional exaltation.* These three symptoms may manifest themselves with any degree of severity, and the severity of the symptoms may vary within wide limits at different times throughout the course of the attack. The three symptoms,

[1] Ztschr. f. d. gesamte Neurol. u. Psych. Orig., Bd. **31,** Hefte 4 und 5, p. 429.

too, may not be all of the same degree of severity, for example, the flight of ideas may be extreme and out of proportion to the degree of psychomotor activity which may show only a slight increase.

Taking the attack as a whole the ordinary acute varieties are generally designated as acute mania or acute maniacal excitement. Still milder grades are spoken of as hypomania and the more severe grades are generally termed acute delirious mania. These three degrees of excitement are the most convenient captions under which to describe the manic phase.

Hypomania, which is the mildest of these three degrees merges, of course, upon the one hand into higher grades of excitement, such as the acute maniacal, and on the other into conditions of cyclothymia, which may be termed hypomaniacal, but which constitute a special group of this class of cases which will be considered separately. In this condition, at least in the simpler cases, there is a disorder which involves the *process of thinking* more than it does the *content of thought.* The particular ideas and acts may not be unusual and yet abnormal. This state of affairs is excellently illustrated by the hypothetical case cited by Mercier.[1] "Its subject rises early, full of schemes of business or pleasure. He fusses noisily about the house, indifferent to his disturbance of other people's slumbers. He is very impatient of delay, he cannot wait a minute for anything that he wants and if it is not forthcoming on the instant, he flies into a rage. The course of the post is not expeditious enough for him. He sends his letters by telegraph and his letters are extraordinarily numerous. They would be numerous in any case, but their number is doubled, and more than doubled, by the frequent changes of his mind, and by the impulsiveness with which he acts upon every passing whim. He determines to make some purchase, probably a very unnecessary one, but one for which he can adduce twenty plausible reasons, and he writes to tell his solicitor that he will call the next morning. Scarcely is the letter posted when he sees that he will attain his object more quickly by asking his solicitor to lunch. He telegraphs accordingly. Before his messenger returns, it occurs to him that he had better ask the vendor to lunch also. Another telegraph is dispatched and since he cannot entertain more than one visitor at his club, another

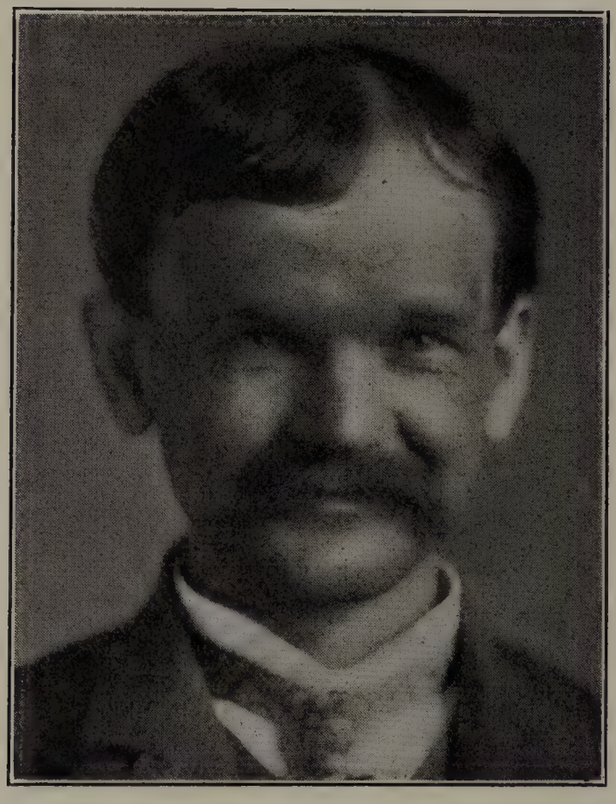


FIG. 436.—Facial expression of hypomanic with flight of ideas.

[1] A Text-book of Insanity, The Macmillan Company, 1914.

must be sent to the solicitor to announce the change to a hotel. Then he remembers that he has been drawing heavily of late on his banking account, and that he may not have the necessary funds available. Another telegram to the bank. But if there are insufficient funds in the bank, he will have to sell stock to raise the funds; another telegram to his broker. Then he determines that it will be better to pledge the stock to the bank rather than to sell it. More telegrams to the broker and to the bank. The broker won't like the contradictory orders—never mind; ask him to dinner—ask them all to dinner. Put off the lunch and have a dinner instead, and ask the solicitor, the vendor, the banker and the broker. Yes, and why not Smith and Jones and Robinson as well? More telegrams; and then since two out of three of the invited guests decline, the whole thing is postponed, also by telegraph. Meantime, in the intervals of telegraphing, his hands have been full. He has been constantly ringing the bell and giving orders—giving them, modifying them and countermanding them—constantly wanting something fresh, running up and down stairs, writing letters, haranguing this person and that, flying into a rage upon the slightest opposition, tearing the bell down on the slightest delay and talking almost incessantly."

In this example the subject's acts might, almost all of them, be considered normal, with of course the exception of those due to undue irritability or anger. Aside from this, however, each act is consistently directed to some definite aim. The disorder is not in the content of thought so much as in the process of thinking, and manifests itself by a rapid and too frequent change of direction. This is the phenomenon known as *flight of ideas,* and may be perhaps better illustrated by a stenogram:

"Do you know I was kidnapped to be sent here twice. I saw a mock funeral of me before I left home. This was done because I am a great inventor. The Pope of Rome is the greatest human being in the universe. He is the head of the Catholic Church. My head (association of the word head in two different meanings) is good and sound, and I am certainly not insane. Do you hear the ticking of the clock? (External association.) It says, 'call the little heifer, the heifer is sick.' Did you ever see the gloves veterinary surgeons use when they doctor sick cows? (Internal association.) Say! what are you keeping me here for anyhow? I want to go home. (Here he was asked how he slept at night.) I have slept excellently; that is because I am of such a strong constitution. The Constitution of the United States (association as above with the word head—probably the association is in large part at least a sound or, as it is called, a *clang association*) was signed by Thomas Jefferson. He was just a man, but he was not the inventor I am."

In this phenomenon of flight of ideas the patient either has no guiding idea or else at once loses it so that there is no consistent and sustained effort directed toward attaining a goal idea, and the thought therefore wanders here and there under the influence of

chance associations. As a result the train of thought instead of progressing, changes direction, frequently returns upon itself, and never reaches a logical end. The various ideas are not on that account, however, incoherent, that is, they do not fail to cohere or to be connected with one another, although it may be quite impossible at times to see wherein this connection lies. If the associations are external, that is, originate in the surroundings, it is often quite possible to place them; when, however, they are internal, that is, originate within the patient's mind, it may be quite impossible to conceive what they are. In the example just cited, while there are many places where the connecting link is missing, probably because it was an association formed entirely within the patient's mind, still the connection can be made out in a sufficient number of instances to establish the characteristics of the train of thought. One of the principal characteristics of this type of the train of thought is, as we have seen, its great tendency to change of direction, and when, for example, this change of direction takes place under the influence of external associations, such, for example, as the ticking of the clock, as noted in the stenogram given, the phenomenon is known as *distractibility*. Any sensory impression is liable to be the starting-point of idea association, so that these patients' trains of thought may be turned at will, almost, by such devices as shaking a bunch of keys before them, saying some word loudly, showing them a newspaper, or in other words, momentarily distracting their attention.

The *emotional exaltation* is also well shown in Mercier's example. The patient is constantly doing things which testify to his idea of his own importance. One is struck, however, by a symptom in the emotional field which is perhaps more fundamental than the simple exaltation and which corresponds to the symptom just cited and the motility disorders to be described, that is, the great lability of the emotions, the rapid play of different emotional reactions, exaltation giving place to irritability, to anger, annoyance and the like. There is no sustained emotional attitude, as there is no sustained direction in the train of thought. The psychomotor hyperactivity is also well illustrated by this case. The activity of the patient is seen to be constant and unremitting, but again it has the same qualities as the train of thought and the emotional attitudes inasmuch as it is not sustained for any length of time in any particular direction, it does not get anywhere, while from time to time, under the influence of rapid changes of emotion the acts tend to impulsiveness. There is marked *pressure of activity* just as there is *pressure of speech*, and the patient appears to be living under terrific and unremitting tension without power of direction.

In this condition, therefore, the patient is constantly active, busying himself about one thing and another, talking continuously meanwhile often in a loud and rather boisterous manner, while emotionally, exaltation is manifested by good humor, a smiling countenance and increased self-esteem, punctuated mayhap by attacks of irritability

or impulsive anger from little or no cause. His confidence in his own ability is unqualified and is shown in the outlining of all manner of schemes of work, investments, business enterprises and the like. Flight of ideas is marked, though not of high degree, the conversation changing at frequent intervals from subject to subject and the activities show a like characteristic, there being no consistent effort directed at any one aim for any length of time. Letters are often written in great numbers and their contents exhibit the same characteristics as do the speech and conduct. The patient is fully oriented, there is no clouding of consciousness nor delusions. In spite, however, of the lucidity and apparent abundance of energy the real efficiency of the individual is greatly reduced because of the lack of consecutiveness in application.

Ofttimes the picture is complicated by the addition of symptoms due to alcoholic indulgence which is very common with patients in this condition, many of whom show marked moral delinquencies, but because of their lucidity and facility of expression often elude the authorities, being at once discharged after examination when apprehended because of supposed mental disorder. This complication with alcohol will be spoken of again under the head of Diagnosis. Sexual excitement is also quite frequently and characteristically in evidence in these cases and leads to moral delinquencies which show a still further departure from the patient's usual manner of conducting himself. When complicated with alcohol the whole conduct may be quite non-understandable except to those trained in the recognition of this class of cases. The sexual excitement, of course, is an especially unfortunate and dangerous symptom in young women and may lead to particularly regrettable actions.

Acute Mania.—The next grade of maniacal excitement presents perhaps the most characteristic picture of this phase of the disease, exhibiting the symptoms to best advantage, though it must be understood that the symptoms of the different grades differ only in degree, intermingle and are found alike in all the conditions.

In this degree of excitement the flight of ideas is well marked and may even become so extreme at times that the train of thought has the appearance of being quite incoherent. Distractibility is a prominent feature and the patients are constantly diverted by inconsequential happenings in their environment. The tendency to rhyme is quite frequent and the words heard by the patient spoken by those about him, although they may have no reference to him and are not addressed to him in any way are often woven into or form the starting-point for these rhymes or for associations. The characteristic of these associations is their superficiality and when words that are heard are introduced into their conversation the basis of their choice is often nothing more than the sound similarity (clang association). It is quite remarkable how such a patient who is apparently paying no heed to what is going on about him will catch a chance word or phrase uttered by some one, perhaps a considerable distance away, and intro-

duce it into the stream of his conversation. Consciousness may be somewhat clouded and there may be at least apparent disorientation, particularly for persons. This apparent personal disorientation, however, is dependent in the main upon two factors. In the first instance the patient does not adequately perceive the environment, he does not dwell long enough upon any one particular element of it to comprehensively grasp it in the rapid and transitory survey which it receives from him; its elements are not adequately perceived and therefore are often misunderstood, partly because of this superficial attitude toward the environment and partly, also probably, because of deeper reasons. Slight resemblances to friends or relatives are often seen in the patients and nurses, and these resemblances are magnified out of due proportion, and so these various persons are addressed by the names of members of the patient's family for instance. These resemblances do not necessarily result in a permanent and fixed mistake. The person who is at one moment addressed by one name is a little later addressed by another, and not infrequently the whole situation is further complicated by the wit reaction of the patient who gets a good deal of fun out of his facetious remarks and his apparently meaningless mistakes. These errors being not firmly fixed are frequently spontaneously corrected by the patient, at least at times.

The disorder of attention, flight of ideas and distractibility are all elements which produce a transitory and a superficial survey of the environment by prohibiting any fixation or dwelling upon any particular element of the environment or even of the ego and tend to produce a condition of the content of consciousness in which all of the ideas are given the same value. No one thing is attended to long enough to enhance its importance over that of others. The patient voices ideas first about this subject and then about that, changing from one to the other, not because of any appreciation of differences of importance, but in response to the pressure which makes it impossible for him to rest anywhere, so that all ideas tend to reach the same level of importance in his consciousness. There is what is called a *levelling of ideas*.

While hallucinations are not an essential part of the picture they may occur, but when they do, like all of the other elements, they tend to be only transitory and usually are rather simple and elementary in character.

The delusions also are inclined to be changeable. They partake characteristically, when present, of the grandiose character, but usually lack the element of extreme improbability found in conditions of dementia precox and general paresis. Occasionally a persecutory paranoid system of delusions develops in the manic phase of this disease, but this class of delusions is more apt to develop and present a fairly well organized system in the milder grades of excitement.

The psychomotor activity is constant. The patients are unable to remain at rest (pressure of activity), they run and jump and turn somersaults, wave the arms about, tear up clothing, destroy plants,

break furniture, howl and yell all night long, and go almost absolutely without sleep. Genital excitement may be prominently in evidence. The excitement may be so great that the patient does not even take time to eat; food placed before him is perhaps tasted and then thrown about like everything else that comes in his way, so that with the lack of nutrition, lack of sleep and with the unremitting activity, emaciation is a constant feature. In less marked degrees of excitement, however, where the feeling of well-being is the controlling factor, it is common for the patient to gain somewhat in weight.

The emotional exaltation is marked and shown by boisterous laughter and remarks showing exaggerated ideas of self-esteem. Patients, however, are spasmodically apt to be irritable, bursting into attacks of anger without adequate reason and often are a constant source of trouble, annoyance and agitation upon the wards where they are confined. The emotional condition is as changeable as the trend of thought or of the direction of the activities and emotions of radically opposed qualities may easily replace each other.

Delirious Mania.—This condition is merely an aggravated state of the acute mania already described. The flight of ideas here has proceeded to almost complete incoherence. The activity is unremitting and consciousness is more clouded and hallucinations more in evidence. The lack of sleep and proper nourishment, with rapid emaciation, soon leads to great physical exhaustion, while the constant activity not unusually results in slight wounds which, even though properly dressed, are soon exposed to infection when the patient tears off the dressing. Local areas of suppuration thus develop, there is a mild degree of infection with some temperature, which coupled with the exhaustion and the toxemia, produce a picture more distinctly delirious with marked clouding of consciousness and great incoherence.

The *acute delirious mania* which used to be described and was regarded as always fatal, was undoubtedly in a certain proportion of cases the hypermania of the manic-depressive psychosis to which perhaps had been added, as just described, symptoms of infection, toxemia and exhaustion, which made the picture one of delirium. Many other cases were undoubtedly also included under this general caption, more particularly deliria associated with acute diseases of the internal organs, such as acute nephritis or pneumonia. One who has had experience with these cases can understand how such conditions might go unrecognized, owing to the almost physical impossibility of subjecting such patients, in their wildly excited condition, to anything approaching an adequate physical examination.

Chronic Mania.—There are a very few cases that pass into a condition of chronic mania and usually, though not always, have mild excitements that may last for a number of years. These conditions, on the other hand, may be practically nothing else but character anomalies, cases of constitutionally heightened (manic) mood. Such prolonged phases of the disease, however, must be borne in mind as possibilities.

Depressive Phase.—Like the manic phase this phase also manifests itself by three cardinal symptoms each diametrically opposed to the corresponding symptom of the manic phase, namely (1) *difficulty of thinking*, (2) *psychomotor retardation*, (3) *emotional depression*.

This group of symptoms may, as with the manic group, manifest itself with any degree of severity, and the three symptoms may severally and individually vary, irrespective of each other. The retardation, for example, may be quite out of all proportion to the depression.

As with the manic phase, it is convenient to consider the depressive phase in three different grades.

Simple Retardation.—The word retardation is here used to refer not only to psychomotor retardation, but to the difficulty of thinking also, probably quite similar phenomena, the one more particularly in the sphere of thought and the other more particularly in the sphere of psychomotility. These patients move and speak slowly and perhaps in a low voice, by preference answering questions in monosyllables. These outward evidences of difficulty of thinking and moving are, however, more marked in the next stage of the depressive phase, that is, in acute melancholia, while here it is more usual to see the patients merely preferring to be by themselves, disinclined to associate with others, keeping to their room and quite unable to make any mental effort. They are not equal at all to going on with their work. They may not, for example, feel equal to writing letters or even to reading the newspaper.

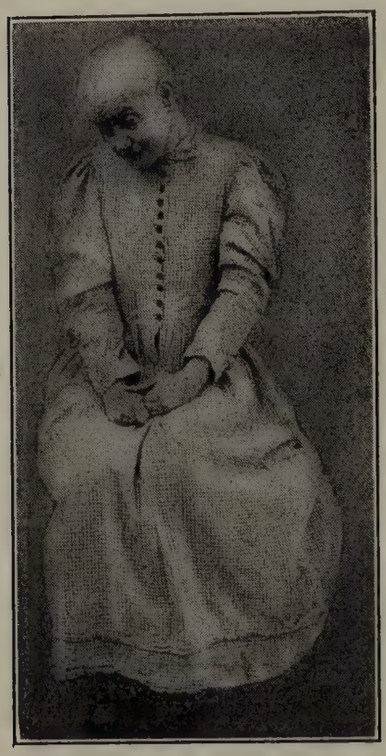


Fig. 437.—Severe depression of several years' duration.

Emotionally these patients are usually somewhat depressed, but the depression may not be especially marked, it may only appear on questioning. Consciousness is clear and the patients are fully oriented and often have a realization of their mental invalidism.

Acute Melancholia.—In this grade of depression the three cardinal symptoms are manifested in a much more pronounced way. The patients are characteristically inactive, sitting by themselves, showing little or no tendency to associate with others, their movements are slow and deliberate (executive retardation) and it often takes a considerable time to initiate them (initial retardation). The speech is similarly affected; it is slow, often monosyllabic and sometimes almost inaudible. Initial retardation is noticeable here also. The emotional depression is profound and is indicated in the general attitude of the patient which is one of flexion of the body, the hands lying limp in the lap, the head inclined forward, the chin resting on the breast and a marked facial expression of sadness. The subjective state of these patients is described by a feeling of difficulty of thinking and grasping the meanings of things and of

their feeling of inadequacy, of incapacity for all effort or even thought. There is a marked feeling of decrease in the mental activities and the patient does not feel that he has control of his mind and can use it effectively. In the same way he feels an interference when he comes to exert his will in the performance of voluntary acts. There is lack of energy, lack of ability to initiate or to sustain an act or a series of acts, and in the mental sphere alone the patient finds himself quite unable to carry out a series of consecutive mental acts which lead to a logical issue. He cannot come to conclusions, he has an overwhelming sense of weariness, of relaxation, of inadequacy.



FIG. 438.—Depressive facies.

This general feeling of inadequacy and difficulty of thinking as above described fits into and forms a part of the emotional attitude and acts with it in determining the character of the delusions. The delusions are typically self-accusatory and hypochondriacal. The patients think themselves responsible for all the sin, wickedness, privation and suffering in the world; they are the cause of the unfortunate condition of their fellow patients; they themselves have committed some great sin and their souls are forever lost. As they occupy themselves with their own moral states so they occupy themselves with their bodily condition and believe themselves sufferers from incurable disease, think that their organs are decayed, something has happened to their brains, their bowels are stopped up, their bones broken and other such somatopsychic ideas. When the organic sensations are altered patients have strange feelings which they interpret as indicating some mysterious thing going on within their body, and such sensations may be at the basis or associated with some of the hypochondriacal ideas. The emotional depression may at times reach a very high grade and express itself in anxiety attacks, moods of apprehension, fear of impending danger, a nameless dread of something going to happen and the like. The whole world is looked at, so to speak, through blue glasses. The sad, depressed mood colors every perception and so the perceptions are more or less incorrect and distorted to fit the mood.

Hallucinations may occur, but consciousness is usually clear and the patient well oriented. There may, however, be a lack of orientation toward their surroundings dependent upon the fact that they are wrapped up in their own thoughts and the environment is not attended to.

Physically there is almost always constipation, a coated tongue, indicanuria, poor appetite, loss of weight, disturbed sleep and often circulatory disturbances with cold extremities.

Depressive Stupor.—This is the third and most severe grade of the depressive phase. In this condition the retardation, both in the field of psychomotility and in that of thought, has proceeded to the extent that the patient lies wholly inactive and mute; he has to be tube-fed, and his every want ministered to.

During this period of absolute inactivity it may be that the patient is suffering from delusions and hallucinations of a depressive and horrifying nature which perhaps are shadowed forth by an anxious expression of countenance, but the details of which can only be learned after the patient has aroused sufficiently from his stupor to be able to express himself. The hallucinations may appear to the patient much as in a dream and absorb his attention to a very great extent. This condition of stupor is not uncommon in the course of the depressive phase, but usually occurs as an episode rather than as a distinct form of the disease.

Benign Stupor.—Hoch[1] has described in great detail the stupor reaction and in his differentiation of the benign and malignant stupors allies the former with the manic-depressive group and the latter with the schizophrenic.

The *mental symptoms* of benign stupor are: *Poverty of affect.* The patients are apathetic to an extreme degree, exhibiting no interest in themselves or their surroundings, their faces are mask-like, their voices colorless. Sudden mood reactions may break through at rare intervals. *Inactivity* is a cardinal symptom there being almost a complete conservation of all spontaneously initiated action and even of reactive movements. Even swallowing and winking may be inhibited. There may be *catalepsy,* which is possible related to this inactivity, and the opposite symptom *negativism.* This latter may give rise to sudden outbursts of anger or to muscular rigidity and may also be the explanation for *wetting* and *soiling.* The *thinking disorder* is a constant feature. When the patient is at all accessible there seems to be a failure of intellectual functions and after recovery the memory is for the most part a blank as to occurrences during the stupor, even striking experiences. The peculiarity of the *ideational content* is a preoccupation with the theme of death. Probably related to this are the sudden suicidal outbreaks. The *physical symptoms* are less definite. A low fever is common as are skin and circulatory anomalies. A loss of weight is the rule and menstruation is almost always suppressed.

This stupor reaction shows many degrees which are roughly described and explained as degrees of regression. First there is quietness, inactivity and apathy with loss of initiative and suggestibility (acceptance of other's ideas), indifference and stolidity; then there is occupation with the idea of death which is associated with anxiety, if the instinct of self-preservation produces fear or if this lapses then death is accepted and spoken of with indifference; the next stage is marked by the reanimation of auto-erotic interests with perverse reactions and negativism and auto-erotic habits; finally all evidences of mentation fail.

Ideas of rebirth are commonly associated with those of death and point to a constructive and progressive tendency. Schizoid admixtures are of ominous significance and indicate that the stupor belongs in the dementia precox rather than in the manic-depressive group.

[1] Benign Stupors, a Study of a New Manic-Depressive Reaction Type, New York: The Macmillan Co., 1921.

Chronic Depression.—There are certain patients who present for long periods of time a depressed mood. These cases may be mild depressive phases of manic-depressive psychosis or they may be character anomalies, cases of constitutionally depressed mood and so show the close interrelations between the normal fluctuations of emotion and those that are pathological.

The Periodical Types.—Under this head are included those forms of the manic-depressive psychosis which from time to time have been severally described as recurrent mania, periodic mania, intermittent mania, recurrent melancholia, insanity of double form, alternating insanity, circular insanity, etc.

All of these psychoses are merely different manifestations of manic-depressive psychosis, the manic and depressive stages being represented in various relations, often separated by a recovery interval. Thus recurrent mania would be recurrent attacks of a manic phase separated by well intervals, similarly for recurrent melancholia, while alternating insanity would consist of manic and depressive attacks, each followed by a recovery interval; circular insanity, on the other hand, being cycles of manic and depressive phases without intervals of separation, while insanity of double form would consist of cycles of excitation and depression, each cycle followed by a lucid interval. Other varieties might be described, but it suffices to say that the three phases—manic, depressive and lucid interval—may be combined in any possible way, and that further in a given case any degree of the manic or depressive phase may occur. It is common, too, to see during attacks of the manic phase transitory attacks of depression, while during the depressive phase it is equally common to see transitory periods of euphoria.

In a number of these cases the attacks reproduce themselves often at very definite intervals with practically photographic accuracy so that the patient leads a life the events of which can be predicted with almost absolute precision. Such patients not infrequently know some little time beforehand when an attack is coming on, and the physician may be able to see the approach of an attack the moment he steps into such a patient's room by a little difference in the arrangement of things that indicates the way matters are going.

It would seem that the patients who present such definite cycles occurring at stated intervals, each exactly like the others, belong to the group of cases with severe constitutional taint. In the other group of cases, that group in which external conditions seem to play a large part in the etiology of the several attacks, there is much less tendency to regularity in their occurrence, and as heretofore intimated, much greater hope for the results of therapeutics.

The following is the account of an intelligent woman of her feelings in both periods of excitement and depression:

"I have suffered all my life from excitements and depressions, although it was not until I was fifty-eight years of age that my family and I realized I was really mentally sick and required institutional

care. During youth and middle age my excitements were of a mild character, and during these periods I considered myself normal. I felt peculiarly happy and care-free. I managed my household affairs with the greatest care. I entertained and mingled in society with pleasure and zest. I was lively, talkative and I have reason to believe I was witty and entertaining. I could work without an effort. I at times accomplished almost Herculean tasks. On one occasion I remember preparing and conducting a church entertainment by which the sum of $800 was raised. Of late years my excitements have grown more severe. I begin by taking an overactive interest in everything going on around me. Everything seems rosy. I feel happy and nothing depresses me. I feel propelled by some unknown force to constant action. I am possessed with the idea of righting wrongs and straightening out things in general. All the faults in the administration of the ward, the hospital and the government must be corrected.

"My excitements have never led me to commit any acts of violence. I occupy myself largely in talking and writing letters. My room is often in disorder because I cannot stay at one job long enough to complete it. As I feel these excitements approaching, I request the physician in charge of me to take up my parole, as I know I shall be moved to do and say many foolish things of which I will be ashamed later. No one who has not had experience can realize the mortification of having been insane.

"My depressions in early life were as mild as my excitements, the onset was gradual. I felt a disinclination to mingle in society. When forced to do so I sat like a 'dummy' and could think of nothing to say. My household duties became a burden. One after another of these was dropped until the care of the household was entirely given over to relatives or servants. I learned from experience a treatment of my own. As soon as I felt a depression approaching, I promptly dropped everything and left home for a time. I found by getting away from family cares and responsibilities, and from the demands of society, to some quiet spot, I could shorten the duration of these depressions. In recent years the depressions have appeared suddenly. One day I went to town to do some shopping for a friend. I went to a grocery store to make some purchases. It suddenly occurred to me that I could make these to much better advantage at the market only a block away. Suddenly I realized that I did not have sufficient energy to go to the market and that another depression was upon me. It was with the greatest difficulty that I ordered the goods, paid for them and came home. At these times my brain feels paralyzed. I have not the strength or ambition to do anything. I am apprehensive lest some harm has befallen the members of my family, but to save my life, I could not write or telephone to find out if my fears are true. I have the impulse to act, but it seems as if something shuts down and prohibits action. I see my clothes becoming soiled —I know I should change them, but I cannot pull out the drawer of

my bureau and get clean ones. This inertia is greater in the morning than at night. Before I came to the hospital for treatment I had servants who slept at home and came to my house early in the morning. When my husband was away and my children were small, it devolved upon me to admit these servants early in the morning. I knew that when morning came to dress and go downstairs would be impossible. I solved the difficulty by dressing the night before and sleeping in my clothes. When the depression is most profound, I move in a fixed groove. I never vary a hair's breadth. At first I have a desire to remain in bed. Once this is overcome I have no choice but to remain up. I sit in the same seat and in the same attitude for weeks. As I come down stairs in the morning I am apprehensive lest my seat be taken, and I wonder what I shall do if it should be occupied, although the sitting room is well supplied with comfortable seats. I bring a shawl with me and place it in the chair so that no one will appropriate it while I am at breakfast.

"After each depression, I suffer from intense pain in my back, side, shoulders and arms. This is dull and aching in character and remains with me for weeks after the depression has disappeared. After the last depression I suffered from a severe attack of the shingles. The skin eruption has now disappeared but the pain still remains."

The Cyclothymias.—This group of cases presents the mildest excitements and depressions. They deviate less from the normal than the other groups and are only considered separately because of their great practical importance. They are quite usually not recognized and the symptoms are attributed to all sorts of things other than the real trouble. It must not be lost sight of in considering these mild manic-depressive fluctuations that a slight depression may recur without the psychosis expressing itself by a fluctuation to the opposite condition of excitement and *vice versa*. So that the picture is seen of patients presenting from time to time mild degrees of depression or mild degrees of excitement without anything approaching delusional formation or disorders of the sensorium and therefore attracting no particular attention from the mental side. The following example illustrates this exceedingly well: He is a man who devotes himself largely to literary work, and the fluctuations in his mental state are shown excellently well by his ability to write. The onset of a depressive phase is usually shown by a gradual, though more or less rapid, falling off in his literary ability. He is first unable to compose, then he gets progressively less able to write until he is only able to write the simplest things. It is the same way in his reading. He gravitates all the way from reading connected with his work down through the different grades of literature until he gets to fiction. He finally finds himself quite incapacitated, sitting for hours gazing out of the window or at a blank wall and while rather enjoying company, it is almost impossible for him to initiate the procedure that is necessary to go anywhere. He finds it almost impossible to dress, to get out, to take the cars and the like. This state is one almost entirely of retardation

without marked emotional depression. During the opposite condition of affairs he has a feeling of well-being and efficiency in marked contrast to his feelings during the depressive period and finds himself quite able to work for long intervals very effectively.

The remarkable transitions from phase to phase are shown well by one of his experiences. One day, having been writing all of the afternoon, he, as usual, went out to dinner, leaving his papers on the table, intending to resume work on his return. When he came back and took up the pen to write he found that the incubus of his depression was upon him. He had difficulty in finding words and finally after two hours' effort he gave it up. This was the beginning of a depression which lasted about a month. During this time he constantly tested his ability for composition but without favorable result. Almost exactly a month after this incident he undertook to answer some personal letters, intending to write only short letters of perhaps three or four pages, but when he started to write them he found himself writing easily and his letters spontaneously expanded to eight or twelve pages and he went on into his work again.

The *hyperthymic types* show exaggerated activities in the way of the usual business occupations, writing letters and the like. The judgment is apt to be rather poor at these times and many of the business ventures come to grief, though not necessarily so. Work is easily done, without having made effort, and the patient expends enormous amounts of energy over long periods of time. Certain types of cases are meddlesome and troublesome, tending to engage in disputes and altercations and to bring lawsuits, while exaggerated eroticism and alcoholic predilections oftentimes very considerably color the picture.

In the *dysthmic types* are found the depressions which are attributed in large part to neurasthenia and to various visceral disturbances.

These cyclothymic cases not infrequently show fluctuations at periods of recurrent physiological activity such as the menstrual period, while it must never be lost sight of that not a few so-called dipsomanias are really recurrent manic-depressive attacks in which the alcohol is resorted to shortly after the attack commences and then quite usually all the symptoms from which the patient suffers are attributed to the alcoholic indulgence. It is important to bear this class of cases in mind, not only for diagnostic purposes, but in order that the patient should be dealt with fairly as a sick man.

Perhaps the most important of the disturbances in this group of cases are the visceral disturbances. There are a large number of conditions, particularly the false gastropathies, enteropathies, cardiopathies of Dejerine, etc., many of which belong here. Inasmuch as the psychosis is not recognized, these conditions are quite naturally credited with being the cause of the condition of the patient. Patients with mild depression are called neurasthenic, those with mild excitement are called nervous and the accompanying physical condition is credited with making the trouble. The patient and the relatives

consistently take this attitude and the physician naturally falls into it. No one wishes to acknowledge the possibility of a mental disorder, and therefore these other explanations are readily accepted. In fact, the condition is hardly recognizable at its true value, even by the practised observer, unless a full account of the patient's life is available.

After a while the symptoms of visceral disturbance clear up along with the disappearance of the mental symptoms and the change is attributed to some form of treatment, a special dietary regimen, or whatever has been resorted to for the relief of the symptoms. Here is a group of cases who during their attacks regularly seek the specialist and are subjected to all sorts of gastro-intestinal treatment, gastric lavage, special dietaries, gynecological manipulations of one sort and another, metabolism experiments, endocrine therapy, treatment for autointoxication, eye-strain and almost everything in the category of medical specialism, and yet characteristically in these cases nothing is found in the physical condition that adequately accounts for the symptoms. Another group of these cases are the paranoid types. These patients present typical paranoid symptoms with emotional accompaniments that seem to be hardly in excess of what is demanded as normal reactions to the delusional state. This is the group of cases that has given origin to a great deal of recent discussion with regard to the real basis of paranoia, its relations to manic-depressive psychosis, the affective origin of paranoia and its basis in what Specht calls the "affect of suspiciousness."

With regard to the whole question of the relation of the manic-depressive psychosis and the paranoid symptom group Hübner[1] indicates as follows: (1) There are periodically appearing paranoid symptom complexes which may be compared to periodic mania; (2) with manic-depressive predisposition there may be pathological suspiciousness during the quiet periods while during the hyperthymias and the depressions there are marked persecutory ideas; (3) in addition to the manic-depressive symptoms chronic delusional ideas are formed. From true paranoia, however, these cases may be differentiated by the concurrent existence of unmistakable signs of circular psychosis and further by the fact that the delusions at times have the characteristics of phobias and doubts. (4) Finally are included those cases in which, on a cyclothymic foundation, a chronic hallucinosis gradually develops, but without dementia.

The Mixed States.—The mixed states are forms of manic-depressive psychosis in which the three cardinal symptoms of the manic and depressive phases are mixed so that the resulting state is neither one. They are: (1) *maniacal stupor,* (2) *agitated depression,* (3) *unproductive mania,* (4) *depressive mania,* (5) *depression with flight of ideas,* (6) *akinetic mania.* It will suffice to merely mention the symptoms of these groups.

[1] Weitere Beobachtungen über die manisch-depressive Anlage, Deutsch. Ver. f. Psychiatr. Jahresvers., Dresden, 1921.

Maniacal Stupor.—Emotional exaltation, decreased psychomotor activity, difficulty of thinking.

Agitated Depression.—Emotional depression, increased psychomotor activity, flight of ideas.

Unproductive Mania.—Emotional exaltation, increased psychomotor activity, difficulty of thinking.

Depressive Mania.—Emotional depression, difficulty of thinking, increased psychomotor activity.

Depression with Flight of Ideas.—Emotional depression, flight of ideas, decreased psychomotor activity.

Akinetic Mania.—Emotional exaltation, flight of ideas, decreased psychomotor activity.

Still the possibilities are not exhausted. It is quite uncommon to see any one of the conditions already described continue pure from the commencement to the end of the attack. In the manic phase symptoms of depression not infrequently crop up and occupy the field temporarily, while during the depressive phase it is quite as common to note transitory periods of excitement. Then it is quite common for manic attacks to be preceded by a longer or shorter attack of depression and sometimes such a period of depression follows, not infrequently but partial depression, of the type of unproductive mania. The depressive phase shows similar variations, more particularly it is followed by a short period of exaltation. Then, again, at any stage of the disease a mixed state may crop up for a time, so that we may see during the course of the manic phase psychomotor retardation occur or during the phase of depression emotional exaltation may develop, while in the various forms of the periodic psychoses it is quite the rule to find these mixed states at the transition places from one phase to the other, all of the symptoms of one phase not equally and contemporaneously graduating into their opposites. Thus during the course of a circular psychosis the affect may change from depression to exaltation before the psychomotor retardation has given place to increased psychomotor activity, thus producing a temporary mixed state.

Involution Melancholia.—The group of cases comprised under the term involution melancholia, which was originally used by Kraepelin, has now been pretty generally conceded to belong to the manic-depressive group. The characteristics of the disease are those of an anxious depression occurring in later life. The group is such a considerable one and of such practical importance, however, that it will be specially considered along with other depressions of later life in another chapter. (See Chapter XXIV.)

Pathology.—There are no specific pathological findings in this psychosis, although certain degenerative products have been described in cases of death from depressive stupor. Patients, however, characteristically recover from this condition, or if they die during attacks, the death is due to some intercurrent disease which itself would produce changes in the nervous system that would gradually cloud and

perhaps entirely obscure any pathology that the psychosis might have. A condition producing death itself must produce serious alterations of the central nervous system, that most sensitive of reacting portions of the human body.

Nature of Manic-depressive Psychosis.—This psychosis perhaps, as thoroughly as any other, has withstood throughout the years any attempt at understanding it, while as opposed to dementia precox the symptoms of which appear quite unpsychological, the symptoms of the manic-depressive psychosis in either one of its phases, more particularly perhaps in the depressive phase, are quite psychological, that is, quite understandable. The patients, to begin with, present largely average types of personality before the advent of the psychosis, and during the symptoms of the psychosis they ordinarily are not so far disordered in their conduct or in the character of their ideas as to place them, so to speak, in a class by themselves. They are still quite like the rest of us. The roots of the psychosis appear to spring more distinctly from the usual life, the fluctuations of the emotions being quite comparable to the fluctuations that occur in everyone.

However, it seemed quite impossible to understand how the patients could vary from one extreme to its diametrical opposite and what could possibly be the explanation of such shifts of position. For many years, under the domination of Meynert, the changes were supposed to depend upon changes of blood supply, upon anemias and hyperemias. When psychiatry, however, advanced beyond such crass types of explanation it was left practically without anything to fall back upon. Recently, however, the suggestive work of Bleuler[1] has seemed to indicate what may at bottom be the true explanation. He has demonstrated what he calls the *ambivalency* of ideas. This ambivalency gives, as he understands it, to the same idea two contrary feeling tones and invests the same thought simultaneously with a positive and a negative character. Along with this ambivalency there is an *ambitendency* which sets free with every tendency a counter-tendency. With this basal supposition it can be understood why the fluctuation of the manic-depressive is a fluctuation between conditions which are diametrically opposed. If each idea has associated with it by preference the idea which is absolutely its opposite, if each feeling has associated with it by preference the feeling which represents its exact antithesis, then there is reason for understanding how the manic-depressive gravitates between these two extremes. It is the *path of opposites* which is met with at every turn in psychiatric experience, and which after all is nothing more than a universal attribute of all reality from action and reaction in the realm of physics to ambivalence in the realm of psychology. All Nature shows these opposites.[2] Nothing suggests white more surely than does black,

[1] The Theory of Schizophrenic Negativism, Nervous and Mental Disease Monograph Series, No. 11.

[2] For a discussion of the biological significance of ambivalence in relation to psychiatry, see White, W. A., Foundations of Psychiatry, Nerv. and Ment. Dis., Monog. Se., No. 32.

nothing suggests love more readily than hate. The opposed ideas and feelings stand with relation to each other in the path of least resistance, and when one would go from a certain idea or a certain feeling in any direction he finds the path to the antithetical idea or feeling more easily passable than the path to any other goal. This fluctuation of the feelings along the path of opposites exhibits a close analogy to the mechanism of the compulsion neurosis considered as a fluctuation between love and hate. This explains some of the symptoms which can be seen, from this point of view, to be compromise formations. For example, one patient wills most of her property to her daughter (love) but so ties it up in a trust that she will never get it (hate). She has solved the problem of expressing both antithetical tendencies in the same act (the will).

Assuming the hypothesis of ambivalency and ambitendency, still what is the explanation of the affect fluctuations in this psychosis? Here as elsewhere in the mental field some fundamental psychic conflict undoubtedly has to be sought to which the patient is making efforts of adjustment. This is precisely the starting-point from which, for example, dementia precox has to be viewed. But here are individuals who present a different possibility of reaction, a different reaction type to the conflict than do the precox patients. This statement, of course, must not be taken as meaning any more than a mere putting into words of what is found, because it is not understood what the differences are that make different people react in different ways. Psychiatrists are only upon the verge of being able to ask such a question intelligently. They are not yet able fully to answer it. An indication of what is at the bottom of the manic-depressive reaction may perhaps be reached.

The manic-depressive psychosis may be conceived of as an effort at compromise and at defense, resulting from an endopsychic conflict. In the depressive phase the affect has broken through and invades consciousness, while in the manic phase the patient by feverish and restless activity, by a constant alertness, fights off every approach that might touch him on a painful point, that might reach a vulnerable spot. It would seem as if he were wildly beating about to keep off all intruders, not only real intruders, but all possible, prospective, or thought-of intruders. And so the manic patient is already quite inaccessible and all of his reactions are especially superficial, as witness the word associations and the clang associations. He moves over the surface, which he endeavors to cover completely, in order to prevent penetration at any point.

This constant activity of the manic, however, has another aspect than simply that of defense. In this constant activity of which such symptoms as flight of ideas, clang association and distractibility are types the patient is constantly occupied with reality. In fact he is so acutely interested in reality that little that occurs about him escapes him and he is constantly showing extremely keen powers of observation. This might be termed a *flight into reality* as a means of escap-

ing from the conflict and as such stamps the psychosis as belonging to the *extraverted* type as distinguished from the *introverted* type (of which precox is the best example) in which the libido turns back to reanimate channels in which it used to flow but which have long since been abandoned. Still this does not quite state the case. It has been found that, while the manic is headed toward and into reality, still if his productions be studied it will be evident that they are wish-fulfilments and that in his regressions he is reanimating longed-for situations.[1] The principle of ambivalency insures that the solution formulation of the conflict shall include both elements—the wished for as well as the tabooed.

The consideration of the manic-depressive reaction from the point of view of an extraversion type of psychosis is extremely helpful in affording a basis of explanation for many of the mooted points which have been raised in the recent literature regarding this much discussed psychosis. In fact it would appear that a great deal of the difficulty encountered by observers depends upon dealing with the psychoses solely at the descriptive rather than at the interpretative level. There is a good deal of effort being expended in trying to split up the whole group into smaller subdivisions and undoubtedly the future will show an increasing success in these efforts. Still it is already not difficult to see why, for example, one should find hysterical symptoms associated with the more purely manic since hysteria is also an extraversion psychosis as is well shown by the extremely strong phenomena of transfer exhibited by hysterics.

Of special importance, however, seems the relation of extraversion to the relatively benign nature of the attacks as compared with the relatively malignant character of the introversion in precox. The fact that the patient attempts to escape from his conflict by a flight into reality rather than by a path that leads, by introversion, through the conflict, as is so often the case in precox, seems to insure that he again and again is able to rehabilitate himself. The efficiency of one's relation to reality is the measure of one's normality and so, in this psychosis, the constant effort to plunge into reality, to become immersed in it, to deal with it at every point, becomes a saving grace and is largely accountable for the frequent recoveries.

Schilder, in a recent article, very interestingly attempts an explanation of the psychology of mania on the analogy of a "manic fluidum." He asks, may not persisting complexes open the reservoir of manic fluid? In the manic state unpleasant experiences surge up from memory. The reaction to these unpleasant experiences is a lively cheerfulness, the purpose of which is to overcome painful emotions. These experiences are not overcome but continue to exist in the mania inciting further liveliness, flight of ideas, etc. Every psychic conflict sets defense reactions in motion which strive to overcome the unpleasant experiences and derive pleasure from the victory, but that

[1] Dooley, Lucile: A Psychoanalytic Study of Manic-Depressive Psychosis, The Psychoanalytic Review, 1921, vol. 8.

this victory has also a further purpose, namely, to render the individual capable of new undertakings, to give him freedom of action. This combination of pleasure and action is what Schilder means by his manic fluid. Even in normal conditions there is this same fluid stored for meeting pressing problems. It can be set free by the disappearance of the problem, as, for instance, when the poor person becomes rich, or the mourner receives news that the person believed dead still lives. Or the problem may remain as a permanent source of production of the fluid, so that finally it pours forth from the reservoir and floods the problem; or the problem may be resolved in a delusional, wish-fulfilling idea which opens the reservoir. If the idea be clearly grasped that the existing condition of the reservoir of energy is the determining factor for the form of every expression of pleasure, but that the same may be essentially influenced by previous experiences, in the sense of having the energy intensified or weakened, then the possibility must be accepted, at least theoretically, that even when the reservoir is biologically normal a mania may result depending on previous experiences. Schilder is attempting a dynamic formulation. In the explanation of a disease he states, it is not sufficient to suppose that functions which are arranged in a soulless mosaic, are somehow excited or inhibited. The course and temporal development of the psychic processes must be taken into consideration. These processes are not of such a nature that a disease could effect selectively and exclusively any one of them.[1]

Of the psychoanalysts Abraham did more work on this psychosis than any other. He believed the repression here takes place at the oral-sadistic stage. (As prominent oral zone symptoms the refusal of food stands out. Probably taking poison and cutting the throat also belong here). The repression of sadism produces the self-reproach, depression, and anxiety. The depressed patient unable to love and forced to hate rationalizes it thus "They hate me because I am cursed with inborn defects—so I am unhappy, I am dejected." The stronger the sadistic impulses that are repressed the greater the feeling of guilt until the patients feel themselves responsible for all the sin and suffering in the world. The sadistic tendencies when they come to action instead of being released upon the external world are directed against the patient himself and he thus derives a masochistic pleasure from his own suffering.

The alternation of the manic and the depressive phases the *affect fluctuation* or the *diathetic proportion* as Kretschmer puts it have been likened to the work of the mythological Sisyphus: The laborious pushing of the stone up the hill is analogous to the work of the manic in warding off all approaches to his complex. The failure just as he is about to reach the summit, to succeed, is likened to the depressive phase with the failure of the work of defense and the consciousness of sin and the feeling of inferiority.

[1] Schilder, Paul: Vorstudien zu einer Psychologie der Manie, Zeitschr. f. d. ges. Neurol. u. Psychiatr., 1921, Bd. **68.**

In a recent publication[1] Freud offers the following hypothesis to explain the manic-depressive reaction: ". . . in cases of mania the ego and the ego ideal have fused together, so that the person, in a mood of triumph and self-satisfaction, disturbed by no self-criticism, can enjoy the abolition of his inhibitions, his feelings of consideration for others, and his self-reproaches. It is not so obvious, but nevertheless very probable, that the misery of the melancholic is the expression of a sharp conflict between the two faculties of his ego, a confiict in which the ideal, in an excess of sensitiveness, relentlessly exhibits its condemnation of the ego in delusions of inferiority and in self-depreciation."

Undoubtedly certain groups of symptoms in this great mass of material will be found to hang together with sufficient certainty and frequency to be classed as disease types but still the manic-depressive way of reaction may still be considered as such aside from such groups. When so considered it would seem that it constitutes a method of handling the conflict that belongs relatively high in the scheme of the psychic organization. In other words, the manic-depressive patient belongs to a relatively superior type of personality. This is seen by the comparative normality of the personality types involved, by the psychological, that is, understandable character of the symptoms which seem to be only exaggerations of normal reactions and also perhaps by such a fact as that brought out by Riebeth (see Etiology) that while manic-depressive psychosis is found in the ascendants of precox the reverse does not seem to occur.

The manic-depressive way of reaction would seem to be much nearer the normal, healthy way than the introverted, or the way of regression. The manic runs away, in part at least, from his wish, while the introvert yields to it. It would seem that the manic reaction does not lend itself to such severe regressions as the introverted type. The regressions may go a long way back but they tend to confine themselves to psychological material. There is not the same wealth or archaic material in the manic-depressive psychosis as there is in precox, nor is there such an evident quantity of organ libido fixation.

Still considering the manic-depressive reaction as an extraversion type one is prepared to find in individual cases that the symptoms do not necessarily run pure to type. It is understandable that in some cases there should be a mixture of introverted mechanisms and when this occurs it can be seen why such a symptom as negativism should be apt to appear because the symptom belongs to the catatonic type of precox which, like the manic reaction seeks to solve the conflict by running away from it, although in a different direction. The constant strain of seeking rehabilitation by a flight into reality causes a break and a resort to a simpler mechanism of escape. In the same way manic-like flights in certain cases of catatonic excitement may be expected. The manic episode evidences a specially great effort to

[1] Freud: Group Psychology and the Analysis of the Ego.

get back to reality which is only partially or briefly successful because making too great demands upon the type of personality involved.

The manic-depressive way of reacting may also be seen in paresis, arteriosclerosis and other organic conditions and also in certain psychopathic and degenerative types as in the prison psychosis, certain litigious paranoiacs and pseudologia phantastica. In all these cases the extreme activity, the constant occupation with reality and the generally exalted mood indicate an extraversion which must be further studied in its roots to know its real meaning.

The *physical conditions* have been dwelt on more particularly in discussing the cyclothymias because here the physical symptoms occupy the foreground. In the more severe psychotic disturbances physical symptoms if present may easily be overlooked. The anamnesis will show very frequently, in the prodromal period, such symptoms as headache, neuralgias, herpetiform eruptions, etc. It is not infrequent, however, to see marked evidences of biological maladjustment in the way of the general habitus of the opposite sex, evidences of endocrinopathies (glandular imbalances), with homosexual symbolizations at the psychological level. These physical accompaniments can be understood in accordance with the principles laid down in the introduction. Undoubtedly the finer studies of the future will uncover many more of them than are now known and indicate further their special meanings in the whole picture. A careful analysis of the visceral states and postural tensions accompanying these extreme emotional reactions will prove of the greatest importance to their adequate understanding.

Course and Prognosis.—The individual attacks vary in duration from a few days to several months. Recovery from the single attack is the rule, while the likelihood of subsequent attacks is considerable. In general, therefore, the prognosis is good for the separate attacks and is rather poor as to ultimate freedom from attacks. As pointed out previously, the severe constitutional types have a worse prognosis than those types in which the etiological factors are capable of removal. In the severe constitutional types also the recurrent attacks tend to repeat with photographic accuracy preceding attacks, while in the long run the general tendency is for an increase in the length of attacks and a decrease in the length of the free intervals.

The disease pursues its course without any special tendency to deterioration. Although mild grades of dementia have been described, terminating protracted attacks, the dementia which usually supervenes in the course of the disease is that which is superadded as the result of the changes incident to arteriosclerosis and the senium. Inasmuch as this psychosis tends to recur throughout life, not a few of the patients ultimately reach senescence.

Differential Diagnosis.—The manic phase in its mildest forms is often mistaken, especially where it leads to alcoholic and sexual excesses, for a form of moral obliquity. In the somewhat more pronounced attacks it may be difficult to differentiate it from other excitements,

more particularly catatonic excitement. In general the manic excitement is more free and open, there is less tendency to constraint along any particular line, while the productivity and the psychomotility are not as meaningless or non-understandable as with dementia precox.

The depressive phase in its milder manifestations is not infrequently mistaken for neurasthenia and in its more pronounced form it is extremely difficult to differentiate it from the depression of dementia precox. This is particularly so because the feeling of inadequacy of the manic-depressive is very close to the empty feelings with loss of affect of the precox, while the blocking of movement and expression in the latter condition outwardly closely resemble the retardation in manic-depressive psychosis. The stupor of catatonia outwardly also closely resembles the manic-depressive stupor, except that it is more apt to be associated with marked negativism, muscular tension and perhaps grimacing. (See Benign Stupor.)

The greatest difficulty, as between manic-depressive psychosis and dementia precox, lies with the differentiation of the mixed states. Here the resemblances are quite close and prolonged observation often necessary to make the differentiation.

It must not be forgotten that the manic-depressive psychosis is by no means a clear-cut definite entity, that it merges in all directions into other conditions, and that its closest affiliation with the other psychoses appears on the surface to be with the dementia precox group. There are quite a considerable number of cases in which a study of the individual attack leaves one in doubt as to which group to place the patient in, manic excitement, for example, being associated with certain catatoniform symptoms, catatonic excitement presenting a fairly typical flight of ideas and the like. In general the principle of differentiation is first, the history of previous attacks, and next to this history of repeated attacks is a history of attacks of both manic and depressive character occurring in the individual and showing no marked tendency to deterioration. Practically, however, it is quite impossible to make a differentiation in many cases and patients that are at one time placed in one group, for example, in the manic-depressive group, are found later to probably belong in the precox group, because, for example, of the appearance of deterioration. And so the history of the diagnosis in these two groups shows a constant tendency to fluctuation, at one time the dementia precox group being enhanced by cases which at another time are placed in the manic-depressive group. And so the situation fluctuates back and forth, the best evidence of an inherent relationship between the two. The reasons for this state of affairs have been set forth in the section on the nature of the psychosis while a consideration of the extraversion features of the reaction as against the introverted features of the precox type will materially aid in the understanding of the meanings of the symptoms.

The whole question of the differentiation of some cases of manic-depressive psychosis from dementia precox involves the greatest

nicety of observation, interpretation, and judgment. Lange,[1] in a study of 100 carefully selected cases of manic excitement, states that the number of cases of mania entirely free from catatonic symptoms is remarkably small, although he extends the meaning of catatonia to include, besides the psychomotor symptoms, the absence of affectivity, the schizophrenic disturbance of association and hallucinations. The catatonic phenomena are especially frequent in youth and advanced age. Symptoms suspiciously schizophrenic, in the form of leveled affects, monotony, and stereotypies, paranoid elaborations, excitements, disorders of the sensorium, and delirious conditions, are especially frequent in advanced age. The schizophrenic symptoms always made their appearance in conditions of clouding of consciousness and hence are attributed to this condition. When, for example, there is clouding of consciousness in symptomatic psychoses, catatonic psychomotor symptoms are more frequent. With clear consciousness the catatonic features constitute a very unfavorable sign. Clouding of consciousness is given a very broad meaning by the author, including many heterogeneous elements (sleep, dreams, dream states, "peculiar stupefactions," narrowing of the field of consciousness).

The most important group from the practical standpoint is the cyclothymic. These patients are practically always wrongly diagnosed at first and often over and over again for considerable periods of time. Most generally, as already mentioned, they fall into the hands of the specialist under the theory of some disorder of the internal organs. When an attempt is made to really understand these patients one is impressed with their close resemblance to the obsessional neurosis. It may be quite impossible, at least by any means other than a very careful and detailed study of the patient, to differentiate between the two conditions and from the discussion on the nature of the manic-depressive psychosis it will be seen why this similarity exists. It should be borne in mind also that it is not difficult to confuse mild cyclothymic attacks with anxiety neurosis.

The relation to compulsion neurosis has already been mentioned in discussing the nature of the psychosis.

It should, of course, be added that one must be careful and not confuse excitements and depressions that may have other origins as, for example, particularly paresis, the symptomatic and toxic psychoses and the more clearly psychogenic states such as prison psychosis—the so-called situation psychoses.

The whole discussion of differential diagnosis is of course, a sterile performance unless it is kept constantly in mind that the diseases considered are only temporary formulations and that the vital factor is really the method of reaction which may exhibit itself at any level, chemical, physiological, psychic, or social, and be associated with a definite type of machine, bodily make-up, which may be found in the

[1] Katatonische Erscheinungen im Rahmen manischer Erkrankungen. Monogr. a. d. Gesamtgeb. d. Neurol. u. Psychiatrie. Hrsg. v. O. Foerster und K. Wilmanns, H. 31. Berlin, Julius Springer, 1922.

ascendents and descendents. The bodily type which is related to the various endocrine constellations has raised anew the whole question of types and on a new, a dynamic, basis.

Extra-neural Pathology.—Postmortem examinations of those who have presented the manic-depressive reaction type frequently revealed various associated pathological conditions of the nervous system and of the other organs, but in the past the field of somatic pathology has failed to contribute to the knowledge of the organism-as-a-whole when involved in the manic-depressive type of reaction. This failure is due both to the lack of systematic investigations in this field and to the fact that the anatomico-pathological material is difficult to interpret in this connection, since the psychosis is so often a prolonged periodic process extending over many years; thus the original tissue organization may be altered by senility and intercurrent disease, as well as by the lethal lesion.

Owing to the prolonged and periodic character of the condition, suitable material available for postmortem study is greatly limited; however, a number of clinically certain cases have been thoroughly studied, and from these one may attempt to drawn some general conclusions as to the somatic construction of the manic-depressive type of individual. Lewis[1] has found:

1. It is a well-known clinical observation that these patients are very prone to develop surface infections, particularly during the excited phases. Microscopic studies on the various tissues have emphasized the fact that numerous infections have occurred in the internal organs of the body during the life of the individual. Small contracted healed areas are found far more frequently than in the bodies of those affected by any other type of psychosis, and although they are not limited to any particular type of tissue, are found more often in the thyroid, liver, lungs, kidneys, and pancreas.

This finding would indicate that the manic-depressive type of individual is not only more prone to infections (lowered local resistance) but at the same time demonstrates tremendous reactive possibilities (powerful combined or total reaction), since the organism-as-a-whole walls off and destroys the invaders and heals up the disorganized tissue. However, the final outcome of 40 per cent. of manic-depressive constitutions is death from some form of acute septicemia, such as that from pyelonephritis, acute suppurative cholecystitis, and streptococcic septicemia, while only 10 per cent. of cases studied by Lewis died of pulmonary tuberculosis.

2. The circulatory system in general is large and compensates easily for extra strain; however, death from circulatory accident, such as cerebral thrombosis, subdural hematomas, cerebral hemorrhages, and cardiac decompensation, occurs in about 50 per cent. of cases.

In this respect the soma of the manic-depressive shows many features in common with the paranoid types (see p. 985), which is to

[1] St. Elizabeths Hospital material.

be expected, since powerful compensatory psychologic features dominate the clinical picture of both disorders.

Generalized arteriosclerosis with associated tendencies to infarction of various organs, kidney disorders, venous thromboses, and miliary hemorrhages, is more often encountered in the aged manic-depressive than in the average senile.

3. The endocrine glands exhibit peculiar combinations of atrophy, hypertrophy, and hyperplasia.

In a number of cases the pituitary has been found grossly atrophied, occasionally to an extreme degree. Microscopic examination of these glands discloses a tendency on the part of individual cell groups to compensate by hyperplasia, and in a few instances typical local adenomata have developed in the anterior lobe. The posterior lobe is usually in a state of fibrous atrophy.

The thyroids exhibit the scars (frequently calcified) of numerous previous infections, which have probably developed secondarily from throat or tonsillar infection, etc. In some cases there is fibrous atrophy of the gland as a whole, but more often there are attempts on the part of the epithelial cells to reconstruct the parenchyma, and thus secondary hypertrophies arise. Miliary hemorrhages may be numerous through the interacinal stroma.

The adrenals generally exhibit cortical hyperplasias, but not to the remarkable degree noted in the paranoid types; however, the tendency to general atrophy of both cortex and medulla, and moderate local attempts to reconstruct on the part of the cortex, are constant findings. Miliary hemorrhages into the substance of the adrenals are often found in addition to the other developments.

The gonads from the standpoint of the group are very well preserved; even in senile cases there may be less atrophy than expected; however, the testes of an occasional case may show some diffuse atrophy of both tubules and interstitial cells and reduction in the spermatogenic functions, while in other instances complete focal destructions have occurred either from vascular involvement or previous infectious invasions.

Treatment.—There has been the general feeling in years past about this psychosis that the attacks were self-limited. This has probably been dependent to no small extent at least upon the extreme regularity of the attacks in certain patients. These patients, however, are the patients with the more profound constitutional taints and it is perhaps generally true that in this class of patients attacks do tend to run a regular course, each attack being approximately of the same duration as the former similar one. With the more frankly reactive types, however, this becomes progressively less true, so that the matter of treatment here easily becomes something more than mere intelligent custodial care.

In the very mild types of the disease the patients have to be carefully safeguarded, because their condition is not appreciated by others. In the excited phases alcoholic and sexual excesses are espe-

cially apt to occur, and it must never be lost sight of that during what outwardly appear to be the mildest depressions suicide is a possibility.

In the more pronounced attacks the handling of the patient calls for the very greatest amount of tact. During the excitement the patient's strength must be guarded, as insomnia is a constant symptom and food may be taken in insufficient quantities. Mechanical restraint should, of course, be avoided if possible and it may be said that it is practically never necessary. Its application in the high degrees of excitement is often not understood by the patient, and produces an increase of excitement and resistance and perhaps a state of anxious apprehension and even terror. Chemical restraint is equally undesirable as it tends to shut out the real world and thereby increase the difficulty of adjustment to reality. Hypnotics may be necessary and such simple ones as veronal are the best—paraldehyde, trional, sulphonal, chloralamid are useful but opium and its derivatives should be avoided if possible. Whereas the various kinds of restraint are highly undesirable, isolation may be resorted to and is not infrequently welcomed by the patient. All that may be necessary is simply to put the patient in a room by himself without locking the door. He may be very glad to stay there and so escape from outside sources of irritation.

It is in the condition of excitement that the continuous bath is so valuable. The patient is placed in a tub, preferably one constructed especially for the purpose, being long enough to permit the body to lie in it without the limbs being flexed. The water, which should cover the body completely, is kept at a temperature of from 96° to 98° F., that is, just above the normal surface temperature. It adds to the comfort of the patient if a canvas hammock can be slung in the tub on which he can lie and a rubber air pillow be placed under his head. The warm water of the continuous bath is the best sedative treatment we have for this class of patients, as it produces sedation without any clouding of consciousness or other disagreeable features. The patient may be left in the tub for such a period of time as is deemed necessary, usually three or four hours at a time. On the Continent patients are not infrequently kept in the tub not only for days, but even for months, sleeping in the tub and being fed in it. On the whole the patients enjoy this, the warm water is soothing and they are grateful for its calming influence.

It is in the excited phases that the nurse's ingenuity will be taxed to the utmost and if she is not tactful all sorts of artificial symptoms will be created in the way of antagonisms toward the nurse, increased irritability, etc., so that the adjustment of the nurse to the patient, particularly in excited conditions, becomes an important practical problem.

In the depressed phase of the disease the patient is often best treated in bed, particularly if the depression approaches the stuporous stage. Under these circumstances refusal of food is a common symptom and tube feeding must be resorted to at regular intervals. With the tube

feeding it is easy to give such medicines as may be required, either hypnotics, cathartics or anything else indicated.

One must remember that in endeavoring to probe the consciousness of depressed patients the depression may be increased and if suicidal tendencies are present this should be borne in mind and guarded against.

If the patients are stuporous the usual precaution should be taken to see that the position of the body is changed from time to time and that the bladder and rectum are not permitted to become overloaded. The danger of suicide in depressed conditions is an ever-present one. The only safe way to deal with these patients is to assume that they are all suicidal. Probably one of the reasons why more patients in this condition do not commit suicide is because of the marked retardation which makes it so difficult for them to initiate any form of activity. It is the depressed patients who are suicidal who most frequently require to be sent to a hospital, although their general condition may not seem to warrant such a move. The watching and the caring for depressed patients with a view to preventing them from committing suicide is practically only understood in institutions for the treatment of mental disease. The general hospital nurse, the general practitioner and the family rarely have any idea of the degree of watchfulness that is necessary and for this reason alone oftentimes the patients must be sent to institutions.

The active excitements may be associated with hyperthyroidism which then might advantageously receive appropriate treatment (roentgen ray, belladonna, iron, etc.).

During the period of convalescence, occupation, outdoor exercise and the like are all in order. Care should be taken not to force the patient too fast.

Here as elsewhere in mental medicine an attempt should be made to analyze the mind sufficiently at least to understand the nature of the disturbing factors that are at work, and if possible the way in which they have brought about the psychosis. This, of course, is essential to an intelligent treatment of the patient. Such analysis, however, is almost impossible with many patients, particularly during the attack, and can only be resorted to when the patient is at least approaching the normal condition. All the information gained, however, is valuable as pointing the way toward regulating the patient's life and in many instances as indicating the nature of the etiological factors and thereby showing what must be avoided in the future if further attacks are to be prevented. Of course much more is to be hoped for in the frank reactive than in the profoundly constitutional types. An adequate psychoanalysis, however, affords the best prospects for ultimate recovery, perhaps furthered by an endocrine therapy calculated to relieve the glandular imbalances which may be operating to prevent a synthesis of the personality at a sufficiently high level of efficiency. Psychoanalysis should, however, be used with great care as the uncovering of the complexes may have the initial effect of plunging the patient

into a profound depression in which suicide should always be considered as a possibility. Such patients should, therefore, be in especially protected situations during such a crisis. The best time to undertake an analysis is during the interval between attacks. During the mild hypomanic period the patient is inclined to be communicative and the repressions are not so apparent, but the defenses are really very active. During the deep depression the patient is inaccessible but when recovering from the depression he may be willing actively to coöperate as at this time he especially wants to get well.

The psychoanalysis of the manic-depressive, has, however, not been very successful. One seems to proceed famously but at the end is astounded at the paucity of results. It is, only too often, like writing in the sand.

Prophylaxis.—Prophylaxis resolves itself into two parts: (1) the prevention of the disease, and (2) the prevention of subsequent attacks after the disease has manifested itself. The prevention of the disease is perhaps a problem of eugenics, but of equal or greater importance a problem in education; the adequate unfolding of the developing personality in relation to its problems. Very much more information is needed as to the way in which the manic-depressive psychosis conducts itself with reference to the laws of heredity. It has not as yet been adequately worked out so that definite advice is possible.

As to the prevention of subsequent attacks the most important thing is to prevent if possible the recurrence of the etiological factors that have been found, by the psychoanalytic study of the patient, to play a part and the reëducation and reorientation of the patient toward his problems of adaptation.

CHAPTER XVII.

THE PARANOIA GROUP.

PERHAPS no term in psychiatry has undergone wider variations of meaning than the term paranoia. In its earliest days, in the Greek period, it meant little more than just craziness, although perhaps it may have been used somewhat more specifically in some instances, and later on, well into the Middle Ages, it was still a term that was not infrequently used to include the whole group of the so-called insanities.

The term[1] did not come into general use as applied to a special grouping of mental symptoms until the early part of the nineteenth century, when a German psychiatrist, Heinroth, made an effort to classify various mental diseases and gave paranoia a distinct placement in his scheme. The classifications of this day, however, were extremely complex and there was a marked tendency to follow the biological method with its binomial nomenclature, which had come into such popular vogue with the appearance of the work of the great Swedish botanist, Linnaeus, in the middle of the eighteenth century. A disease that was classified under the specific names of hallucinatory, confusional, depressed, or what not, might change its name and its nature overnight, as it were. This led to great confusion and to the final revision of the whole scheme by the French, under the leadership of Pinel, who made a new synthesis of a few elemental forms—the manias, melancholias, and dementias. Esquirol followed with his monomania, under which the paranoias found a place, and this term has been in use ever since, largely by the English school, and it still finds application in the courts. It is based upon the simplistic conception that the brain is one organ and that it has one disease, and that disease is insanity, and not only simplistic to this extent, but that the disease may affect any part of the organ and therefore a person may be insane upon one subject, conceptions which are hardly worthy of a school boy, but yet are still held in some quarters today.

From this time on the general concept of paranoia became somewhat more definite and it tended more and more to concentrate and crystallize itself about a condition which presented essentially delusions, more or less clearly formed and of a persecutory type, generally associated with hallucinations, especially auditory. Even this concept, however, included such a mass of material of such dissimilar types that it never became very well defined. The paranoia contro-

[1] Jelliffe: Study of the Origin, Transformations and Present Day Trends of the Paranoia Concept, Jour. Nerv. and Ment. Dis., 1913.

versy during this period, namely from the middle to the end of the nineteenth century, is largely taken up with a discussion upon the basis of the old faculty psychology, and the division of the mind into three parts, the intellect, the emotions, and the will. There had already been a tendency to consider paranoia as a primary intellectual disorder. The discussion took up the question as to whether the emotions were involved primarily, secondarily, or at all. This whole bootless procedure, based upon the faculty psychology, of course came to naught, simply because the mind is not split up into mutually exclusive compartments. Another one of the concepts upon which such discussions were based, and which was assumed in the discussion, was that there were such things as mental disease entities which had as much individuality and definiteness in the conception of the psychiatrist as tumors had in the mind of the pathologist.

Without going into a description of the different ideas of paranoia which have been extant, and which in their later development will be described in the body of the chapter, it need only be added that the general result of all this discussion is first that the brain is not a single organ. It is a great number of organs crowded into a very close space and the functions of its different parts need be no more closely related to one another than the functions of the adrenal glands and the hypophysis. The cortex alone consists of at least fifty histologically differentiated organs, while the thalamus is composed of at least nine ganglia. The red nucleus is an organ by itself, as are the different portions of the lenticular nucleus, and the separate ganglia innervating the ocular muscles. In some way or other there issues from this complex of organs, or more properly is associated with it, the phenomenon of mind. Mind is not a single thing any more than is the brain. It is not only as complex as the organ which subserves its function, but infinitely more complex than this organ as it is known today. The mind cannot be conceived as divided into compartments like the will, the intellect, and the feelings, each presided over by a mythological demon, so to speak, but must be conceived of as a complex of adaptive mechanisms interrelated with one another in the most intricate manner, so that the mind must be conceived of as capable of having not only one, but many kinds of disorders, which disorders are not entities in the sense of foreign bodies or diseases which enter from outside, but are inefficient ways of functioning, special combinations of mechanisms, and so there are not so many mental diseases after all as types of mental reaction. The disease is not, therefore, something which comes from without, but it is a function of the interrelation between the individual and his environment, and only in proportion as this interrelation is inefficient may it be conceived of as disease, and only in accordance with the type of mechanism which is utilized, the special trend of reaction, can a disease be spoken of in any specific sense at all. This is quite parallel with the concepts on the physical side and is important to bear in mind if one is not to be enslaved by a limiting terminology.

The most recent advances, therefore, in the concept of paranoia are, a getting away from the consideration of it as a disease entity, or as involving a special faculty of the mind, or as a merely monosymptomatic classification, and a coming to consider it as a type of reaction which manifests itself in certain individuals. The descriptive attitude toward the problem is being replaced by the interpretative. This is significant, as paranoia has long been the stronghold for descriptive psychiatry and it has been the last to yield to anything like an interpretative approach.

Description.—The general concept of paranoia which has been prevalent for many years is that of a psychosis presenting delusions of persecution of a pretty clearly defined type, well supported and defended by the patient, in other words, systematized. These delusions generally involve a more or less circumscribed portion of the mentality, although they tend to spread out slowly and involve more and more. With this state of mind there is no marked tendency toward deterioration, the disease having essentially a chronic course and remaining unchanged for years. Associated with the delusions and harmonized with them in content are frequently auditory hallucinations—voices.

This is the general concept of the disease which has received various modifications and descriptive clothings by different authors. For many years the *délire chronique a évolution systématique* of Magnan in France and the paranoia of Krafft-Ebing in Germany have been the paradigma under which the various forms have been arranged. The délire chronique of Magnan was a disease which progressed regularly through four stages: first, a hypochrondriacal stage, or stage of subjective analysis; second, a stage of persecution; third, a stage of transformation of the personality; and sometimes, fourth, a stage of deterioration.

In the first or the hypochondriacal stage, or stage of subjective analysis, the patient is self-centered and depressed and has ideas of reference. He also complains of many physical symptoms, such as dizziness, weakness, headaches, etc. Everything that happens about him tends to be referred to himself, so that he is in a constant state of morbid introspection about things which he does not understand. In the second stage the explanation of all these things finds itself in the delusions of persecution. The reasons why people have slighted him, why they have said disagreeable things about him, why they talk about him and spread rumors about him is all understandable because of the conspiracy which there is against him. These delusional ideas are reënforced by hallucinations of hearing, and he hears actual evidences of all of the things which are being done to annoy, to persecute, or to destroy him. Explanatory delusions follow which give the reasons to the patient why he is thus persecuted, and usually attribute the persecution to some special person or group of persons, or some society or institution. In this persecutory period the patient may be very dangerous. He may react upon his persecutors if he knows who they are, that is, if he has defined them in his own mind.

On the other hand, he may develop elaborate defenses against them or may endeavor to flee from them entirely. Later on, as a result of the elaboration of the whole system with the effort at finding further explanations for them all, there are developed the ideas of self-importance. The patient concludes that if so many people are leagued against him that he must indeed be an important personage. When this occurs the third phase of the disorder, namely, the transformation of the personality, has developed. By this time the delusional system has become extremely complex, with interminable ramifications in all directions involving all sorts of occurrences, all manner of people, and reaching backward into the past and finding delusional explanations of events even in the patient's childhood. These explanations often refer to facts which antedate the period of the psychosis, and are therefore known as retrospective falsifications. Following the transformation of the personality there may occur a certain amount of mental enfeeblement.

This, in a few words, is Magnan's délire chronique, a disease which is extremely rare if this gradual evolution of the several stages described by him is looked for; that it does occur from time to time cannot be disputed. In all probability a number of cases of paranoid dementia precox were included in this group, while today the group has been recognized in a much restricted sense by Kraepelin[1] in his paraphrenias.

Krafft-Ebing, in Germany, developed a picture of the disease which for many years had much vogue in this country, and found particular favor, largely, probably because of its easy applicability. In the first place he divided all paranoias into two great groups, the *original* and the *acquired.* The original paranoias, after Sander, were those which presented their psychosis as the natural unfolding of an abnormal character, while the acquired or *late paranoias* were the paranoias which developed in people who up to a certain point had appeared normal. A further subdivision of these groups was into *paranoia hallucinatoria,* in which hallucinations played a prominent part, and *paranoia combinatoria,* in which they were absent or of minor importance. Following these great groups there were secondary groups which stood upon a purely symptomatic basis, and so he described *persecutory paranoia, expansive paranoia, querulous* or *litigious paranoia,* and *inventive, reformatory, religious,* and *erotic types.*

More recently there has been a tendency to gradually restrict the paranoia group to narrower and narrower limits. One of the most recent attempts at more clear definition is that of the French authors Sérieux and Capgras,[2] who have described two types of psychosis to which alone they think the term paranoia applicable, namely, the *delirium*[3] *of interpretation* and the *delirium of revindication.* In the

[1] Psychiatrie, 8te Auflage.

[2] Les folies raisonnantes, Paris, 1909; and Diagnostic du délire d'interpretation, Revue de Psychiatrie, January, 1908.

[3] The word delirium is here used in the sense given it by the French, namely, to apply to the sum of the patient's delusional experiences.

delirium of interpretation the patient has ideas of reference, and because of his lack of critique and egocentricity comes to all sorts of false interpretations of what is going on about him. These delusional interpretations become systematized and reach more or less coherency without any special dependence upon disorders of the sensorium. There is no deterioration, and lucidity is maintained throughout the evolution of the psychosis. Unlike certain other paranoid conditions the false interpretations have their origin in actual facts.

In the *delirium of revindication* a chronic systematized psychosis, which takes its origin in a fixed idea, appears. It is a monoideism, and its various ramifications, like the other form of paranoia, do not tend toward dementia. They describe two varieties of this psychosis, the *egocentric type,* the subjects of which are usually persecutors making claims for wrongs suffered that may or may not have some foundation in fact. Then there is the *altruistic type,* characterized by abstractions and impersonal theories. To this group belong the inventors, the reformers, and the prophets, becoming, however, in their endeavor to realize their ideals, oftentimes dangerous fanatics, mystics, anarchists, regicides.

In Germany Kraepelin limited the paranoia concept perhaps more than anyone else. He confined the term to a very circumscribed and very small group. His conception of the disease is a chronic incurable psychosis of insidious origin developing slowly by the gradual systematizing of endogenous delusions. This system of delusions is enduring and unshakable and exists along with the retention of the logical and orderly process of thinking. There is no marked tendency to mental deterioration, and hallucinations play no essential part in the picture.

Kraepelin has recently, in the eighth edition of his work, still more clearly defined his paranoid group by describing a group, *paraphrenia,* which contains certain paranoid types that closely resemble his paranoia, but which provisionally he includes in this group for purposes of greater definition. This group of paraphrenia is divided into four subgroups, as follows:

Paraphrenia systematica, which is for the most part Magnan's délire chronique, with the exception that the well-defined precox types with marked deteriorations are excluded.

Paraphrenia expansiva.—This form affects only women and is marked by the development of ideas of grandeur with mild excitement and exaltation. No dementia follows.

Paraphrenia confabulans.—Here the delusions both of persecution and grandeur are specially marked by their foundation upon and reference to memory falsification, as the name indicates.

Paraphrenia phantastica is the term applied to cases with a certain amount of exaltation, with the recounting of remarkable adventures and incoherent changing delusions. This condition is characteristically accompanied by hallucinations of hearing. This group includes the cases previously described by him under the term dementia paranoides.

Mayer[1] has recently followed up the destinies of the paraphrenia material of the Munich Clinic much as Dreyfus did the cases of melancholia from the Heidelburg Clinic. It was possible to observe 78 cases of paraphrenia for a considerable time. These cases were grouped as follows: 45 cases of paraphrenia systematica; 13 cases of paraphrenia expansiva; 11 cases of paraphrenia confabulans; and 9 cases of paraphenia phantastica. The course of the 9 cases of paraphrenia phantastica was such that the whole group without exception had to be credited to the group of dementia paranoides. Of the 13 cases of paraphrenia expansiva the further development showed that 5 were unmistakable schizophrenics; 1 case was not clear; 1 case was organic brain disease; 1 case belonged to the manic-depressive group; the 5 remaining cases occupied a unique position. Of the 11 cases of paraphrenia confabulans 5 changed more or less acutely in the direction of schizophrenia; the other 6 continued their course as paraphrenia in the sense of Kraepelin; 1 case terminated in recovery. Of the 45 cases of paraphrenia systematica the course of 11 showed them to be unmistakably schizophrenia; 2 cases had to be considered as paranoia; 3 were considered as abortive paranoia; 1 a paranoid psychopath with schizoid reaction; another had to be considered, from the course, as a paranoid psychopath; 3 turned out to be circular psychoses; 2 cases developed into organic senile psychoses; 2 others were not clear; the course in 17 remaining cases was in keeping with Kraepelin's pictures. The beginning of these psychoses was in nearly all cases about the fiftieth year.

Interpretation.—The rather simplistic attitude which dominated shortly after the term paranoia came into general use and which saw in every combination of fairly well defined and fixed persecutory ideas, especially those supported by hallucinations of hearing, the disease paranoia had to give way in a very few years to a broader, if somewhat less well defined, attitude. It soon became evident that a fairly systematized and fixed delusional system of persecutory character might occur as the expression of a psychosis from which recovery took place. And so the element of the concept of paranoia which considered it as essentially chronic, progressive, and incurable had to be readjusted. These so-called acute paranoias have been studied quite extensively by Friedmann,[2] and their origin traced to actual situations in the patient's life, so that the delusions appear as logical outgrowths of experience, and have as a consequence fallen into the group of the psychogenic psychoses.

Not only was the idea of chronicity associated with paranoia seriously shaken, but from other sources the idea of the specificity of the persecutory delusion also had to give way, for it was soon found that ideas of persecution of paranoiac character were not at all infrequent

[1] Ueber paraphrene Psychosen., Deutsch. Forschungsanstalt f. Psychiatr., München, May 12, 1921.

[2] Contributions to the Study of Paranoia, in Studies in Paranoia, Nervous and Mental Disease Monograph Series, No. 2.

in connection with other psychoses. This was particularly evident in the psychoses of chronic alcoholism. It soon developed that there was a special form of dementia precox presenting paranoid ideas, while later studies showed luetic forms with paranoid symptomatology, presenile forms, paranoid states of mind of the deaf, and others who are isolated from close contact with the world, to say nothing of the paraphrenia group of Kraepelin and in many other less well defined conditions, which have included more recently not only the manic-depressive psychosis, but certain of the milder cyclothymic manifestations of this disorder.

From these considerations it appears that here, as elsewhere in the field of psychiatry, the important thing to consider is not so much the special content of the particular psychosis in a given individual as the mechanisms which are involved, for here is seen a similar content in all sorts of mental disorders, some acute, some chronic, and we are therefore forced to look beneath and see whether it is not possible to understand these manifestations by attributing them to a common mechanism.

The studies of Friedmann, already mentioned, went a long way toward showing the dependence of paranoid trends upon actual situations in the patient's life, and demonstrated how paranoid delusions in given cases might grow as a result from these situations. In other words, they are of psychogenic origin and are perfectly understandable when all of the circumstances have been uncovered.

Among others, Gierlich[1] has shown that paranoid ideas often accompany fluctuations of affect which could only be considered as manifestations of a manic-depressive psychosis and that many of the paranoid conditions which were associated with only slight affect manifestations which belonged to the manic-depressive psychosis might easily be overlooked as coming under that group and be mistaken for true paranoia. In this way he accounted for a very large number at least of the so-called acute paranoias, as these patients of course got well from the attacks as the manic-depressive cases usually do.

More recently Specht[2] has at great length endeavored to demonstrate that the underlying condition in paranoia was an affect of suspiciousness and therefore he brought the paranoid group into close alliance with the great affect group of the psychoses, namely, the manic-depressive group. This whole discussion has broadened out in all directions and has become very complex and extremely involved, and therefore it is not a proper subject for further elaboration in a text-book. It might be added, however, that Bleuler,[3] who has made a most incisive study of the psychology of paranoia, denies absolutely

[1] Periodic Paranoia and the Origin of Paranoid Delusions, in Studies in Paranoia, Nervous and Mental Disease Monograph Series, No. 2.

[2] Ueber den pathologischen Affekt in der chronischen Paranoia. Cited by Bleuler, in Affectivity, Suggestibility, Paranoia, New York State Hospitals Bulletin, vol. 4.

[3] Affectivity, Suggestibility, Paranoia, New York State Hospitals Bulletin, vol. 4; February 15, 1912.

that suspiciousness is an affect at all, and therefore departs radically from Specht's position. He believes suspiciousness is a state of mind based entirely upon perceptions and the resulting conclusions, and is therefore of purely intellectual origin, but that it is accompanied by affect, as are all mental states. Here, again, the fallaciousness of the old faculty psychology that would separate the mind into different parts, such as the intellect and the emotions, should be emphasized. The two invariably occur together, and suspiciousness, therefore is, of course, accompanied by affect.

Bleuler is of the opinion that paranoia takes its origin in certain constellations of ideas or complexes and the dominant affect with which they are loaded, that these complexes are precisely of the same nature as are found in healthy individuals, and that the disease element which leads to the elaboration of a psychosis is the fixation upon this complex, the inability to get away from it, or as might be said, the inability to reach an efficient adjustment to it.

Kretschmer's[1] recent very elaborate attempt at an interpretation of the paranoia problem should be mentioned here. He endeavors to separate out a special pathological reaction type. These peculiar, sensitive types, in which the emotions are dammed up and there is a lively intrapsychic activity, but no adequate outlet, belong to those tender natures which are expecially impressionable but elaborate their experiences in a quiet repressed manner without external manifestations. This sensitive type is related in one direction to the compulsion neurosis and in the other direction to the neurosis or psychosis of reference. This sensitive type Kretschmer believes constitutes a disease entity with a definite etiology, symptomatology and course. He believes it to arise upon a profoundly hereditary taint, and to be biologically characterized by fatigue from work and affects. The ideas and affects are exclusively centered on the pathogenic experience and the symptoms of the psychosis represent an exaggerated expression of the peculiarities of the sensitive character. The disease picture may be colored with neurasthenic symptoms of exhaustion. The course is on the whole benign. The sensitive psychosis of reference assumes various forms. Kretschmer describes systematized paranoid pictures, acute sensitive delusional psychosis, habitual neurosis of reference, saltatory delusional formation after the manner of the compulsive neurosis. The normal type of sensitive psychosis of reference is systematized with the preservation of formal logic and the intellectual faculties generally. Kretschmer presents a scheme for the solution of the paranoia problem that shows his sensitive group at one end and the expansive group at the other with various paranoid pictures in between. It is needless to say that interesting and perhaps important as such efforts and schemes may be, they fail to grasp the dynamic point of view, they stay at the level of descriptions.

One here sees what is everywhere apparent in dealing with mental

[1] Der Sensitive Beziehungswahn, Berlin, 1918.

disorders, that the delusion is not the disease, the delusion is only one expression of the disease. The mechanism involved has to deal with a certain content; this content is delusional, but is therefore only the outward expression of the disorder beneath. Therefore the delusion really expresses an effort upon the part of the individual to reach an efficient adjustment. The constellation of ideas with its dominant, painful affect has been one to which the patient could not effectively relate himself, and therefore the next best thing had to be done, and this next best thing was the formation of certain delusions which rendered the existence of the painfully affective complex more endurable. The delusion, therefore, speaking in physical terms, is more comparable to scar tissue than to disease tissue. It represents the location of the wound and the result of the reparative process. To make the matter more clear, an individual who is ambitious, and yet who lacks ability, may develop the delusion that his lack of success is due, not to his lack of ability, which he persistently refuses to see, but to the interference of enemies who are jealous of him and who persecute him and try to belittle him in the eyes of his superiors. In this way an inacceptable fact—his inefficiency to which he cannot make adequate adjustment—is so distorted (mechanism of distortion) that it would appear that the results of this inefficiency emanate not from within, but from interferences from without (projection). This illustration shows well the simultaneous presence of both delusions of persecution and delusions of grandeur. And so the patient creates a situation in which he, so to speak, finds himself able to get along, for as painful as a system of persecution of the sort which he creates for himself may be to him, it is less so than a realization of his own inherent defects. It will be seen, therefore, what is meant when it is said that the delusion, speaking in physical terms, represents scar tissue, and it will be seen also how a destruction of the delusion could in no way cure the disease. If the delusion could actually be destroyed the patient would be in much the same position as a patient who had a scar cut out; another delusion would have to take its place, because the patient would be thrown back upon the same inacceptable situation to which he would find himself again incapable of making efficient adaptation.

The other mechanism which is revealed in this illustration is of great importance as being characteristic of the paranoid reaction-type, namely, the mechanism of projection, whereby the individual projects, as it were, upon the outside world his own mental difficulties which return to him in this instance in the form of persecutions. This mechanism of projection appears to be fundamental in paranoid trends. It is at the basis also of the ideas of grandeur. Here the patient projects, not his difficulties, but his ambitions, and his hopes come back to him from the outer world realized. In fact, the mechanism of projection is not necessarily a pathological mechanism at all but is constantly used by healthy persons. This is true of all the mechanisms found at work in the psychoses, it is not the mechanism that is wrong,

it is the failure of the mechanism to effect adequate adjustment which brings the individual to grief. Ideas of grandeur are always present. Persecutory and grandiose ideas are the opposite sides of the same mechanism. Either may dominate the picture but both are always present.

The most elaborate attempt at interpretation of the paranoia syndrome was made by Freud[1] in his analysis of the Schreber case. Herein Freud voiced the view that paranoia was dependent upon a homosexual fixation in the psychosexual development of the individual. To make this statement somewhat clearer it should be recalled that the individual in his psychosexual development is first auto-erotic, that is, interested only in his own body, that his next interest is in those immediately about him, the members of his own family, and particularly those of his own sex, in other words, those who have bodies most like his own, and that these stages have to be passed through before the normal end-result in a heterosexual love object is attained. The unconscious auto-erotic interest in his own body or someone like himself (homosexual) is designated as narcissism, and the paranoiac mechanism is dependent upon a fixation and development at this period. In the normal development of the individual the unconscious homosexual tendencies are not entirely eliminated by any means, but the homosexual libido is sublimated, that is, its energies are utilized in other channels, more particularly it is utilized in all those forms of association with the same sex that one sees in friendships, social organizations, clubs, games, and in the higher social activities. But with a fixation-point at the narcissistic-homosexual period of psychosexual development the patient is constantly in danger. Any serious conflict is liable to cause a regression of the sublimation to the point of fixation, and this is considered by Freud to be the mechanism at the basis of paranoia. "Persons who cannot rise completely out of the stage of narcissism and are thus prematurely fixed or arrested in the evolution of their dispositions, are exposed to the danger that a flood of libido, which finds no outlet, sexualizes their social tendencies and reverts the sublimations achieved in the course of development." The libido of the paranoiac is then projected upon those about him.

The whole process is briefly and ingeniously set forth by Freud by means of ringing the changes—supposing the paranoiac to be a male —upon the basal sentence "I love him," thus:

Delusions of persecution contradict the verb. "I love him" is resented by the individual who reacts to the feeling by "I do not love—I rather hate him." Then this feeling of hate is projected with the result "he hates (persecutes) me, which justifies my hating him." As a result, this feeling, appearing to come from an outer perception

[1] Psycho-analytische Bemerkungen über einen autobioghraphischen beschriebenen Fall von Paranoia (Dementia paranoides), Jahrbuch für psycho-analytische und psychopathologische Forschungen, Bd. iii, 1911. A very excellent and full account in English of Freud's analysis of this case can be found in the Psychoanalytic Review, vol. 1, No. 1.

becomes "I really do not love him—I hate him—because he persecutes me."

Erotomania contradicts the object. "I do not love him—I love her," then "I notice that she loves me," then finally, "I do not love him—I love her—because she loves me."

Delusions of jealousy contradict the subject. "Not, I love the man —she loves him."

Delusions of grandeur result from a total contradiction, a rejection of the whole sentence. "I do not love at all," and hence, "I love nobody." As the libido must be accounted for, this is equivalent to "I love only myself."

This mechanism is an excellent illustration of the necessity for never forgetting that the symptoms are the creation of the patient and not of his environment. The paranoiac tries by the nicest subtleties to prove that the trouble is not with himself but is due to outside circumstances, unfortunate occurrences, the machinations of enemies, and he succeeds only too often in convincing others that this is the fact. The symptoms can only be understood when the alleged disturbing factors, the persecutors, for example, are understood to be but symbols of one aspect of his psyche, the regressive aspect which really embodies the energy which is working to his detriment. In hysteria an analogous situation is produced by a projection, so to speak, of the patients' difficulties upon their own bodies. (See Hysteria.)

In speaking of the voices heard by paranoid patients Reik says[1] very illuminatingly: "We know that people suffering from them hear voices speaking to them in the third person, incessantly observing and criticizing all they do and leave undone. According to Freud this critical faculty can be traced back to parental criticism and the development of the conscience is reproduced in retrospect by the sufferers, who now again project the voices back to the outer world whence they came. It is a characteristic fact that the voices they hear talk about them in the third person; it is supposed that this represents the memory they have retained of their attendants in infancy who observed and commented together on the child's movements. Their place is later taken by other people and eventually by the community ('public opinion')."

From this point of view paranoia, and paranoid types of reaction, are problems resulting from an inadequate sublimation at the homosexual stage of psychosexual development. The problem of paranoia is therefore but one aspect of the much larger problem of homosexuality the fundamental nature of which we are still far from understanding.

Diagnosis.—Attention has already been called in the body of this chapter to the different conditions which have to be borne in mind in making a diagnosis. There are many paranoid states, and wherever the paranoid mechanism is present then it is proper to speak of a paranoid state. These paranoid states are found in many of the psychoses.

[1] Psychoanalysis of the Unconscious Sense of Guilt, Internat. Jour. Psa-An., October, 1924, **5**, Pr. 4.

They may be more or less permanently associated with the special attack, as in the manic-depressive psychosis, or as in alcoholic hallucinosis, or there may be transient episodes, as in general paresis. It will be seen, therefore, that there are many and various types of symptomatic paranoid states and that perhaps the main consideration in the matter of diagnosis is that a condition which is symptomatic and transitory should not be mistaken for a chronic, progressive, and probably irrecoverable psychosis. This differentiation cannot always be made on the basis of a cross-section, but the patient must be studied carefully over a considerable period of time, and a reasonably full history antedating the period at which he came under observation must also be had in order to see what the general progress indicates. A psychoanalytic investigation of the symbolic meanings of the delusional ideas is essential.

Pathology.—Paranoia has heretofore had no definite somatic pathology. The reason for this seems to have been the feeling that pathological changes, in order to account for mental disease, must be found in the cerebral cortex. With the present conception of the organism as a unity it can be seen how any inadequacies of functioning of the individual as such, as a whole, may be reflected at the psychological level as abnormal mental states. In harmony with this idea Lewis has found[1] that in paranoid states the somatic pathology can be expressed as can the psychopathology, namely, as a tremendous effort at compensation. Just as, at the psychological level, the delusional system compensates the patient for a reality that is unendurable so at the somatic level there are, throughout the body, areas of defect and areas of hyperplasia (compensation). The main stress, however, seems to fall upon the circulatory system for these patients all seem to die a vascular death. (See p. 969.)

The hereditary taint is sometimes manifested by errors of development—giving rise to malformations generally known as *stigmata of degeneration;* abnormalities in the course of the cerebral vessels and asymmetries and abnormalities of gyral configuration have been noted.

Treatment.—For a considerable time past the general attitude toward the group of cases included under the designation of paranoia has been that they were incurable. The outlook has been an extremely dark and pessimistic one, and correspondingly therapeutic efforts have been paralyzed at their very inception. A somewhat changed attitude toward the whole group was the natural result of the development of a concept of paranoia which was more circumscribed and applicable to a more limited number of patients, and when correspondingly it was learned that there were many paranoid states associated with essentially recoverable psychoses; in other words, as the paranoia concept has become more and more contracted it has been realized that a great many of the paranoid conditions, which formerly were grouped under the head of paranoia, really belonged to

[1] Saint Elizabeth's Hospital material.

recoverable transient conditions, and therefore the outlook for them was good. On the other hand, as the paranoia concept has contracted it cannot be said that there has been any increase, at least until very recently, in the hopefulness for this limited group of cases.[1]

It could hardly be expected that at a time when paranoia was considered to be an absolutely hopeless and irrecoverable psychosis, chronic and progressive in its very nature, that therapeutic results would offer much, or that there would be found many who would even give any material effort in this direction, but here and there scattered through the literature are reports of cases of paranoid type which seemed to have been influenced by this or that form of therapeutic procedure, and more recently, since the doors have been opened and one has been able to enter more intimately into a knowledge of the mechanisms that are involved in the development of the psychoses and when it has been seen that these mechanisms at least are quite the same in mental disease as they are in health even, and that the mechanisms of chronic psychoses are quite the same as those found in recoverable conditions and also in health, and that the diseased feature was not so much the mechanism as the fixation of the individual at certain periods of development or with reference to certain constellations of ideas, it became at once an open question whether these conditions might not be susceptible of the same sort of modification as they are in less serious conditions. And so within the past few years there has been an awakening of interest in these chronic psychoses and efforts are being made here and there to penetrate their mysteries and to modify their course, with the result that already a number of cases of paranoia have been reported as having had a favorable outcome.

The general principle of treatment, at the psychological level, resolves itself into as complete as possible an unravelling of the tangled skein of the patient's mental life, an uncovering of the activating circumstances in his career which have been the etiological factors in the development of the psychosis, and by so doing modifying his mental trends by a progressive process of readjustments and reëducation. This is the work of one skilled in the analysis of psychological situations and is of quite the same nature as the psychoanalytic treatment of the neuroses.

It seems certain, from the results of dealing with these paranoid conditions, that, aside from any definite ability to modify the course of the psychosis or to produce a definite curative result, the psychoanalytic method of attack may, not infrequently at least, lead to a certain amount of transfer, that the physician may come to be highly respected and affectionately regarded by the patient to such an extent at least that he may very largely control the patient's activities. This has been known to happen under rather extraordinary circumstances, showing a very high degree of personal influence by

[1] A. Meyer: Treatment of Paranoia in Modern Treatment, Nerv. and Ment. Dis., White and Jelliffe, Lea & Febiger, 1913; Bjerre: Psychoanalysis, Boston, 1917.

the physician over the patient, despite the fact of well marked and fixed delusional beliefs.

In dealing with paranoiacs it must always be remembered that one is dealing with a class of patients who are potentially dangerous, that to this group belong perhaps the most dangerous of the so-called insane, and aside from matters of psychoanalysis, or in fact of any questions of therapeutic endeavor, it must be realized that where it is evident that the welfare of the individual and the welfare of society cross, the welfare of the individual must give way in favor of that of society. If the paranoiac is actually dangerous it is necessary to intern him in some institution where he will get proper care.

The question as to whether a given paranoiac is dangerous or not, in the absence of any overt acts, is often an extremely difficult one to decide. In any case it is a question to be decided by a study of the individual case and it always includes a consideration of many factors. Among these factors the following may be mentioned: It is important to find out how completely the mentality of the patient is permeated by the delusional system, in other words, how much or how little freedom he has from delusional control, whether all of his mental forces, so to speak, go to reinforce the delusion, or whether, on the other hand, he is left reasonably free for a considerable portion of the time, in contact with reality, rather than plunged into the depths of his unreal world. It is important, too, to note how clearly defined may be his belief in the activity of any specific individual in his delusional system, whether he believes some person who is living, perhaps nearby, someone whom he can easily come in contact with, is responsible, at the bottom of his persecutions. It is important to see whether the patient, in the consideration of his delusional ideas, is at all subject to the reality motive, whether he has any critique left, or whether his belief is shakable in any degree by others, whether he can be influenced materially by his physician when it comes to the question of his delusional beliefs, or whether they dominate the situation absolutely. It is important to judge the general attitude and mood of the individual, whether he is entirely shut out from any consideration of others, of the world at large, whether he considers himself quite a law unto himself, whether he, for example, is exalted, egotistic, beyond criticism, self-sufficient, and believes that anything that he may decide to do is justifiable. It is important to know whether the patient in his past career has been impulsive, whether he has shown tendencies to do unusual, bizarre, or grotesque things, or to fly into passions, or to be uncontrollable from slight, inadequate, or unexpected reasons. Threats have to be evaluated and an opinion reached as to whether the patient really means to carry them out, or whether they are used as a means of emotional catharsis. The general education, bringing up, and ideals of the individual are important as indicating what he is liable to do. A person who was brought up originally with a proper regard for the proprieties, who is essentially

a gentleman or a gentlewoman, is by that very token not so apt to commit some vulgar, rowdy, indecent act.

And finally, it may be said that while the general attitude toward paranoia has perhaps not materially changed, while there is still pretty good ground for believing that a certain class to which the term paranoia is perhaps alone applicable, is inaccessible to therapeutic endeavor and are doomed to suffer from their psychosis throughout their lives, still even if this is so there is no absolute way of determining this fact except by a consistent and sufficiently prolonged effort to modify the course of the disease, and with the several cases already in the literature which indicate that conditions that might well have been considered chronic and irrecoverable if taken at their face value can still be materially benefited and perhaps cured, no one is in a position to pass final sentence upon any patient after an examination or two, but on the contrary, has the right to feel that there is some hope for all of them and that at least hope should not be abandoned until consistent therapeutic efforts have been applied for a reasonable time.[1]

[1] Consult Critical Digest of the Paranoia Problem, by C. R. Payne, Psychoanalytic Review, vol. ii.

CHAPTER XVIII.

EPILEPSY AND CONVULSIVE TYPES OF REACTION.

EPILEPSY, the "falling sickness," has been known from the earliest times, the very word itself carrying in its history (it is derived from a Greek verb meaning "to seize upon") evidences of the animistic hypotheses of earlier and relatively more primitive ways of thinking.

The word epilepsy is used as a symbol under which are grouped a great variety of conditions which in general are characterized by sudden and relatively transient *attacks* involving for the most part disturbances of consciousness ("faints," "absences," "blanks," amnesias) and convulsive seizures involving the voluntary and involuntary musculatures. Such attacks are the outward manifestations of a wide variety of conditions ranging all the way from the so-called functional neuroses and psychoneuroses (hysteria, compulsion neurosis), the more frank psychoses (dementia precox), toxemic states (uremia, alcohol), many organic diseases (paresis, cerebral syphilis, abscess, softening and tumors) to the grosser defects of development (idiocy).

The natural evolution of the concept symbolized by epilepsy in the recognition that similar "seizures" may result from such a multiplicity of conditions has resulted in a tendency to speak of "the epilepsies" rather than of "an" epilepsy and makes it worth while to consider the attack as due to a faulty distribution of energy which may be brought about in many ways and through divers mechanisms. The wide variety of conditions, as a part of which convulsive reactions with associated disturbances of consciousness occur, cannot be too much emphasized. The toxic states (endogenous or exogenous) are usually transitory and depend upon the continuance of the toxemia, but in defective parathyroid functioning with disordered calcium metabolism the convulsive phenomena continue because the underlying metabolic disorder cannot be permanently relieved. Marked organic changes which are responsible for convulsive attacks are usually cerebral (tumor, softening, hemorrhage, meningitis), but certain organic conditions resident elsewhere, notably the cardiopathy of Stokes-Adams disease and the condition resulting in animals from thymus extirpation, appear to be sufficient causes, while still more obscure factors determine vagal and vasovagal, gastric and intestinal attacks which appear to be dependent upon elements of constitutional make-up at the level of the vegetative nervous system. It is worth while, therefore, to attempt to get a viewpoint of all these conditions dependent upon their common element—faulty energy distribution.

For the purpose of comprehending the epileptic phenomena then,

the nervous system may be viewed in a very simple way. From the standpoint of structure it may be thought of as consisting of receiving organs, designed either to come into contact with the external world (exteroceptors) or with other parts of the body (proprioceptors) spoken of collectively as receptors. The combined material accumulated through these receptors forms the basis upon which certain extensions of the nervous system (effectors) are devised, whereby the reactions of the body are conditioned in a way to bring about that adaptations essential to life or to the maintenance of the social structure. In this way the nervous system is viewed as a mass of interrelated reflexes redistributing the energy received, for the purposes of the organism.

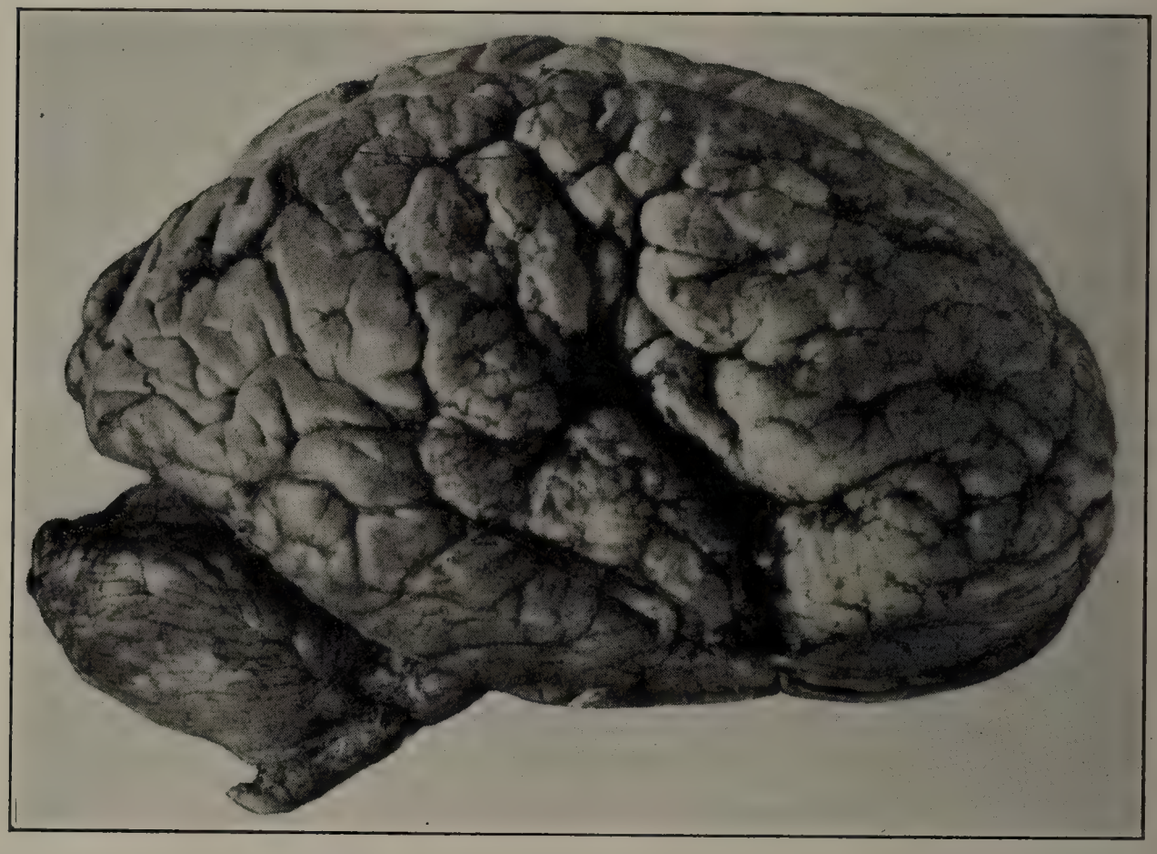


FIG. 439.—Diffuse sclerosis of cortex with atrophy along the Rolandic and Sylvian fissures (epileptic).

As the incoming stimuli are multitudinous, so the outgoing activities are correspondingly diverse, and a healthy organism is able, by reason of its nervous mechanisms, so to distribute the energy received as to bring about a series of harmoniously adjusted activities, be they physicochemical, sensorimotor, or psychic. This viewpoint, that energy distribution takes place at all of these levels, should not be lost sight of, as there is a tendency to think of the problem solely in terms of muscular work. Atwood's "man in the box" broke up more nitrogen compounds during mental than during mechanical work. This breaking up of nitrogen compounds is, however, only one form of registering the energy output.

That which characterizes a well adjusted nervous mechanism is its ability to properly and in an orderly manner distribute its energy,

but that which characterizes the pictures of the disorders included in this chapter is inefficiency in this regard which may be more especially emphasized at the physicochemical, the vital, or the psychic levels.

An explanation for epileptic attacks which finds its ultimate expression under such symbols as eye-strain, floating kidney, gliosis, or like specific indictments fails to realize that the nervous system contains representations of all of the organs and that the final activity of the human body is the result of the balance which has been struck among

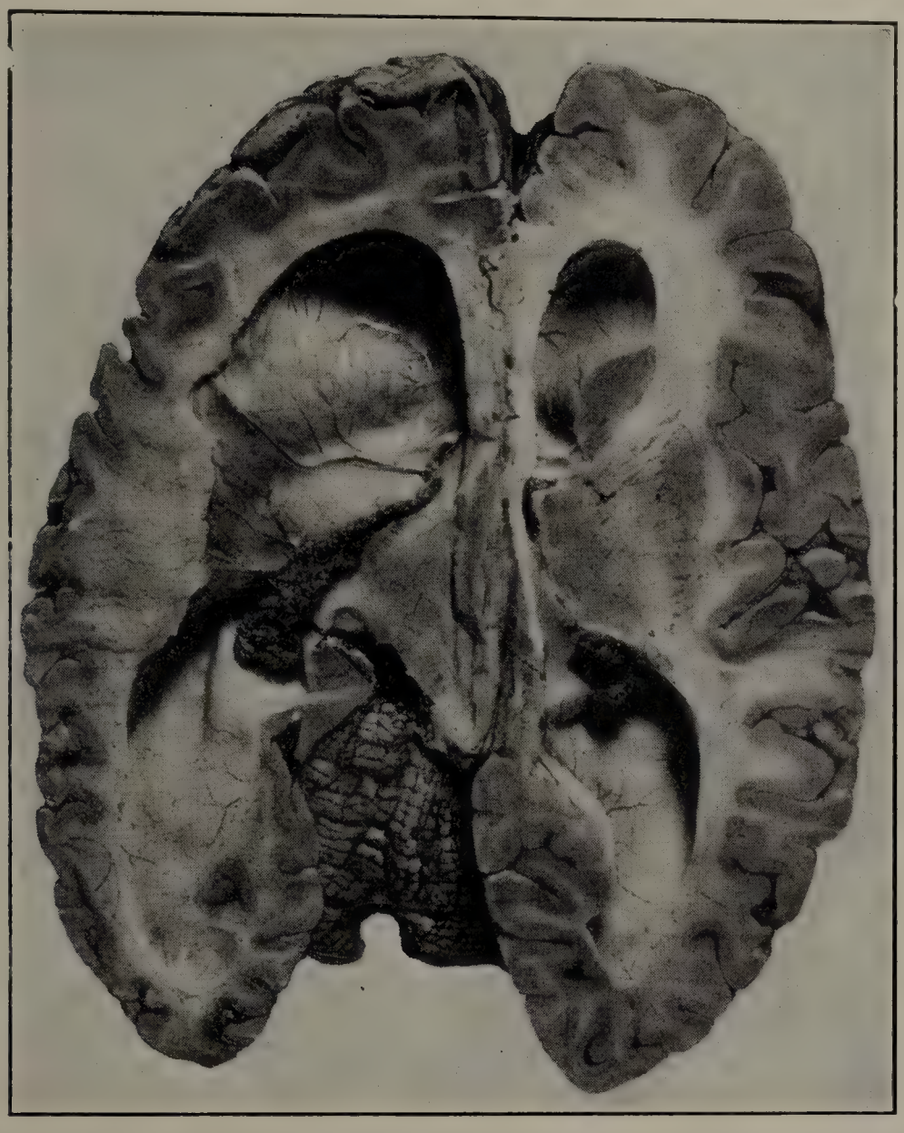


FIG. 440.—Gross anatomical lesions associated with epilepsy. An asymmetrical internal hydrocephalus which also showed a small tumor at the lateral border of the cerebellum.

innumerable tendencies. The part that any particular organ plays can only be understood when taken in consideration with the organism in its totality and realizing the specific part that the organ in question plays in the whole problem.

Bearing in mind this view of the nervous system, as a great mass of complexly interrelated reflexes, and further, the law of avalanche (Cajal) which insures the continuous breaking up of the original sensory stimulus into an ever-increasing number of avenues of discharge, it will be seen how many ways are open to interfere with the

orderly procession of energy through this complicated series of reflex arcs. The nature of the epileptic discharge, essentially a manifestation of energy at greatly reduced adaptive efficiency, and the destructive character of certain pathological lesions which initiate it (impaired metabolism, gross destructions, psychic imbalances) indicates that the essential defect is not irritant but destructive, the blocking or closing of many paths of outlet structurally or by inhibition, and so accumulating the discharge within relatively narrow confines. Such a conception would apply equally well to the "idiopathic" or "genuine

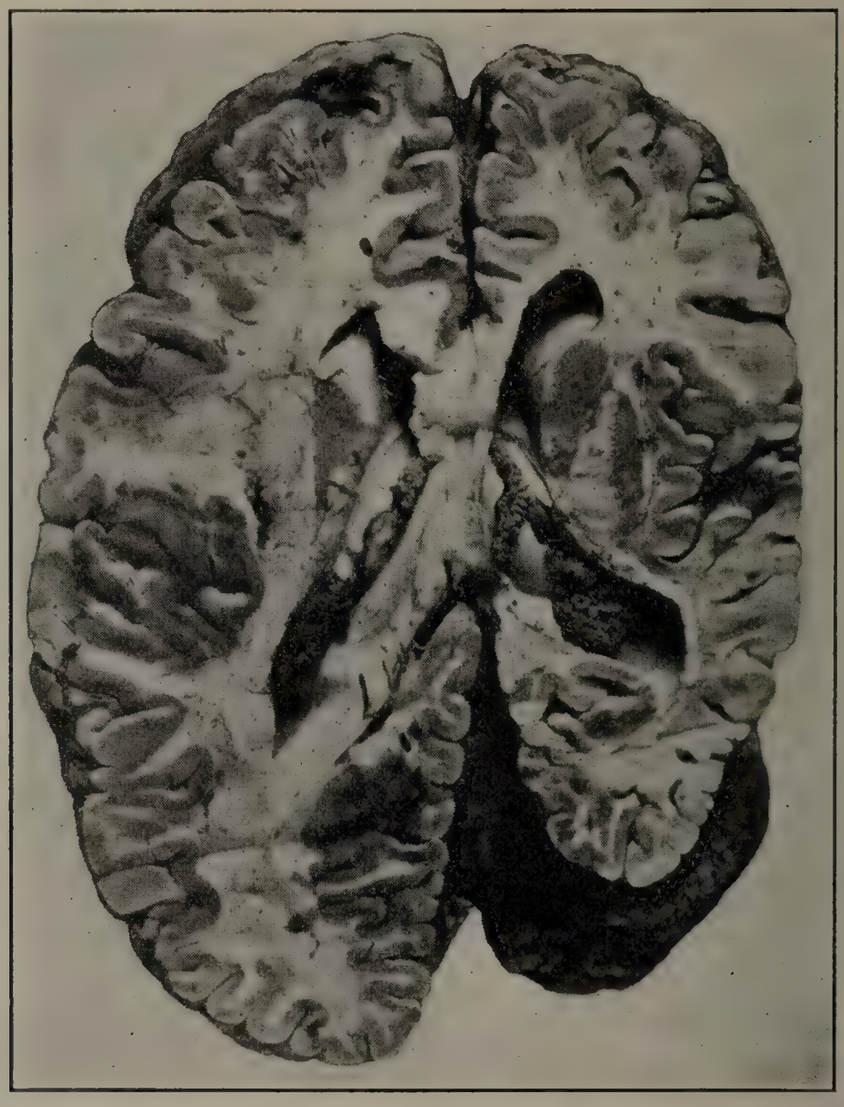


FIG. 441.—Gross anatomical lesions associated with epilepsy. Cerebral asymmetry and ventricular dilatation following an early meningitis. The dura over the atrophic hemisphere was of the consistence of an egg-shell. Section of preceding brain.

epilepsy" with Ammon's horn gliosis and the typical character traits, and to the epilepsies associated with marked developmental defects (idiocy) in which it may be conceived that the wider paths for avalanche discharge have not been laid down. This viewpoint is also consistent with the different levels at which the discharge may take place—psychic, physiological, and physico-chemical—the character of the attack as limited to certain levels, the psychic (hysteria, dementia precox), the physiological (Jacksonian types), the physico-chemical (tetany); the distribution of the discharge, general attacks ("genuine

epilepsy"), localized attacks (Jacksonian types), and as being initiated by sensory, motor, or psychic prodromes.[1]

Symptom Groups.—Convulsions may occur, as already indicated, in a great variety of conditions while distinctly explosive attacks not convulsive in the sense of muscular spasms, occur under still wider conditions in states not definitely epileptic but, with reference to the more essential epilepsies, in what may be referred to as borderland conditions.

The essential epilepsies have been divided into the *late epilepsies* occurring relatively late in life and dependent upon toxemias and gross organic changes and the *early epilepsies* which occur relatively early in life, generally before or during adolescence.

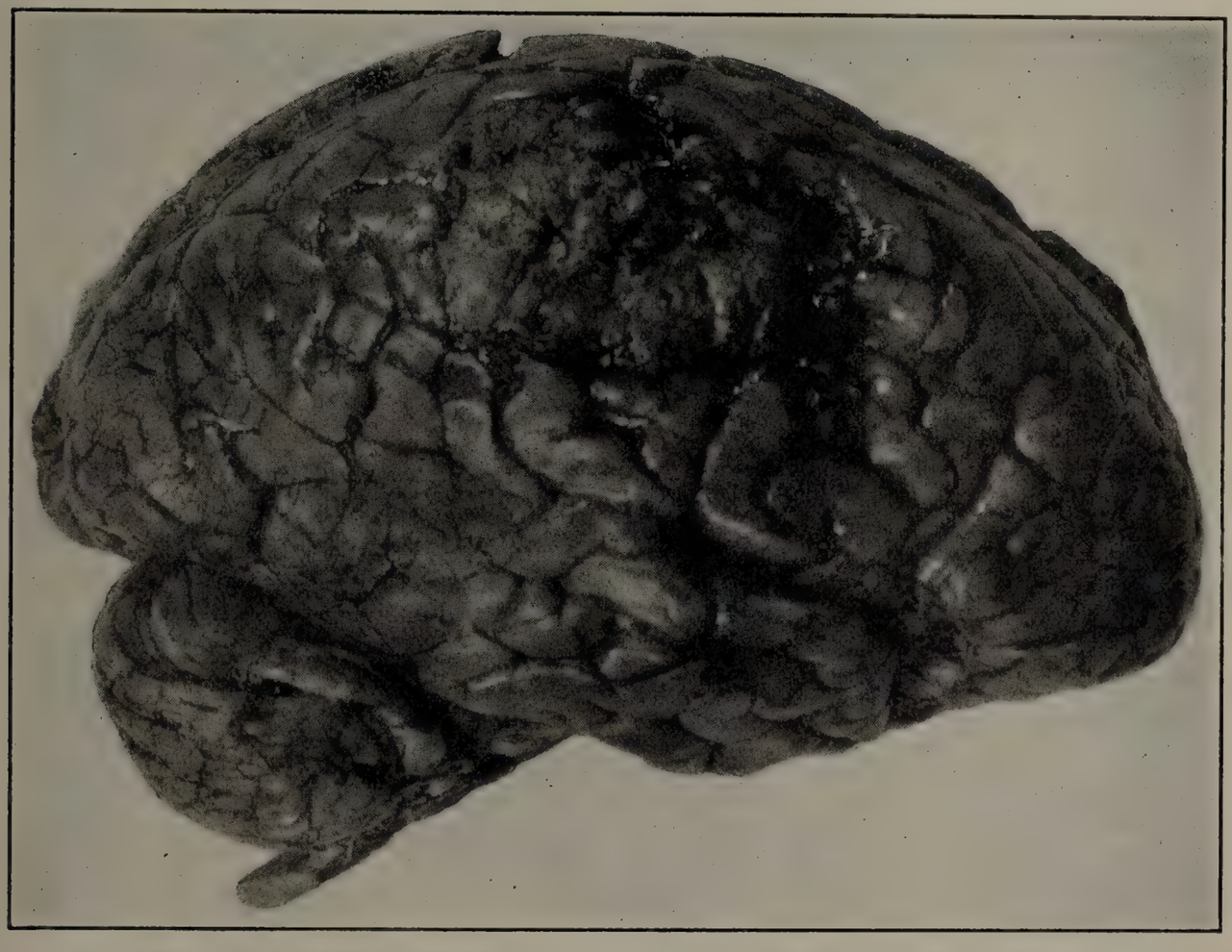


FIG. 442.—Gross anatomical lesion associated with epilepsy: Tumor.

Epilepsies of Gross Brain Disease.—These occur in paresis, cerebral syphilis, brain cysts (echinococcus, etc.), hydrocephalus, the cerebral meningitides (syphilitic, tuberculous, serous, and pachymeningitis), bony tumor of skull, traumatisms (fractures, insolation, concussions, hemorrhages), multiple sclerosis, cerebral sclerosis and gliosis, cerebral arteriosclerosis, the encephalitides and myoclonias.

The Epilepsies of Toxic and Infectious Origin.—The toxemias include those of endogenous origin (acetonemia in children, uremia, diabetes), of exogenous origin (metallic—such as lead and arsenic, and strychnine, alcohol, absinth, santonin, and carbon monoxide).

[1] For literature from 1900 to 1910 see Referate by Gruhle. Ztschr. f. d. gesamte Neurol. u. Psych., Band ii, Heft 1. Binswanger: Epilepsie.

The infections are more especially the exanthemata, influenza, rabies, malaria, rheumatism, syphilis, etc., operating either through the mechanism of an overwhelming toxemia or by meningitides or encephalitides.

Anomalous and Borderland Conditions.—Here are included certain internal secretory imbalances, particularly diseases of the thymus, thyroid, and parathyroids and degeneratio-adiposo-genitalis.

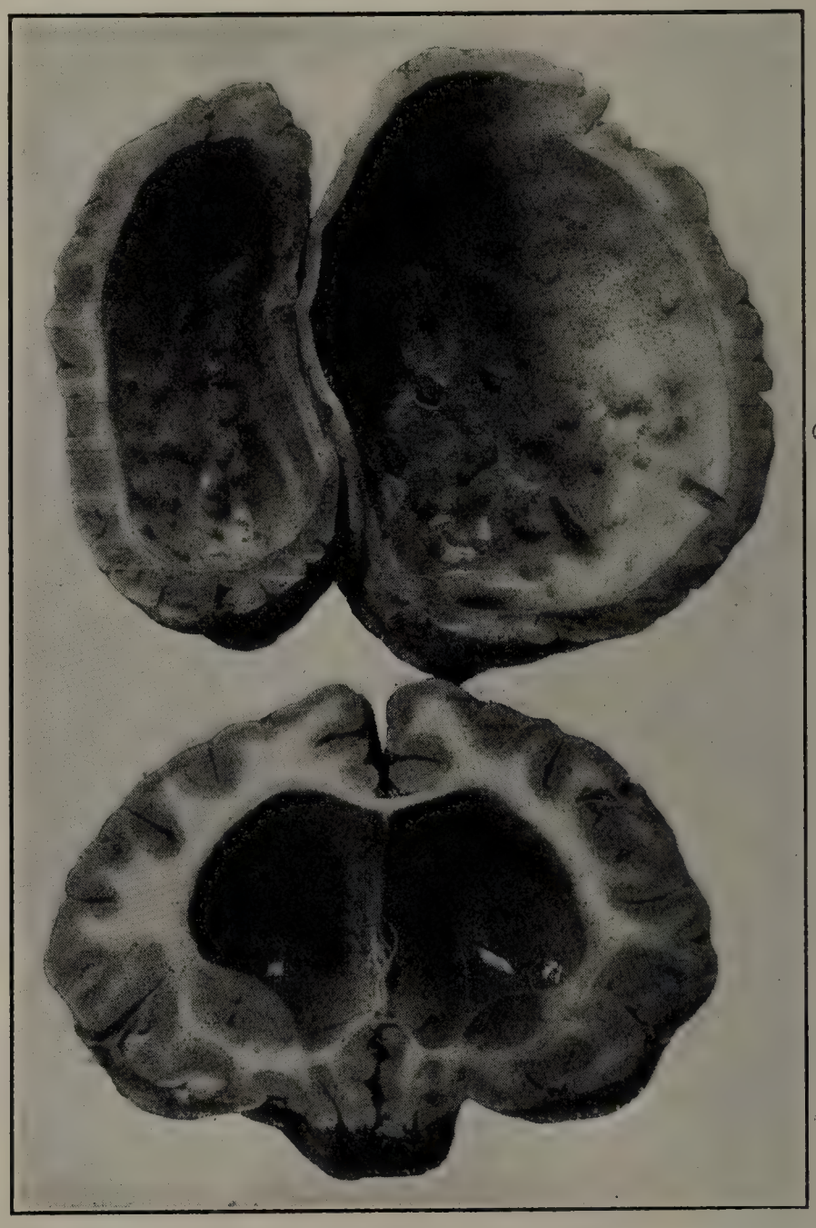


FIG. 443.—Gross anatomical lesions associated with epilepsy. Internal hydrocephalus. Marked feeble-mindedness.

Bolton[1] includes the genuine epilepsies here as being due to insufficiency of the thyroids and parathyroids. He thinks that when the parathyroid function is absent tetany is the result; when the thyroid and parathyroid functions are both absent then appears both epilepsy and tetany (postoperative cases); if, however, there is a congenital reduction of the functions of both thyroid and parathyroids, the result is epilepsy alone (the genuine form). This result is due to chronic

[1] Drei Aufsätze über Epilepsie. Monatschr. f. Psychiat. u. Neurol., 1916, **39**, and Epilepsie und Tetanie, Deutsch. Ztschr. f. Nervenheilk., 1919, vol. **57**.

autointoxication of intestinal origin. Under normal conditions the products of intestinal decomposition are further broken up by the thyroid and parathyroid and rendered innocuous, but when these glands are unable to function adequately a toxemia results. Curschmann[1] sees the key to the situation in the deficiency of calcium in the central nervous system due to parathyroid insufficiency. Calcium has a sedative effect on the cortex and its removal results in an irritability (release of function) and leads to a pure parathyroid eclampsia or to epilepsy and sometimes to a late spasmophilic epilepsy.

Certain high level attacks occur in hysteria, compulsion and anxiety neuroses, and in dementia precox, especially the catatonic form.

Here also should be included the vagal[2] and vasovagal attacks, slight passing disturbances of consciousness associated with vertigo and sometimes sensory disorders as loss of sight, possibly certain sensory disorders of fulminating character such as migraine, and some disturbances of sleep (narcolepsy), disturbances of consciousness of syncopal nature, and the affect epilepsies of the Bratz type.

Convulsive attacks may be combined with a number of conditions. The association of myoclonia is one of the most intimate—*myoclonus epilepsy*. This combination is familial, several children of the same family suffering and sometimes successive generations being affected. Here are epileptiform attacks, particularly at night, and myoclonic contractions affecting especially the muscles of the tongue, pharynx, and diaphragm. The relation of the two symptom groups is not clear.

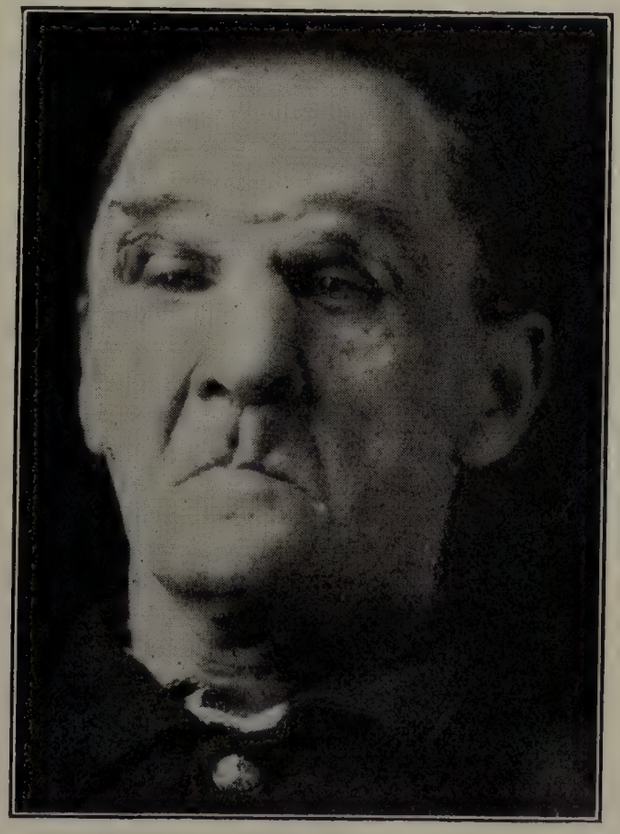


Fig. 444.—Epileptic, showing scars over eyebrows from falls.

Pathological Groups. — The preceding clinical grouping gives a fair idea of the pathological conditions which may be found. The following grouping is given by Alzheimer[3] as a result of the histological examinations of 63 cases. These simply indicate the reasons why and how the structure of the brain is modified, thus changing its functional capacity as an energy distributor.

A. Cases with very obscure etiology (genuine epilepsy):

1. This group comprises 60 per cent. of the cases:

[1] Epilepsie und Tetanie, Deutsch. Ztschr. f. Nervenheilk., 1918, vol. **61**.

[2] Wm. R. Gowers: The Borderland of Epilepsy, Philadelphia, 1907.

[3] Alzheimer and Vogt: Die Gruppierung der Epilepsie, Jahresversammlung des deutschen Vereins für Psychiatrie, 1907; Ref. Allg. Ztschr. f. Psych., Band **64**, 1907.

(*a*) With sclerotic changes in Ammon's horn.
(*b*) With superficial gliosis of the hemispheres.
(*c*) With signs of an acute process (status) besides *a* and *b*.

2. This group comprises 4 per cent. of the cases: The process in these cases can be called an *atrophic sclerosis*. There is considerable atrophy of different regions; no important changes in the vessels; deep impairment of the nervous structures, especially the third cell layer of the cortex.

B. Cases due to external poisons:
1. Alcohol: Different anatomical changes, as in chronic alcoholism. Besides these sometimes acute changes, as in delirium.
2. Lead: Different changes. Experimentally lead produces a genuine encephalitis.

C. General diseases:
1. Syphilis: Different forms of brain syphilis, especially the endarteritis of the finer vessels (Nissl, Alzheimer).
2. Arteriosclerosis.

D. Focal diseases: Most of the cases in this group are cases of epilepsy with idiocy after encephalitis.

E. Arrests of development:
1. Stadium verrucosum (Rancke).
2. Sclerosis tuberosa.

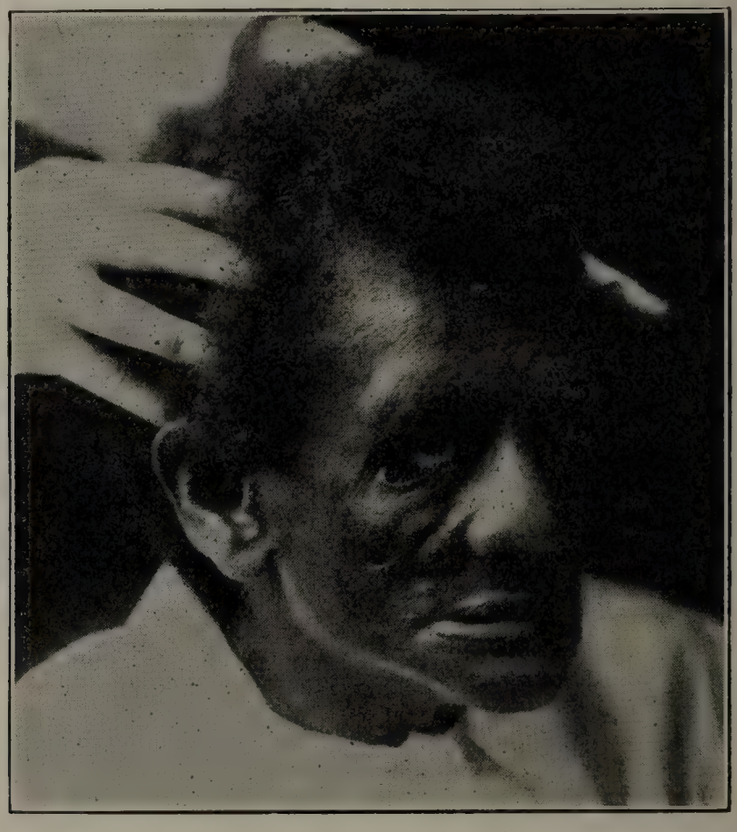


FIG. 445.—Epileptic, showing scar from burn due to injury during convulsion.

This survey will suffice to show what a wide variety of conditions have been included under the term epilepsy and also to point out the

various groups that are being at present split off and separately identified. What has been said about the distribution of energy applies to the broad group of convulsive reactions. The disease "genuine epilepsy" will now be briefly described, although it is extremely difficult to do this at all accurately as it must be borne in mind that heretofore all matters of description and questions, such as those of heredity, are with practical uniformity considered with reference to "epilepsy," without effort at discrimination, much as is the case with the corresponding conglomerate "insanity."

CLASSICAL EPILEPSY.

This roughly corresponds to the group "genuine epilepsy" of Alzheimer and includes those cases which are found to have Ammon's horn and cortical sclerosis although there are cases of "genuine epilepsy" which do not give these findings.

Heredity.—The genuine epileptic usually comes from a badly tainted stock. Epilepsy may not appear in the ancestors but they and often the collaterals show evidences of ill-defined nervous disorders (according to Davenport and Weeks,[1] migraine, chorea, paralysis, and extreme nervousness). Epilepsy and feeble-mindedness show a great similarity in their hereditary reactions and both appear to be due to a defect of the germ plasm, that is, they are both recessives. As should be expected, therefore, the two conditions are frequently found associated. This is also of significance in relation to what has already been said about the developmental failure to lay down the paths for the higher avenues of sensory avalanche.

The Epileptic Constitution.—The classical epileptic is apt to be morose, irritable, suspicious, and hypochrondriacal. He is quite characteristically unreliable and with it all frequently presents a very aggressive form of sentimental, shallow religiosity. This type of epileptic, in general then, is very sensitive, irritable and insincere. He is egocentric to a very considerable degree, paying great attention to himself, his own feelings, his state of health, his physical comforts, and his immediate surroundings. His interests are variable and he presents light variations of mood with perhaps headache and a tendency generally to hypochondriacal fixations. His interests all tend to be concentrated in this egocentric constellation. His reactions of irritability and unreasonableness present infantile characteristics. Many epileptics are feeble-minded or more profoundly defective, and rather in conformity with this frequent finding the word associations from epileptics have close analogies to the word associations of the imbecile. In addition to these traits of character these epileptics are usually lazy, frequently they lie openly, present an attitude based on high moral standards of great respect and consideration to one's face and quite the opposite when one's back is turned. Their general health

[1] A First Study of Inheritance in Epilepsy, Journal of Nervous and Mental Disease, 1911, **38**, No. 11.

is apt to be good and they often have enormous appetites, and are especially fond of proteids. While good-natured, even-tempered, well-disposed epileptics exist they are more apt to be most difficult problems to get along with, and as a class in the hospital they are extremely difficult to care for. Passing attacks of mental disturbance occur in the interparoxysmal period without apparent relation to seizures. Attacks of *transitory ill-humor*, according to Aschaffenberg, occur in 78 per cent. of cases. This is a condition of irritability, unreasonableness, sometimes associated with delusions and hallucinations. The patient is in a "touch-me-not" state and very apt to get into quarrels or make attacks. Rarely the disturbance is expansive in type and in these cases may be associated with religious fervor.

A study of the sexual characteristics of epileptics has been made by Maeder.[1] As a result, he finds the sexuality of the epileptic still largely undeveloped, still only little removed from the infantile stage. The sexual feelings are very prominent and are aroused in many ways: autogenically, constituting *auto-erotism*, and giving rise to such phenomena as masturbation, and by stimuli from without, constituting *allo-erotism*, which gives rise to a normal libido reaction (heterosexuality), homosexuality, exhibitionism, etc. Maeder uses the term *polyvalent* to describe that characteristic of the epileptic sexuality which permits it to be aroused by many kinds of excitants.

The epileptic state leads in a certain proportion of cases, if it has begun in early life, to conditions of feeble-mindedness, imbecility, and idiocy, or, depending upon the same causes, is associated with these conditions. Epilepsy may seem in many cases, to produce a general mental deterioration (*epileptic dementia*) which may become very profound. These cases, at least many of them, on prolonged observation prove to be cases of dementia precox with convulsive manifestations.

A study of the personality of epileptics by Clark[2] has led him to the conclusion that this general type of character as described above is a result of the disease and not a precedent condition.

Scripture and Clark[3] have described the epileptic voice sign and found it in 75 per cent. of cases. The voice has been studied by the "air puff" method of recording on the kymograph. A measure of the wave gives the rates of vibration. A line connecting the tops of the ordinates produces the "melody plot." Normally each vowel has a rising and falling melody. In epilepsy the vowels run along on an even tone—"plateau speech." This is very characteristic and easily recognized.

Muskens, in his study of the muscular phenomena, has found fatigability and weakness of single muscles or muscle groups, startings, shocks and cramp-like contractions especially just before or after going

[1] Sexualität und Epilepsie, Jahrb. f. psychoanalytische u. psychopathologische Forschungen, 1909.

[2] A Personality Study of the Epileptic Constitution, Am. Jour. Med. Sci., November, 1914.

[3] Researches on the Epileptic Voice, Proc. New York Neurol. Soc., November 12, 1907.

to sleep. All sorts of physical disturbances may be associated with the epileptic state which, like the character, show infantile characteristics. Epileptics get along best under very carefully protected circumstances and they are especially prone to suffer from a change in the accustomed routine, a change of diet being accompanied by excesses and a subsequent gastro-intestinal upset.[1] As the disease progresses and becomes chronic and confirmed one will note many evidences of disturbance at the vegetative nervous level.

The Seizure.—The classical major epileptic attack (grand mal) is sudden in onset, often preceded by a warning—aura. The patient falls and the attack immediately develops into a tonic spasm with unconsciousness. The tonicity is replaced in a few moments by clonic spasms which gradually subside. There is then often a short period of automatic activity followed by a gradual return to full consciousness, or the patient sinks at once into a deep sleep from which he awakes complaining of lameness and weakness in the muscles that were convulsed, and perhaps headache.

The attack has been described by authors in great detail, but is really different in practically each case, although the type tends to remain the same in each patient.

The *aura* may be sensory, motor, or psychic. The sensory warnings may occur in any of the sensory fields: the visual (flashes of light, hallucinations), the olfactory (odors, usually bad—uncinate fits), the auditory (simple sounds or hallucinatory voices), etc. The epigastric aura is most common and consists of a wide variety of disagreeable sensations in the epigastrium.

The muscles first involved vary greatly, as does also the order in which they are involved—march of the convulsion. The patient falls at the beginning of the spasm, the direction of the fall being dependent upon the muscles first affected. In a few moments all of the voluntary muscles are convulsed, including the muscles of respiration, producing cyanosis, and the jaw muscles, resulting in biting the tongue. With the beginning of the clonic stage frothy, bloody saliva issues from the mouth and the cyanosis gradually disappears with the resumption of respiration. Urine and feces may be passed during the attack—usually only the former. In the partial or incomplete seizures—petit mal—the convulsive phenomena are much milder and may even escape observation altogether, while the disturbance of consciousness is shorter in duration and less profound. The patient may blanch, become confused for a few moments, perhaps falter in what he is doing, or fumble for a few moments about his clothing in a dazed fashion and then go on about his affairs as if nothing had happened. These attacks are also often preceded by an aura.

The psychic disturbances associated with the attack, before and after, or replacing it are many and varied.

[1] MacCurdy, John T.: A Clinical Study of Epileptic Deterioration, Psychiatric Bulletin, April, 1916, vol. **9**, No. 2.

In a great many epileptics there is a marked disturbance preceding the convulsion, sometimes of several days' duration, and those who are accustomed to the patient can tell that a fit is impending. This change manifests itself in increased irritability, complaining, sometimes by depression or dulness, and there may be associated disturbances of the sensorium, hypochondriacal complaints and hallucinations. All these conditions are commonly promptly relieved by the fit.

Immediately after the convulsion there is often a temporary condition of confusion. The patient rises clumsily, looks about him in a bewildered manner and often does some semi-automatic acts, such as taking off his clothes. Also following the attack a transitory exhaustion paralysis in the overacting muscles makes itself apparent.

Just before, or more commonly after, the convulsion a condition of active excitement may occur which may reach the stage of frenzy. In this state the patient is a veritable wild man—*epileptic furor*. He is liable to kill anyone who approaches or even himself. Fortunately his efforts are diffuse and not coherently directed. During this attack, which is usually brief, he has to be restrained and at the end is quite completely exhausted.

An attack of mental disturbance may take the place of the convulsion and thus become an *epileptic equivalent*. These attacks of *psychic epilepsy* frequently take the form of so-called *epileptic automatism* or *epileptic dream states*. In these conditions the patient may do almost anything and when he comes to himself he has absolutely no recollection of what has happened. Usually the attacks are of short duration and the acts rather simple—more simple than in the dream states of alcohol or hysteria. However, they may last for days, all sorts of things may be done, crimes may even be committed, so that the condition often becomes of great medicolegal importance. The crimes of violence are often noted for their ferocity and brutality.

It must not be forgotten that these states may be associated with a seizure that was so slight as not to have been noticed. Evidences of such a seizure, especially in medicolegal cases, should always be looked for.

Transitory conditions of *depression, excitement, confusion, delirium* and *stupor* may develop and quite characteristically a condition of *ecstasy* with hallucinations. The patient sees the gates of Heaven open and as the heavenly hosts appear he hears himself addressed by the voice of God.

The transitory states of ill-humor, as described by Aschaffenberg in the interparoxysmal state, might also be considered as psychic equivalents; these are frequently associated with drinking.

Besides these conditions, *paranoid psychic states* are quite common, while of the more transitory psychic manifestations *fugues* are frequent and certain types of *dipsomania* appear.

In addition to the symptoms thus far indicated various observers have found evidences of an altered blood picture such as leukocytosis and hypo-eosinophilia, while disorders of metabolism with hyper-

toxicity of the secretions, has long been adduced as proof that the manifestations were dependent upon chemical poisons due to faulty metabolism.

Meaning of the Attack.—Bearing in mind what has already been said about the distribution of energy, it will be of advantage to pursue this line of thought somewhat further.

Energy flow may be blocked, dammed up, and break through in diffuse discharge at any level—psychic, sensorimotor, or physicochemical. This being so it would be expected, as is the case, as already indicated, to find disturbances at each of these levels. In the classical epileptic attack all these levels are involved, but what is of equal or greater significance to the general hypothesis is that there are attacks practically limited to one level, and a study of the several types of convulsive reaction will show a series of cases reaching through all of the intermediate stages from the highest to the lowest instinctive levels.

The hysterical convulsion offers an example of a high level convulsive type of reaction. This seizure is admittedly psychogenic in origin and presents the picture of a conversion of psychological into physiological symbols. In other words, the patient escapes from his painful ideas by converting them into physical symptoms. (See Hysteria.)

The disturbance of consciousness in these hysterical attacks is relatively slight, much less than it outwardly appears to be, while the whole situation is quite near the surface and with very little effort can be brought to conscious control.

Next lower in the scale of levels are the psychasthenic convulsions of Oppenheim (compulsion neurosis type) which are expressions of a more severe grade of neurosis but still within strictly psychological levels.

Then comes the very interesting affect epilepsies of Bratz and Leubuscher. These are distinctly epileptoid types of reaction conditioned by purely psychological situations. Here the outward semblance to a deeper level epilepsy is much greater but the situation is still a psychological one. The reaction of the patient here is to conditions that are absolutely intolerable and to which no adjustment is possible, such a situation, for example, as a young man has to confront when the key is turned upon him and he is called upon to realize that he is in prison with a life sentence to face. Under these circumstances the patient may become a veritable "wild man," beat his clenched fists against the bars, rush aimlessly about destroying clothes and bedding, and beat his head against the walls in ineffectual attempts at self-destruction. Hallucinatory disturbances may accompany these attacks and amnesia follow them, though consciousness during the attack is not entirely lost. That these patients are much more seriously burdened constitutionally and more nearly allied to "genuine epilepsy" than the psychasthenic types of Oppenheim is indicated by the fact that they give a history of "fits" in childhood while the psychasthenic types show tics, phobias, and compulsions.[1]

[1] See Galsworthy's drama, Justice.

In the classical epileptic seizure the greater severity and seriousness of the attack is indicated by the apparent complete loss of consciousness and the still further reduction in the purposeful and coördinated adjustment of the muscular reactions. These have now become utterly disorganized. The attack has involved far more than the psychological levels and include the sensorimotor and, as indicated by the toxicity of the secretions, the biochemical.

In terms of energy discharge the fit is a manifestation of energy at greatly reduced adaptive efficiency.

From the psychological point of view, the attack, inasmuch as unconsciousness is its most common symptom, may be considered as a *flight into unconsciousness* which occurs at times of stress such as an emotional crisis, or developmental crisis, such as puberty, which calls for all manner of new adjustments, physiological, psychological and social. The fit, therefore, is a break in the life demand for adaptation or an attempt at escape from an intolerable stimulus whether from within (toxin, tumor) or from without (life situation).

The actual disturbance of consciousness in the epileptic attack is perhaps not as profound as it appears. By a study of the various grades in severity of the attack from the mild petit mal to the severe grand mal attacks. Clark[1] comes to the conclusion that the disturbance is rather one of the quality of consciousness. The quality of consciousness which is lost is what might be called object-consciousness or awareness of the environment, while subject-consciousness or awareness of self is correspondingly greatly heightened. This condition of contraction of object-consciousness and increased self-awareness can be assumed from the content inferred from the productions during coma and deep lethargy. Though self-awareness is more intense it is so narrowed in range, in the more profound disturbances, as to be comparable to the consciousness at the time of birth or even before. As this acute self-awareness disappears following a grand mal attack there is an inflation of the unconscious which may result in acts of violence and automatism instinctively motivated and indicating an infantile feeling of all-powerfulness.

The low instinctive level to which the epileptic is reduced by his seizure can be appreciated by observing his activities as he is "coming out" of the attack. His respiration is at first distinctly abdominal (infantile type), he makes characteristic sucking movements with his lips, and his movements, from the complete disorganization into which they have been thrown, assume at first an aimless fumbling with his clothes, a tentative feeling about as he instinctively tries to readjust himself to reality, to "find himself" again. In this tentative "feeling about" he repeats in a few minutes the process of relating himself to reality which is a normal period of development in the child.

Ferenczi[2] has endeavored to classify the neuroses with reference to

[1] Remarks upon Consciousness in the Epileptic Fit, Boston Med. and Surg. Jour., 1921, vol. **185.**

[2] Entwicklungsstufen des Wirklichkeitssinnes, Internationale Ztschr. f. ärztliche Psychoanalyse, 1913, vol. **1.**

the stage of development they represent and in accordance with this scheme suggests that epilepsy belongs to the period of wish-fulfilment by means of incoördinate movements. It is known how some children when thwarted will cry out, thrash about and sometimes straighten out rigidly, "lose their breath" and become blue. Attacks of "temper" the mother calls them. Later on the child will kick the chairs and tear up its books under similar circumstances while regression to approximately the same level is shown when an adult stamps his foot, clenches his fists, grinds his teeth, and otherwise shows reactions of anger which are quite ineffectual to effect any change whatever in conditions. The meaning of it all is an absolute inability to accept or to adjust and an equally determined attitude that it *is not* so because it just *cannot* be so. An effort to force circumstances to be different by a supreme effort of thinking them different, which, when it fails results in a flight from the whole thing into the rigidity and unconsciousness of the epileptic seizure.

The phenomena of the fit at the immediately following period show it to be a profound regression to infantile levels. It might be likened to the *period of wish-fulfilment by means of incoördinate movements* (Ferenczi). In coming out of the attack the respiration is distinctly abdominal, sucking movements are quite characteristic, urine is frequently passed, and sometimes the patient expresses himself in baby talk, all testifying to the infantile setting of the attack in terms of function.

The extreme egocentricity of the epileptic, his great failure to project his interests into the outer world, his tendency, therefore, to retreat further and further from reality and to revive earlier ways of finding pleasure, results in a profound regression, which, in the unconsciousness of the fit, reproduces the helplessness of the child *in utero* and demands the same degree of absolutely complete care. Clark, who calls this a state of metro-erotism, has brought forward an abundance of material[1] to demonstrate that the fit has a psychological setting.

The seriousness of the classical epileptic convulsive reaction as compared, for example, with the hysterical type of convulsion is indicated by its involvement of the so-called partial tendencies. As has been indicated in the discussion of hysteria the split there is massive. A convulsion there, for example, may be found to reproduce a struggle against a sexual assault. In epilepsy, on the contrary, the split is much more profound and as seen by the symptoms, involves more particularly the muscle erotic plus perhaps the respiratory erotic as more or less isolated rather than as adequately integrated tendencies. A well developed musculature is quite common with typical epileptics as is also a feeling of muscular well-being. Equally characteristic of certain epileptics is a marked cruelty (sadistic) tendency. All these characteristics indicate a deep-seated disorder of the personality and so account for the difficulty that the problem of epilepsy presents from a therapeutic standpoint.

[1] Clark, L. P.: Clinical Studies in Epilepsy, Psychiatric Bulletin, January and April, 1916, **9**, Nos. 1 and 2.

As bearing upon the importance of the psychic element it is surprising to note how, as a result of acute questioning in intelligent subjects, it may very frequently be demonstrated that a particular fit served the purpose of the patient in some way, by enabling him to escape from some responsibility, avoid some necessity for adaptation. The way in which this is done, by a return to a condition demanding the sort of care which a mother gives her baby, and the assumption of the characteristics of infancy, Clark[1] illustrates by abundant material, as for example: talking baby talk, assumption of the fetal position, covering the head with the bedclothes, passing of urine during the attack, are all infantile types of conduct.

In the course of the introversion of the libido, until the fit occurs, Clark, in a recent chart for graphic recording of the epileptic reactions suggests that the degrees of regression are indicated as follows: spontaneous interest, directed interest, lethargy, irritation, anger, elation, psychic phenomena, petit mal, grand mal.

This way of viewing the epileptic attack is warranted because it follows along a path that proceeds from the known to the unknown. The mechanism at the higher psychological levels can be worked out, and although those at lower levels cannot, it would appear that the two extremes are connected by a regular series of intermediate stages as represented in types of cases. It need only be assumed that the disorder in "genuine epilepsy" has to do with adjustments at deep instinctive levels, adjustments that are profoundly biological in character and that the conflict may almost threaten life itself in order to understand the severity and seriousness of the attack as a reaction to failure. A study of the aura as a clue to the point at which blocking of energy commences, and a detailed analysis of all the elements of the attack plus an analysis of the make-up of the individual in the interparoxysmal period would seem to offer the mode of approach to a further understanding of the mechanisms in individual cases. The depth to which such an analysis would have to go and the severity of the constitutional burden in the classical types of the disease is indicated, for example, by the prevalence of the epigastric aura the characteristics of which indicate the possibility, at least, that the disorder reaches as deep as the vegetative nervous system level.

Recently Rosett has offered an hypothesis to account for the convulsion, particularly its physiological mechanism, on the basis of its analogy to decerebrate rigidity.[2] The convulsion, with its emphasis on the tonic contraction of the voluntary musculature. with a certain predominance of contraction of the extensors, adductors and pronators, gives the picture of decerebrate rigidity, so that considered from this point of view it would be an attack of functional decerebration.

The real significance of this hypothesis is seen more clearly from his discussion of this type of rigidity as under certain circumstances

[1] Loc. cit.

[2] Rosett, Joshua: The Mechanism and the Fundamental Cause of the Epilepsies, Arch. of Neurol. and Psychiatry, June, 1923, vol. **9**.

a perfectly normal type of reaction. Rigidity of the decerebrate type occurs normally under two sets of circumstances: first, as a result of an outside stimulus of great intensity which completely focuses the attention with a resulting great constriction of the field of consciousness. Pain and fear are such stimuli which immediately throw the individual into a state of generalized tonic contracture. The significance of this contractual state is that the muscles are all tensed or set, as it were, for maximum movement in any direction, so that the individual is in a position of maximum advantage to undertake, with the greatest rapidity, movements of offense or defense. Second, this state of rigidity is produced as the result of certain physiological functions. such as sleep, defecation, sneezing, coughing, and parturition. In these states the purpose of the muscular contraction is to fix the points of muscular attachment so that the function that is in progress, say parturition, can be adequately performed. It will be noted, too, that in such states there is also a pin-point contraction of the field of consciousness limited to the actual essentials of the performance of the function in hand.

The significance of the reduction of consciousness is that it is the outward symptom of the functional decerebration; in other words, that it indicates that the functions of the higher centers have been removed from their control of the lower centers, and that when this happens the lower centers act uncontrolled and the state of tonus results. The fit, then, from this point of view, would be divided as follows: First, a stage of disintegration of the conscious state, which may be long or short, and which includes the aura, and when prolonged the preconvulsive mental disturbances. Second, the stage of tonic rigidity, which is considered as an attack of functional decerebration. Third the clonic movements, which are explained as redistributions of muscular tonus. And fourth, the stage of recovery, which includes the postconvulsive mental disturbances.

This theory is quite consistent with the psychological theory outlined above, and as the psychological theory involves a progressive uncovering of older and more archaic methods of reaction as regression takes place during the attack, so this theory, at the physiological level, likewise involves a progressive uncovering of older and more archaic action pattern systems as functional decerebration progresses.

It can be seen from this discussion why the epileptic, burdened by deeply instinctive defects of biological adjustment, should deteriorate. It is because each attack is a concession to the cruder self and enriches the egoistic interests that the disease is progressive in character and tends toward dementia in the sense of a degradation of interests and capacities for adequate social contacts. It is also consistent with this view that, in general, this should not be true of symptomatic epilepsy. A localized lesion of the cortex, for example, may produce convulsive reactions in well-defined groups of muscles only without loss of consciousness (Jacksonian type). Here there is no defect of biological adjustment involving the individual as such. A group of muscles only

has been cut off from effective associational relationship with higher levels and so becomes reduced in its possibilities of reaction to relatively incoördinate, automatic and purposeless types. A portion only of the machinery has been damaged, the individual remains otherwise intact. The disorder is confined to the sensorimotor level.[1]

From this point of view it seems that the toxicity of the blood and urine is only an outward evidence of the depth of the disorder rather than an indication of its cause. It is true that certain toxic substances do produce convulsions, but they do it by damaging the machinery like the cortical lesions just referred to and the seizures cease with the removal of the poison. This is seen in severe forms of infection, febrile states in children, alcohol, uremia, etc.

It is interesting and significant to note, in this connection, that the form of dementia precox in which convulsive seizures are most prone to occur is the catatonic. In the other forms types of compromise formation take place while the catatonic endeavors to cut out, to enucleate, so to speak, a certain portion of the psyche, a condition much more favorable to blocking.

Varieties of Convulsive Attack.—As already indicated there is a great variety of convulsive attacks ranging all the way from that of purely psychogenic origin (hysteria), to the purely organic (post-traumatic, brain tumor). The Jacksonian type has already been referred to as has also the distinction between the grand and petit mal attacks and various equivalents (larvated or masked epilepsy).

In addition to *isolated seizures*, there are *serial attacks* in which several seizures follow each other at relatively short intervals and finally *status epilepticus* in which large numbers of attacks accumulate, following each other at short intervals until unconsciousness becomes continuous, the attacks then merging into one another—overlapping. The temperature rises in this condition, life is threatened and indeed it is the typical mode of death of the epileptic and his ever-present danger. Serial attacks are usually grand mal, but may be petit mal, while status, even if it begins as petit mal, soon takes on the seriousness of the major variety. Psychic seizures may also be serial and status attacks are possible without or with very minor convulsive manifestations. Status may, and frequently does, develop in the symptomatic epilepsies due to gross lesions of the brain as well as forming a frequent termination of the partial or incomplete seizures of the Jacksonian type.

Myoclonus epilepsy appears to be an association of myoclonia and epilepsy. It would seem that the myoclonic shocks, however, gradually eventuated by a process of summation into an epileptic seizure. Some epileptics have myoclonic shocks between their attacks which appear to be forerunners of the seizure rather than true forms of myoclonia.

[1] See Clark, L. P.: Nature and Pathogenesis of Epilepsy, New York Med. Jour., February, 1915, *et seq.*, for a complete statement of this viewpoint with numerous clinical proofs of its value.

Continuous Epilepsy.—Similar to the myoclonic varieties are the polyclonia epileptoides continua of Choroschko and the epilepsia corticalis continua of Koshewnikow. This is a condition of continuous myocloniform shocks in single muscle groups, usually unilateral and without loss of consciousness.

Narcolepsy.—Sudden attacks of sleepiness are usually psychogenic in origin and will often be found to disguise auto-erotic phantasying and to be compulsive in character.

Course and Prognosis.—Epilepsy is not a unitary concept. Numerous conditions are covered under the name, anatomical, etiological and symptomatic. It is better, therefore, here also, to speak of the reaction type as manifested clinically. The epileptic type of reaction tends to cripple the individual more and more. One can only become efficient in dealing with reality by constantly keeping in practice, as it were. To withdraw from difficult situations means less ability to meet the next problem that arises. The attacks therefore tend to become more frequent and, in a considerable number of cases to end in that permanent renunciation to efficient adjustment to reality—dementia.

Diagnosis.—From what has been said it can be seen that epilepsy is not an entity, that the term includes a great multitude of widely different conditions and that the problem of diagnosis is therefore the problem of differentiating the particular one of these several possibilities in an individual case. Diagnosis, however, does not mean giving a name to a thing, but understanding it. Every individual epileptic should be accepted as an individual problem and although it may often be quite impossible to fathom the mechanisms involved, it is only by such a method of approach that anything worth while can be hoped for when the question of treatment comes to be considered. Genuine epilepsy is a total life reaction.

Because of the frequency with which the epileptic attack is simulated, and because of the ease with which certain types of psychopaths develop convulsive reactions as a result of suggestion, and because of the multiplicity of convulsive types the diagnosis is often difficult. The personality make-up of the patient should be given prime consideration. The hysteric and the epileptic, for example, present very different types of character. In fact the personality make-up of the epileptic is sufficiently distinctive to warrant its recognition even when there is no history of seizures. Such individuals are potential epileptics—"epileptics without epilepsy."

The possibility of exclusively nocturnal attacks—*nocturnal epilepsy*—should be borne in mind. It is suspicious if the patient awakes tired and lame, as if his muscles had been beaten, particularly if he shows conjunctival ecchymoses, a wounded tongue and flecks of blood on the pillow. A localized muscular weakness that passes off promptly would add certainty to the diagnosis.

Treatment.—The only efficient prophylaxis is not to transmit the defective germ plasm. It would seem that in the purely symptomatic epilepsies, such as those due to cortical traumatism, that the germ

plasm might escape indictment, but this does not necessarily follow. A certain proportion of these cases will be found to have had convulsions in infancy so that they might have been considered as potential epileptics predisposed to react by convulsion-producing mechanisms. This possibility is emphasized by the frequency with which the symptomatic epilepsies develop status attacks.

Treatment of the Attack.—Once the attack has started it is essential to so care for the patient during his period of helplessness that he may not be injured in any way. If he has fallen in a safe place he may be allowed to remain there, perhaps only removing him from proximity to furniture or the wall against which his limbs might be injured as they are in the throes of the convulsive seizure. The clothing should be loosened about the neck to permit free breathing, and if possible a towel end rolled up and pressed between the teeth to prevent injury to the tongue. As a rule he should be permited to remain on his back or side, according to the position the contracted muscles force upon him. A wound received in falling may need care and a broken limb needs protection from the severity of the convulsive contractions to prevent additional injury by the broken ends. Epileptics, in general, should not be permitted to sleep unobserved or alone for fear they may roll over and smother during an attack. If vomiting occurs the patient should be rolled on his side and care should be exercised to prevent aspiration of the vomitus. In the automatic period following, watchful care is needed but direct efforts at control should be avoided if possible, as they are not understood and may only excite antagonism. The patient should not be permitted to get up until it is seen that no bones are broken. In this automatic state an attempt to walk on a broken leg might easily compound the fracture for consciousness is so reduced that pain would not be felt or reacted to.

Treatment of Status.—Serial attacks indicate possible danger of status and sedatives should be used to control their frequency, while sudden withdrawal of bromides should be avoided as tending to precipitate a number of seizures. Clark[1] recommends the following as an emergency prescription when status is threatened:

℞—Tr. opii deod. ♏v
Potas. bromid. gr. xxv
Chlor. hyd. gr. xx
Liq. morph. sulph. (U. S.) ʒj—M.
S.—One dose: repeat in two hours if necessary.

This prescription may be given after the first four or five seizures, after that sedation must be pushed to control the attacks as they are themselves a source of serious danger to life. Chloroform may be given in emergency by inhalation, but the most valuable of the drugs are chloral and the bromides. While it is necessary to push them, it must be constantly kept in mind that these very drugs are contra-indicated in the next, the stuporous stage, and therefore no more should

[1] Wm. P. Spratling: Epilepsy and its Treatment, Philadelphia, 1904.

be used than is actually necessary to control the situation as the following coma will be deepened thereby. Chloral and bromides are, as a rule, best given by rectum. The heart needs watching and may need stimulating, especially if large doses of chloral are administered.

In the stuporous stage the treatment is stimulating and supporting, careful nursing and feeding and protection during the great exhaustion.

Treatment between Attacks.—As there is no disease entity (epilepsy) there is no treatment that applies to all of the cases included under that term. Each of the various conditions requires treatment suited to it as does each individual require individual consideration.

The various surgical conditions, tumor, cyst, abscess, trauma, etc., require appropriate surgical intervention. In tumor, for example, as in other organic conditions, when the location of the trouble is not evident, a study of the attack together with the aura may give valuable evidence to guide the surgeon. It is desirable to have all such cases reside in a hospital long enough for their attacks to be accurately observed before operating.

Conditions of infection and toxemia require no special mention here In arteriosclerosis with softening the general condition overshadows the special manifestation as is also generally the case following hemorrhage. Conditions of marked arrest of development, either congenital, as due to serious birth injuries or early inflammations naturally offer little prospect for improvement.

Internal secretion imbalances should be corrected as far as possible, but for the most part little more than palliation can be expected, although the near future may well have something to offer in this realm.

Middle-ear disease should be adequately treated before meningeal symptoms and lateral sinus thrombosis take place.

The syphilitic meningitides, whether acquired or inherited, offer a promising field for relief by appropriate antisyphilitic treatment which should be intensive and include intravenous injections of salvarsan.

As to the pharmacotherapy of "genuine epilepsy" it can be inferred from what has already been said that there is none except such as may be called upon to meet or prevent emergencies such as status. Bromides have been so constantly used, however, that they need to be briefly discussed.

Bromide acts as a motor depressant—it raises the threshold of motor discharge and does therefore inhibit the convulsive expression. That it does this is sufficient explanation for its extensive use. Many popular medicines have much less to recommend them. When we realize, however, that the convulsion is not the disease, that it is not the cause nor a first expression even of the disease, but only its outward expression and the end-result at that, it may be well questioned whether bromide medication is rational. Experience seems to show that the fit postponed by bromides comer to pass ultimately any way and that the bromide may, in fact, operate unfavorably by tending to produce a summation of attacks and thus increase the danger of status. Added to this is the potentiality for disturbing digestion which the bromides

possess so prominently, so it may be said that they had best not be given at all unless under most carefully regulated conditions. The true function of the bromides is to control the convulsive manifestations when they, as such, become a source of danger as in serial attacks and threatened status.

Recently luminal, among the drugs, seems to hold the center of the stage. Reports of its use, are, for the most part favorable in reducing the number and severity of the attacks. It may be used with or without bromides or as luminal sodium. The usual dosage is about 0.3 gm. daily. Serum therapy has also been recently tried with reported good results. The serum is derived from animals injected with fluids from the patient.

Bearing in mind the theory of the essential epileptic attacks that has been elaborated, the rational treatment in all cases where the underlying mechanism cannot be unearthed (as in the symptomatic epilepsies), is to assist in the orderly discharge of energy, to help the process of sublimation. This is best effected by manual training, steady occupation graded to suit the intellectual level and other requirements of the patient and preferably conducted under institutional (colony) supervision.

As Clark[1] has shown, the disturbance of consciousness during the epileptic fit is an involution of the normal development of consciousness. Thus the fit has a deteriorating effect upon sustained interest and attention, the normal development of object-consciousness and objective interests. Each attack, therefore, tends to satisfy the cruder ego tendencies and enrich the egoistic interests so that the epileptic finally comes to be indifferent toward getting well. The therapeutic appeal must therefore be along lines of developing his interests and intensifying his personal satisfactions in ordinary living to the point where they will favorably compete with those derived from his epileptic regressions.

In many individuals much is to be expected from psychoanalysis. No results will follow from short treatment, however. The most favorable cases need from twelve to eighteen months. The treatment by this method is reëducational and consists in carefully regulating the entire life and the living conditions so as to slowly lead the patient into reality by arousing his interests in things outside himself, a gradual leading away from the egocentric fixation. It is in just such cases as these that much may be expected in the future from organotherapy in conjunction with a psychoanalytic handling of the personality problems. This latter must needs here as elsewhere often include a reëducation of the other members of the family whose attitude often serves to help to keep the patient ill.

Surgery of the colon is ineffective, unintelligent, and must be, in most cases at least, actually harmful. It is but another one of those attempts already referred to (hysteria) of trying to cut out the psychosis.

[1] Loc. cit.

The social position of the epileptic is most pitiable, often so impaired mentally that his labor is at a discount, he loses his job on the occasion of the first fit, even though he may have succeeded in getting one that is free from the dangers to which he is particularly exposed on account of his infirmity. He is thus seriously handicapped in the struggle for existence and barely gets to earning his livelihood in one position before he finds himself jobless again, without recommendation and forced to begin all over again. He is thus apt to be poorly nourished, poorly clothed and the subject of intense social repression operating from without that drives him back upon himself and aggravates greatly his trouble. He becomes discouraged and depressed and only too often takes to alcohol, the very worst possible thing he could do.

In the colony all these social handicaps are removed. Here he may have a fit in peace and comfort without feeling that he is disgraced or in imminent danger of losing his means of livelihood. This relief alone goes a long way toward permitting him the use of his avenues of expression and in bringing about a relative peace and quiet, so essential as a therapeutic adjuvant. If in addition to this he is trained in some form of healthy occupation, preferably outdoor, that is interesting and affords an added means of expression, the best possible has been done for him. In the colony, too, he is provided a home, congenial surroundings, a regulated diet and is under that careful and continuous skilled supervision for a prolonged time which is so essential to the best results. Under colony care Spratling thinks 5 per cent. of cases as they come can be cured and that this percentage could be doubled or perhaps trebled if all the cases could be gotten under treatment early.

CHAPTER XIX.

DEMENTIA PRECOX (SCHIZOPHRENIA) GROUP.

The term dementia precox has been the occasion of a great deal of discussion. Coming into general use as it did as the result of the studies of the Kraepelinian school it was conceived to apply to a group of psychoses belonging to the period of adolescence and presenting dementia as a fundamental element in the symptom picture. When, however, it was seen that what appeared to be the same syndrome might occur later in life, even after thirty years of age, it seemed hardly proper to use the term precox as applied to a psychosis of early life. It was therefore proposed that the term precox should refer not to the age of the patient, but to the relatively early appearance of dementia in the course of the disease. The term dementia was here used to mean a permanent mental impairment, and when it was realized that many cases made good recoveries without any apparent or at least material defect remaining, another reason was evident for the inapplicability of the term. The concept, then, might be formulated, that it was a syndrome in which dementia was a relatively early symptom and that the recoveries occurred only when the disease had not progressed to any extent. This also, unfortunately, does not meet the facts, because many cases get well after prolonged and apparently chronic courses. In the absence of any well defined criteria of dementia it was impossible to predict when it was or was not present, and therefore the term presents very many undesirable features. Although it is somewhat of a bootless task to discuss names, and although it is much more important to know what the names stand for than to quibble about their applicability, still it is, of course, desirable to have a name that fairly represents the thing named. To meet this demand Bleuler has suggested the name schizophrenia, implying a splitting of the personality, which he thinks is the fundamental symptom. Although this term as used by Bleuler includes a number of conditions that many psychiatrists would object to as being included in the dementia precox concept, still it is generally conceded that the splitting of the personality, as indicated by the name, is fundamental in this group, and the name is coming into gradually more and more general use.

Dementia precox must undoubtedly have always existed and have been observed by physicians, and in particular the grotesque cases of catatonic rigidity and peculiar mannerisms must have always attracted attention. In the early history of psychiatry, however, few descriptions of cases exist that could be unequivocally said to be

cases of precox, as the group had not been defined from other groups superficially resembling it, as for example, imbecility. Willis, the English anatomist, recognized as early as 1672 that many young people underwent deterioration and Sydenham, a hundred years later, in 1772, describes similar conditions under the description of stupidity, while later on, after mania and melancholia had been more or less defined, many of the excitements and depressions that are incident to the course of dementia precox were undoubtedly grouped under these headings, while at one period, only a few years ago, there was a distinct group supposedly representing a special disease described by the name of catalepsy, where also undoubtedly a certain number of precox cases were arranged.

At the present time three pretty well defined groups of cases are included in the general concept of dementia precox, namely, the hebephrenic, the catatonic and the paranoid. Kahlbaum was the first to describe hebephrenia as a disease entity in 1863 and in 1871 his pupil, Hecker, published some excellent descriptions of this disease. In 1869 Kahlbaum described catatonia under the term Spannungsirresein or vesania catatonica, of which he gave an admirable monographic description in 1874. In 1896 Kraepelin, in the fifth edition of his Lehrbuch, arranged dementia precox, catatonia and dementia paranoides as disorders of metabolism. Clouston, the Scotch psychiatrist, had already described what he termed adolescent insanity and objected to the term dementia precox as being too inclusive. Kraepelin, however, worked over his material with great thoroughness and arrived at the concept that includes the three forms, hebephrenic, catatonic and paranoid by tracing the life histories of his patients and grouping all these cases, however dissimilar they might appear on the surface, from the standpoint of prognosis. They were cases that had a fairly definite course and outcome, eventuating always in a certain degree of dementia.[1]

Etiology.—The question of *heredity* in precox has been studied, by Wolfsohn,[2] who carefully analyzed the material from this standpoint at the Berghölzi asylum in Zurich. The study of 2215 admissions disclosed 647 cases of dementia precox of whom 90 per cent. showed hereditary taint. Of four factors, mental disease was the most frequent—about 64 per cent.—followed by nervous diseases, alcoholism and other forms of hereditary taint. Heredity was combined in 34 per cent. The most frequent combinations were those of psychoses and alcoholism, and psychoses and nervous disease. She concluded that a distinct influence of heredity could not be proved in the cases in which the taint was alcoholism, nervous disease, or other forms. The catatonic was the most and paranoid the least affected by the mental taint, while the influence of the taint had no striking effect on the character of the first symptoms of the disease.

[1] Jelliffe: Dementia Precox, a Historical Summary, New York Med. Jour., 1912.

[2] Die Heredität bei Dementia Precox, Allg. Ztschr. f. Psych., 1907, Band **64**, Heft 2 and 3.

Boven[1] studied all the ascendants of 20 manic-depressives and 30 patients with dementia precox. In the structure of the personality he believes that the abnormal characteristics play an important role in reference to the psychosis developed; in manic-depressive psychosis taking the form of a predisposition to sadness and in dementia precox of a predisposition to unsociability. Only after an accumulation of defects in the inherited psychic structure the familial psychoplasm, does the psychosis establish itself. In dementia precox there is always found a union of two families with hereditary taint whereby, even in childhood, the pathological features due to the heredity come to light, *i. e.*, first as shyness, etc., and later as unsociability. If in the family there is a taint of both dementia precox and manic-depressive predisposition then in the descendents either both diseases or a combination makes its appearance. These cases cannot be considered as belonging exclusively to either disease, and in reality there is a mixed psychoplasm. On this base, cases arise, for example, of paranoid melancholia, sullen mania, cyclic dementia precox.

It would seem that there is a certain element of direct heredity, inasmuch as families are found in which several cases of precox occur, just as there are families found in which several cases of the manic-depressive psychosis occur.

The most elaborate study of the hereditary factors in dementia precox is that of Rüdin.[2] His study is based upon the conceptions formulated by Kraepelin.

A study of 701 cases from parents both of whom were free of the disease gives the percentage as 4.48 or from $\frac{1}{32}$ to $\frac{1}{16}$ of all the children living or dead. Dementia precox does not behave like a simple Mendelian factor in heredity. In view of the percentage which closely approximates $\frac{1}{16}$ it is not improbable that a dihybrid crossing mechanism is involved in which two concurrent factors enter into action. The analysis of 34 patients with one precox and one non-precox parent gives a percentage of 6.18. The small material forbids a final judgment but points strongly to the conclusion that a Mendelian recessive factor is in evidence. When both parents are free from precox, but one parent has some other psychosis the percentage is 8.21. When one parent has some other psychosis and there is alcoholism on one or both sides the percentage is 15.78. When both parents are psychotic, independent of type, with or without concurrent alcoholism the percentage of children with dementia precox was 22.7. A study of the children of 20 old dementia precox patients disclosed only 2 of the 81 to have dementia precox. If precox was due to a Mendelian dominant at least 50 per cent. should have developed the disease. Males and females are equally involved. Anticipation, that is, the earlier appearance of the disease in succeeding generations, occurs,

[1] Étude sur les conditions du développement au sein des familles, de la schizophrénie et de la folie maniaque-dépressive, Schweiz. Arch. f. Neurol. u. Psychiatr., 1921, **8**, H. 1.

[2] Studien über Vererbung und Entstehung geistiger Störungen. (Zur Vererbung und Neuenstehung der Dementia Præcox) pub. by Julius Springer, Berlin, 1916.

the anticipation age averaging about six and a half years. Whether this is a real law or a statistical artefact, Rüdin thinks is open to question. Finally statistical evidence is unable to decide whether a polymorphous trend or a general disposition to mental disturbance only lies in inheritance.

Bleuler[1] criticizes Rüdin's conclusions as too statistical, rigid and inflexible and as not taking into account sufficiently latent, undeveloped cases who do not get into institutions, who are unrecognized or who have not been subjected to sufficient stress to develop the schizophrenic tendency. Heredity studies are not as yet able to define what is inherited. The clinical delimitation of the disease and the significance of its various symptoms are as yet too vague to warrant clear cut conclusions. Studies should take into account the sound members of family groups as well as the diseased. The whole question is probably infinitely more complex than even Rüdin's investigations would indicate. The inheritance factors in mental disease may well be manifold and variously related.

Of the exciting causes severe shocks, both mental and physical, are not infrequently found, as for example, severe hemorrhages and infections following parturition. In the latter cases precox breakdowns occur as a result of that train of emotional disturbances which follows upon seduction and desertion.

For a number of years there has been a tendency to ascribe the disease to disturbances of metabolism with possible toxic factors and to suppose that its origin might be traced to disorders of the glandular secretions, more particularly of late, of the internal secretions of the ductless glands and inasmuch as the disease tends to focalize about the period of puberty and adolescence, it has been supposed that perhaps the testicles and the ovaries might be the offending organs. This theory has received confirmation, more especially by Fauser, who, working with the Abderhalden technic has isolated defensive ferments against the gonads and the cortex. More will be said of this matter later when the discussion of the nature of the disease is taken up, but it may be mentioned here that whatever its ultimate nature may be the existence of toxic factors, or internal secretory disturbances is largely hypothetical so that at the present time it is more useful to formulate the upsetting factors as well as the general symptomatology in psychological terms rather than in terms descriptive of disturbances at physicochemical levels. In any case toxic factors may as well be effects as causes or perhaps concomitant factors in a vicious circle.

The formulation of the disease in terms of the affects or of complexes or, in accordance with Meyer,[2] continued unhealthy biological reactions, or as an outgrowth of a "shut in" character is after all more of an effort at description of what is found. Everyone has complexes,

[1] Mendelismus bei Psychosen, speciell bei der Schizophrenie, Schweiz. Arch. f. Neurol. u. Psych., 1917, **1**, No. 1.

[2] Fundamental Conceptions of Dementia Precox, Brit. Med. Jour., September 29, 1906.

but it is not clear why in certain cases they lead to the development of a precox psychosis, while a "shut in" character might itself in certain cases at least be considered to be an early expression of the disease process, a latent precox perhaps, in the sense of Bleuler.[1]

Recently von Monakow and Kitabayashi,[2] as a result of exhaustive studies of the choroid plexus believe that plays a considerable role in the development of precox. Aside from its function of secreting cerebrospinal fluid they believe it acts as a selective filter for the products of metabolism and endocrine gland secretions. Its function therefore is to neutralize, detoxicate, or transmit the products of internal secretion so far as they have to do with brain function. Emotional upsets, the maintenance of strong passions for long periods, attended, as they are, by excess productions of the glandular secretions, make heavy demands upon the plexuses. In all of the 12 cases they examined degenerative changes were found in the plexuses. They believe that in all similar conditions diseased conditions of the plexuses will be found and that all higher mental processes are the product of the continuous coöperation of the glandular and central nervous systems, particularly the cortex.

Symptoms.—*Mental.*—A patient from time to time writes letters appealing for his discharge and his liberty and signing himself "The Emperor." It is this incongruity, this lack of oneness of the individual that for a long time has attracted attention in the symptomatology of this disease. How is it possible for a person so exalted as to think himself an emperor to plead in quite a natural way for his discharge from an asylum? The two positions which the man takes, acknowledging himself as a patient and pleading for his discharge while still proclaiming himself an emperor, show the possibility of maintaining two distinct and mutually opposed trends of thought at the same time without the one apparently interfering or serving at all to correct or modify the other.

This possibility is dependent upon a fundamental associational disturbance which has caused Bleuler to see a *splitting of the personality* as the foundation symptom of the disease and to give it the name, therefore, of schizophrenia. This splitting of the personality has been expressed less clearly in many of the theories that have been advanced to account for the symptoms, while a massive splitting of the personality has long been described as a fundamental symptom of hysteria under the designation of dissociation. The symptoms have been said to be dependent upon a disintegration of the personality, a disintegration of consciousness with the consequent impairment of the function of the real. Reality is unable to correct or adequately modify the delusional ideas, with the result that such bizarre, strange and

[1] Bleuler: Die Schizophrenie, 1911; also see Bleuler: Lehrbuch der Psychiatrie, 1921, 3d Edit; Kraepelin: Lehrbuch der Psychiatrie, 8 Edit.

[2] Von Monakow, C. and Katabayashi, S.: Schizophrenie und Plexus choroidei, Schweiz. Arch. f. Neurol. u. Psychiat, 1919, vol. **4**.

apparently "*unpsychological,*" "*incomprehensible*" modifications of conduct are seen.

These unpsychological appearances are dependent in part, upon what Bleuler calls *autistic thinking,* that is, a form of thinking to which Jung would apply the term introverted, in which the individual's interests are withdrawn from reality, and he occupies himself with himself to the more or less complete exclusion of the outside world. This is the field of dream formation, of phantasies, wherein things wished for come true. An analysis of cases of dementia precox shows that the delusion formations are based upon wish-fulfilling mechanisms which result oftentimes in highly symbolic, and to the observer, non-understandable expressions which are formulated in accordance with the particular complexes which may be operative. These mechanisms are the same as those observed in normal people, in hysterics, and those suffering from the various neuroses, but it is impossible for these individuals to adequately utilize them; they therefore result in impairment of efficiency and withdrawal from the world of reality. It would seem, too, that in precox the regression is very much more profound than in the neuroses and the psychoneuroses, and for some unknown reason involves a serious disintegration of the personality which tends to become chronic and crippling. From the standpoint of this schizophrenic splitting of the psyche, based upon autistic thinking, many points in the symptomatology of the disease become understandable.

The unpsychological and incomprehensible character of the ideas and conduct of the precox depend upon several factors. The strange content is either highly disguised, symbolic material or it may be the crude breaking through of the unconscious. In the latter case it is quite as incomprehensible as in the former. An infant soiling itself is quite understandable, but the same act in an adult setting puts us quite beyond our ability to feel ourselves into a sympathetic attitude of understanding. Distortion serves further to make much of the material seem strange while the fact that the regression reaches to archaic levels is another factor of importance. All these factors produce a picture characterized by the outcropping, here and there, of unconscious material, distorted or not. The net result of such mechanisms is a series of psychological manifestations which seem to have lost their unity. They no longer seem to be connected, to be severally parts of a coördinated whole. This appearance of *fragmentation of the psyche* is in marked contrast to hysteria in which a disease state posseses a unity of its own. The productions of precox seem to be disconnected, to lack coherence, to be, in fact, incoherent. This incoherence is only apparent, however, and not real. If the language of the precox could be read it would be found to hang together though to outward appearance it might not seem to.

The peculiar *emotional dulness* and uncertainty of emotional response of the precox has long been noticed, and Stransky[1] has par-

[1] Ueber die Dementia Precox, Streifzüge durch Klinik und Psychopathologie, Verlag von T. F. Bergmann: Wiesbaden, 1909.

ticularly designated it by the term *intrapsychic ataxia*, by which he means a disturbance of the coördination between the intellectual and the affective attributes of the psyche, which are respectively known as the noopsyche and the thymopsyche. This noothymopsychic ataxia gives the appearance at times of emotional dulling and at other times of a senseless emotional reaction. Thus a patient who receives news of the death of a near relative unmoved, may a little later on laugh heartily at apparently nothing. He is happy when he should be sad, sad when he should be happy, angry without cause, experiencing fear without reason, for the most part cold and impassive, but occasionally showing outbursts of marked and accentuated emotionalism. This is the "April weather" behavior of the affects, in the language of Stransky.

The mood swing, the *psychesthetic proportion*, according to Kretschmer is between hyperesthetic (sensitive) and anesthetic (cold).

The lack of conformity of the emotional reaction and the idea content, *affective dysharmony*, is shown not only with reference to a given time but in relations of succession. Moods and affects change in all possible ways without visible inner or outside causes. Here we see the same ataxic tendency. The April-weather behavior of the affects and moods suggests the relationship with hysteria.

This dysharmony exists only on the surface. The key to the situation lies in the unconscious. The affect which is out of harmony with what appears on the surface is really in harmony with what is going on in the unconscious.

This condition of affairs is understandable upon the basis of the splitting of the psyche. The emotional reactions occur when the complexes have been touched, which the individual is constantly in an attitude of trying to prevent. This method of dealing with the emotions is well known, particularly in the formation of the dream, in which the *mechanism of displacement*, by removing the affect from the constellation of ideas to which it belongs and attaching it to an indifferent set of ideas serves to hide from the patient the realization of the actual difficulty. For example, a patient identifies herself with a schoolmate of her's and then accuses the schoolmate of being "bad" and pregnant. It will be easily seen that in such a case the patient is protecting herself from the realization that she has thoughts to which the term "bad" might be applied, and that the pregnancy which might result from being bad is transferred to her schoolmate. Under such circumstances, she, so to speak, unloads her emotion upon this schoolmate and very easily may produce the impression of indifference toward herself with an unmotived affective attitude toward the schoolmate.

This *withdrawal from reality*, this looking within, occupying themselves with themselves, no longer subject to the corrective influences of the outside world, produces many surface indications, among which are *failures of voluntary attention, lack of interest, disturbances of orientation, disorders of memory*. The disorders of attention, lack of

interest, failure of voluntary attention can easily be seen to be due to the turning of the interests within. The capacity for attention may be as keen as ever, but the patients are not attending to the things going on about them, but rather to the things going on within, and so they apparently take no interest in the people or the events of their environment. They may even express themselves as perfectly satisfied with their confinement in a hospital, and be so manifestly heedless of those about them that it is practically impossible to draw them into conversation. This lack of interest and attention naturally produces what appear to be disturbances of memory and orientation. The patient, who is heedless of his surroundings, may easily not know the day of the week or may have forgotten the events that only recently took place about him, because they were not sufficiently attended to to make any profound impression, while a patient who identifies himself with some great public functionary might easily not give the correct date of his own birth. Such considerations as these show how necessary it is to penetrate beneath the surface indications which the patient manifests and find out their true meaning. They also indicate unequivocally that what may appear quite unpsychological is really perfectly understandable.

The same type of explanation serves to render clear the meaning of such surface indications as the *shallowness of thinking* and the apparent *dilapidation of thought.* The interests and the energies are occupied with things which are not accessible to casual questioning. In fact, as will later on be seen, the patient may be wholly inaccessible to any form of approach, and when there is considerable speech productivity what is said may be so incoherent upon the surface as to be quite non-understandable and constitute what has been called a "word salad."

The *delusional formation* of precox is notoriously grotesque and partakes of this characteristic to such an extent in harmony with the grotesqueness of the thoughts in dreams that the similarity has not been overlooked. The mechanisms, too, are probably quite the same, although much more difficult to fathom because of the inacessibility and lack of coöperation of the patient. If the eye is properly trained, however, to see meaning in the apparently meaningless, there will be little difficulty in seeing how certain expressions may be interpreted, even though in an individual case it may be impossible to verify such an interpretation. For example, an old precox who talked in a thoroughly dilapidated manner but was able with some patience to give a fairly good account of himself, injected into his series of replies to questions that the President was confined in an almshouse and that he had come to Washington to be President. He also stated that he had had something to do with his sister when he was a young boy, that he did not tell the priest and that his shadow was very heavy, that it was black and that he saw the Devil in it. Here an expression of grandiose ideas which, speaking generally, may be considered as compensatory, is seen. In his autistic thinking he

wishes to be the great man that in his real life he is not. Then one sees the possibility of a serious moral conflict, the result of incestuous relations with, or perhaps only incestuous thoughts about, his sister, while his dark and heavy shadow in which he sees the Devil can be easily seen to be a symbolic representation of the destructive effects which his moral delinquencies have had upon him. Here one also sees that the *hallucinatory experiences, the disorders of the sensorium* express themselves as *symbolisms of the conflict* and receive their interpretation with a knowledge of the nature of that conflict.

The delusions are essentially endogenous in origin, that is, ultimately dependent upon factors that are within the individual, and they tend to be colored and determined by complexes which lie at the very foundation of the personality, which have to do with the region of the psyche which has long since been forgotten and to which Freud gives the name "unconscious." It is largely because of their profoundly unconscious origin that they are inaccessible and it is largely owing to this also that they produce such serious disturbances and such marked departures from conduct that is at all efficient.

It is, too, because the motivation of the activities of the patient and the origin of his delusions are in the unconscious that his conduct and productions strike us as unpsychological. That which originates in the unconscious, because its origin is unknown, seems strange and mysterious.

Just as the hallucinations are symbolisms of the elements in the conflict so are the various *mannerisms, stereotypies* and *neologisms.* All of these motor disturbances are in effect *symptom activities, complex indicators,* and serve in some way to portray the nature of the conflict. In a recently reported case, for example, an old precox was observed to keep pounding one hand with her clenched fist in a rhythmic stereotyped fashion. It was discovered that in her earlier days she had been jilted by a shoemaker. This peculiar action could be seen, in the light of this knowledge, as but the movements of the shoemaker pounding at his last. Many of the old cases of precox have such stereotyped activities which it is often, in fact usually, quite impossible to fathom, but in the light of such a case as this it will be seen that they must all be looked upon as having meaning and that for a complete reconstruction of the psychosis it is necessary to determine what that meaning is.

As an example of what painstaking analysis may disclose may be mentioned a patient of Jung's. She expressed herself in stereotyped and apparently meaningless phrases interspersed with neologisms. One of her statements was: "I affirm a million Hufeland to the left on the last fragment of earth on the hill above." A detailed analysis disclosed this sentence to mean, approximately: "For the bad treatment of the physicians which I have to endure here and with which I am tortured to death I claim a high indemnity."

Negativism, one of the characteristic symptoms of precox, may be expressed passively by the patient's not doing what is expected of

him, or what he should do, or by actively doing the exact opposite of what is requested. The passive negativism may show itself in refusal to attend to the promptings of normal desires, so that the bladder and the rectum are permitted to become overloaded and the saliva to collect in large quantities in the mouth, or, on the other hand, active negativism may show itself in the patient doing the exact opposite of what he is asked to do; for example, if he is asked to shut his eyes he opens them wider and if he is asked to open them he will shut them tightly, and if he is asked to put out the tongue he shuts his lips tightly, and if he is asked to shut his mouth he opens it, etc. This peculiar symptom is dependent upon what Bleuler terms the *ambivalency of ideas*, by which he means that every idea has connected with it by association its exact opposite more intimately than other ideas, and so the patient who is withdrawn from reality and objects to being invaded by the world of real things, who would, in other words, live within himself, finds that in refusing to accord with a suggestion from the outer world the path of the diametrically opposite reaction, is more patent, more accessible than any other. This is a type of reaction which one sees also in other conditions, as in hysteria, and also quite naturally in children.

The *suggestibility* of the precox receives a similar explanation. Some patients find it easier to follow blindly any suggestion which comes to them from any source than to actively initiate contact with reality. One precox patient, for example, had to be fed by placing his food before him and a spoon in his hand and repeating each time the command to take another mouthful. After having responded to the command he remained inert until it was repeated. This form of activity requires no initiative on the part of the patient, no actual effective contact with reality of his own devising and permits him to remain within himself, much as does the negativistic tendency.

The *catatonic rigidity* of the precox is a still more active shutting out of the world to the point of absolute inattention to the environment, but an inattention which is positive and active rather than passive, as in catalepsy and command automatism, while *stupor* still more effectively shuts out the world of real things.

There is a reaction type in dementia precox which is important for an understanding of the nature of the disease. It is the *archaic type of reaction*.[1] In the illustrations that have been given it is seen that when the individual under the influence of mental disease regresses he not only reaches lower levels but frequently reaches levels corresponding with his early infancy, and so it is frequently found that the delusions and other morbid manifestations only find their explanation when traced back to the infantile period. In the same way regressions may lead back to lower cultural levels so that patients show symptoms that are understandable in terms of the psychology of primitive peoples. The materials of experience are used by the patient in an

[1] White, W. A.: Foundations of Psychiatry, Nerv. and Ment. Dis. Pub. Co., Monograph, No. 32, 1921.

archaic way. One such patient with a very complicated delusional system states that he is the father of Adam, that he has lived in his present human body thirty-five years, but in other bodies thirty million years, and that during this time he has occupied six million different bodies. He has been the great men in the history of the development of the human race; he himself created the human race; it took him three hundred million years to perfect the first fully developed human being; he is both male and female and identifies all the different parts of the universe with his own body; Heaven, Hell and Purgatory are located in his limbs, the stars are pieces of his body which have been torn apart by torture and persecution in various ages of past history; he is the father and creator of the various races and elements of the human organization, etc. Here is a very primitive type of thinking in which the patient identifies himself with the whole universe somewhat as the baby does and somewhat as primitive man does. He is quite in the position of the chief of some primitive tribes in whom all the forces of the universe center and from whom radiate all of these forces for good or for ill. This is the archaic type of reaction which shows how deep the split of the personality may be, how fundamental it may be and gives an insight into the seriousness of the disease process.

This archaic type of reaction results from regression, or in other words an *introversion of the libido* which is the exact opposite of what is found to be the case in the manic-depressive psychosis. Dementia precox is an *introversion psychosis*.[1]

Physical.—A considerable number of precox patients, particularly the catatonic and the hebephrenic, show marked physical symptoms, and not infrequently have all the outward appearances of being quite ill. They often emaciate during the early period of their illness, suffer from anorexia and insomnia, circulatory disturbances, disturbed cardiac action, cyanosis of the extremities, vasomotor disorders of which dermographia is not an infrequent manifestation. The deep reflexes are commonly exaggerated, while the pupils in this class of cases are characteristically widely dilated. *Convulsive seizures* of an epileptiform, but more often of an hysteriform, variety may occur. The nature of these seizures is not clear. They may be an expression of the disease process in dementia precox, they may be epileptic psychoses with catatonic coloring, or they may be a combination of epilepsy and precox.

Vorkastner,[2] regarding only those cases as genuine epilepsy when no etiology can be discovered, and when etiology is in evidence regarding the epilepsy as symptomatic found only 2 cases of the symptomatic variety in 217 cases of precox. Even when doubtful cases, fainting fits and vertigo were counted in there was only 4.6 per cent. which is essentially lower than that of Urstein and Kraepelin. From a long

[1] Jung: Psychology of Dementia Precox, Nervous and Mental Disease Monograph Series No. 3.

[2] Epilepsie und Dementia Præcox, Beiheft z. Monatschr. f. Psych. u. Neurol., 1918.

series of cases he assumes that there is not infrequently a combination of the two diseases, precox and epilepsy, if the previous history, especially the prepsychotic period is taken into account. Such confusion as to the significance of the clinical material must necessarily continue so long as epilepsy and precox are considered as entities and so long as a functional and dynamic viewpoint which considers processes and mechanisms is avoided.

In the very early stages of the disease physical symptoms which do not lead to the suspicion of mental disturbance are not infrequent. Such symptoms as headache may be in evidence for a considerable time as may also vertigo, and Urstein[1] has called special attention to the occurrence of gastric disturbances. Other physical disturbances may also of course occur, and if no adequate foundation can be found for them a mental origin should be thought of.

It is this type of physical illness that has led to and maintained the belief in etiological factors at the biochemical level.

The Abderhalden method of research, especially as applied by Fauser, has indicted the adrenal, the thyroid and the gonads. A number of investigators have also found a reaction to brain tissue (cortex).[2]

Symptoms of disorder of the various viscera not infrequently are noted in the prodromal period of the disease and are treated for some time before the psychosis is sufficiently in evidence to force recognition. Eppinger and Hess find a tendency to instability of both parts of the vegetative nervous system and many of the gastro-intestinal, cardiac, circulatory and cutaneous conditions are dependent upon disturbances at this level. Vagotonic symptoms are not infrequently in evidence. Laignel-Lavastine records pathological changes in the sympathetic ganglia, notes the commonly observed fact of the frequency of death from pulmonary tuberculosis and observes that the precox often has a small heart. Many other abnormal conditions have been found but their correlation to the larger concept of precox is not yet clear.

In connection with this whole matter of defective organs Adlers' concept[3] of the part they play at the psychological level is helpful. The psyche is the region where the final correlations and integrations of all the reacting levels find final expression so that a badly constructed body must express itself at the psychological level in defects of adjustment. The correlation of the psychological symptoms with the several organ defects is not always possible in the present state of our knowledge. Evidence is accumulating, however, to indicate that ultimately we may expect a rich symptomatology at the vegetative level (endocrine glands and vegetative nervous system) and that many of the functional physiological disturbances and even organic changes (cortical

[1] Die Dementia Precox und ihre Stellung zum manisch-depressiven Irresein, Berlin u. Wien, 1909.

[2] Lessing, O. Innere Sekretion und Dementia præcox, S. Karger, Berlin, 1921.

[3] Adler: Organ Inferiority, Nerv. and Ment. Dis. Monograph Series, No. 24.

aplasias) will find their explanation in anomalies at this level. We are just beginning to glimpse the possibilities of correlating all these disparate symptoms into a comprehensive and satisfying concept.

Bodily Types.—Kretschmer[1] has described two main types of bodily configuration that seem to be closely correlated with the schizoid make-up: Their characteristics are briefly as follows:

Asthenic Type.—Characterized by a deficiency in thickness, but not in length, of all parts of the body—face, neck, trunk, extremities, and with similar deficiencies in skin, fat, muscle, bone and vascular system. The chest is long, narrow and flat, the ribs are easily counted and the rib-angle sharp. The stomach is thin and devoid of fat. The skull is small, 55.3 cm. in circumference, short (sagittal diameter 18.0) and broad (15.6). The face from the front is a shortened egg form: in profile it is angular with long nose and hypoplastic under jaw.

Athletic Type.—Characterized by the strong development of skeleton, musculature and skin. The man is middle-sized to tall, with wide projecting shoulders, superb chest, firm stomach, a trunk which tapers to a narrow waist and magnificent legs. The head is long, carried upright on a free neck with a firm trapezius with a sloping linear contour from the front. There is marked bone as well as muscle relief. Next to the shoulders the trophic accent is on the extremities which may be reminiscent of acromegaly. The face, from the front, is steep egg-shaped with marked bone-relief and thick, firm skin, The skull circumference is medium, on the average high, narrow, and of a fair medium length. The back of the head is sometimes strikingly steep, sometimes projecting. Sometimes there is a tendency to tower skull. The face in profile is prevailingly heavily boned, with snubbed nose, well-molded chin and a gently curving profile line. The face is long, the chin usually long.

There are many variations from these specifications and many types are mixtures of the two. He also describes a *dysplastic type* which contains a number of subgroups indicating glandular anomalies, particularly the dysgenital group.

Mode of Onset.—The early manifestations of precox vary greatly. As already indicated the disease may remain latent for a considerable period, manifesting itself only in slight disturbances, predominantly of a physical nature, such as headaches and gastro-intestinal disorders. In quite a large percentage of cases special types of character make-up have been found to have existed for a considerable period before the outbreak of the symptoms, in fact to have been characteristic of the individuals before the break-down. Whether these are to be considered as symptoms of the disorder or as expressions of the type of individual in whom the disorder is possible cannot be answered at this time.

Kraepelin has described four such types of character make-up: (1) A quiet, shy, retiring disposition, making no friendships and living only for themselves. This type was found mostly in boys. (2) An

[1] Physique and Character.

irritable, sensitive, excitable, nervous, self-willed type with a tendency to bigotry, observed mostly in girls. (3) A smaller group of children, mostly boys, who from childhood up are lazy, restless, dislike work, inclined to nasty tricks, do not persevere anywhere, and become vagrants or criminals. (4) In contrast to these are those, mostly of the male sex, who were conspicuous for their docility, good nature, anxious conscientiousness and diligence, and as patterns of goodness held themselves aloof from all childish naughtiness. These types Künkel[1] names the *autistic*, the *unstable*, the *asocial*, and the *pedantic* respectively. He thinks these syndromes are to be regarded as early manifestations of the disease and so sets down the beginnings of schizophrenia to a very early age. The disease thus falls into three phases, the infantile, the adult, and the senile or a childhood syndrome, the frank psychosis, and the terminal stage..

In sharp contrast to these latent periods the disorder develops not infrequently with great suddenness. After some emotional shock the patient becomes almost immediately greatly confused or catatonic.

It is quite frequent to have the breakdown be a slowly progressive, developing condition. During the early period the symptoms may take all sorts of forms and may easily be mistaken for types of manic-depressive psychosis, compulsion neuroses, neurasthenia, hysteria, hypochondria, acute confusion and paranoid states. Anything atypical in these psychoses should make one think of the possibility of dementia precox.

Symptom Groups.—The varieties of dementia precox will be described under five heads: I. Dementia Simplex; II. Hebephrenia; III. Catatonia; IV. Paranoid Forms; V. Mixed and Atypical Forms.

I. *Dementia Simplex.*—In this group of cases the origin is usually insidious, perhaps manifesting itself only by slight physical disturbances, such as headache, gastro-intestinal attacks, by some irritability, and perhaps a tendency to withdraw from the association of others. The patient may suffer from insomnia, perhaps disagreeable dreams, and there may be passing evidences of hallucinations and delusions. These are apt to be expressed only at times, perhaps under the influence of a little excitement, and show little tendency to organization or progression.

The patient emotionally is more apt to be somewhat depressed, or at least indifferent, lacking in initiative and presenting outwardly an appearance that frequently leads to the diagnosis of a "nervous breakdown," "nervous prostration," or neurasthenia.

Such mild attacks as this may be pretty well recovered from but may recur and one not infrequently finds a history of one or more such attacks having preceded a more severe breakdown.

It is this group of cases of mild abortive forms, *formes frustes* of the French, which after recovery from a "flurry" or "excited epi-

[1] Künkel, F. W.: Die Kindheitsentwicklung der Schizophrenen. Monatsschr. f. Psych. u. Neurol., 1920, vol. **48**.

sode," gives one the impression of "peculiar characters." Many such cases are found in the ranks of the criminals, hoboes, prostitutes, pseudogeniuses, cranks and eccentrics, and accurate analyses of the histories in these cases would not infrequently show a precox episode which separated a period of relative efficiency in their lives from the period following of relative inefficiency, in which latter it might be possible to detect certain precox residuals.

An analysis of the life histories of this class of cases shows quite regularly a constant tendency to slip from under all forms of responsibility, and a lack of capacity for any kind of continuous application and inability to develop the habit of work. They characteristically resort to the hobo type of existence, are unable to adapt, with any degree of efficiency at all, to complex conditions of life and wander from place to place, occupying one position after another from which they are either dismissed because of inefficiency or leave voluntarily, giving reasons for so doing which are totally inadequate. Such cases as these, sometimes by a steady progress become very greatly dilapidated and it is quite surprising at times to find the amount of deterioration in such cases after their admission to an institution and to realize how long they have gotten on in the outer world in a serious mentally crippled condition. Of course their continuance in the outer world was made possible only by their having sunk to low and relatively simple social levels that made little or no demands upon them.

When such patients as these get into situations which require continuity of effort and constant adjustment and from which they cannot escape they not infrequently suffer from severe and more acute breaks. This is seen in the military service. The army and the navy naturally attract this wandering horde of inefficients who see in the military service only the glitter of brass buttons and the opportunity to see the world. After enlistment, however, when they are required to take up the grind of daily work, their defects soon come into the foreground and manifest themselves either by distinct psychotic episodes or by minor infractions of military discipline, such as staying away from the post beyond the leave granted, or perhaps more serious offences, such as desertion. Such cases when they finally come under observation in a hospital easily show their defect.

Many women of this type marry, have children and although looked upon as "unique," or "queer," nevertheless get along if there are no serious stresses.

II. *Hebephrenia.*—This form of dementia precox is more severe than the preceding. It, however, is not essentially different, being practically the same condition occurring with greater severity, more apt to be somewhat more acute in its onset and manifesting itself more prominently by hallucinations and delusions.

The onset of hebephrenia not infrequently presents the outward appearance of a depression and so resembles the depressive phase of a manic-depressive psychosis, or perhaps some other form of depression. It not infrequently happens on the other hand, that the first

appearances of the disorder are those rather of mental confusion than of a marked depression. These early manifestations are the method of reaction of the individual to the first outbreak of the conflict. He may become either greatly depressed or may be quite unable to react along any well defined line and become quite confused. From this acute condition, after a longer or shorter duration, he settles down into a more stereotyped expression of psychotic symptoms.

During these early stages the productivity is not infrequently delirioid in character, accusing voices are heard and rather ill-defined and not well formulated delusions are expressed which are characteristically of a self-accusatory type and in harmony with the depression. Attempts at suicide not infrequently occur during this period.

After the active symptoms of the onset have subsided there is a settling down into a more stereotyped delusional expression, but not infrequently with a more or less incoherent productivity and with expressions that sound fantastic and silly. One patient complains that the sheets stick to his feet, another that he is the "wandering planet." Such ideas appear to have no adequate reason and are expressed quite disconnectedly from the general train of thought and little or no attempt is made to support them by logic. One patient, for example, says that his enemies are following him, and that he has been killed a number of times. Another complains that other patients are trying to injure him. All of these statements are made without show of emotion in a decidedly matter-of-fact way. Such appearances as these have led to such descriptive phrases as *looseness of the train of thought, poverty of ideas, emotional deterioration.* From the previous discussion, however, of the meaning of symptoms it is known that the hallucinations are expressions of the conflict, that the delusions are compromise formations, and that the apparent incoherency of the speech does not necessarily imply an incoherency in the thought content. It is usually not especially difficult to find some fairly direct connection between the accusations of the voices and actual occurrences in the patient's life. Similarly with other disorders of the sensorium. One patient, for example, who had seduced a girl, who bore him an illegitimate child, saw visions of his mother and heard the young woman's voice telling him to come home, to go to work and lead a decent life.

The peculiar emotional reaction, the lack of interest and apparent emotional dilapidation, as noted, is due to displacement, and so it is perfectly understandable that when an effort is made to gain access to such individuals they show little interest and characteristically reply to questions addressed to discovering the reasons for their action by "I don't know." It is really quite true that they do not know the reasons for their actions any more than any person understands the reasons for the different appearances that have come to him in a dream. And then again these patients are so occupied with themselves that they do not care often to be interfered with, and the "I don't know" is as much as to say "leave me alone."

The general conduct of the patient may also be listless, apathetic and disinterested in character and in harmony with his emotional condition, whereas mild alternations of depression and of excitement with the development of little peculiarities of conduct and speech show the alliance between this group of cases and the catatonics. One patient, for example, who thought that he was ordained to preach and that the bread was impure, would be quiet for months, and then exhibit a certain degree of restlessness by following the doctors and nurses about the ward telling them his troubles for a few days. The alternation may be much more marked as between a state of stupor and a state of excitement in which the patient eats paper, strings, and bedecks himself with all manner of trash and shows outbreaks of violent anger. This condition approaches much closer to catatonia.

Many of the cases, however, do not show that amount of dilapidation which the previous description would imply, while any degree of incoherence may be seen up to the production of a veritable "word salad." On the other hand, the patients may be found well oriented, making outwardly a natural appearance, being able to give a very good account of themselves, but presenting a grotesque delusional system, supported by hallucinations the expression of which stands in rather striking contrast to the outward appearances. These delusional systems represent all degrees of coherence and it would seem that the ability on the part of the patient to formulate a coherent and consistent delusional world was one expression at least of his capacity to react to the destructive influences of the conflict.

One such patient complained of illness on a train and told the conductor that she had had a hemorrhage. No sign of any hemorrhage was in evidence, however, and when she reached the sanitarium to which she was removed nothing abnormal was found on examination. When her mother arrived shortly afterward she was found delirious. Later, upon her admission to a hospital she was very hypochondriacal, talked at length, complained of attacks of hemorrhage from the vagina, which hemorrhage she said was due to an assault while she slept either by a man or some sharp instrument. Later on she claimed to be married to a Mr. O. and wrote long letters giving an account of her troubles and giving an account of the hemorrhages and the supposed operation, showing an extensive delusional system. She claimed to have been the victim of a criminal operation performed by a doctor. She later said that she had been engaged to a Hebrew before she was born and that at twenty she broke this engagement, and at three years of age a Catholic priest requested one of her offspring be given to that church for a clergyman and that the Hebrew family consented. In the midst of a great mass of incoherent, jumbled, delusional expressions evidences of a compensatory wish-fulfilling delusional system are found. Wishing for a child she became impregnated. Being a virtuous woman this has to be accounted for. She therefore has the delusion that she is married to Mr. O. Inasmuch as no child appears, a delusion that she has had

an abortion performed accounts for its absence, and as this is a criminal operation it was performed without her knowledge while she slept. These delusions are mixed up with all sorts of ideas about the dislocation of her various viscera, numerous hemorrhages from the vagina, and a fractured skull, etc. It is loosely organized and interrupted by violent outbreaks, restlessness and irritability.

She has been unable to adjust to reality, the effort at compensation by delusional formation has also failed, with the resulting progressive dilapidation of coherence in the stream of thought.



FIG. 446.—Appearance of patient in catatonic stupor.

III. *Catatonia.*—This variety of precox, like the hebephrenic, may come on suddenly with symptoms of confusion or depression, or may be of somewhat slower onset. It is more apt than the other forms to be of relatively acute onset, in which case it sometimes follows a sudden shock of a highly emotional character. It is characterized more especially by a predominance of motility disturbances and tends to express itself in alternating conditions of *catatonic stupor* and *catatonic excitement.*

In the conditions of *catatonic stupor* negativism reaches a very high degree. The patients are perfectly immobile, sitting off in corners by themselves or lying in bed without paying any apparent attention to what goes on about them and are quite inaccessible, fail to answer questions, and do not react at all to stimuli from the outer world. They characteristically often refuse to speak at all. This mutism is a manifestation of the negativism. Besides this the patients often refuse food, pay no attention to the promptings of the bladder and the rectum which become overloaded with urine and fecal matter, often to a serious extent. Saliva may be permitted to accumulate in the mouth where, if attention is not paid to it, it may undergo putrefactive changes. The patients quite characteristically show peculiar theatrical attitudinizing, make grimaces, occupy peculiar positions, and if they speak the productivity is often incoherent and apparently senseless, with a tendency to constant repetition of the same phrases —*perseveration*—which may also manifest itself in the movements of the body, such as a constant swaying movement or the like.

The muscular system may be in a condition of *waxy flexibility*, permitting of the molding of the limbs into any position where they remain indefinitely—*catalepsy*. When this is present the patients tend to show a more or less high degree of suggestibility and *command automatism*, doing mechanically and in a perfectly automatic manner that which they are commanded to do. This suggestibility shows itself further in *echolalia*, the repetition of words, or phrases that are addressed to them or that they hear others speak, and *echopraxia*,

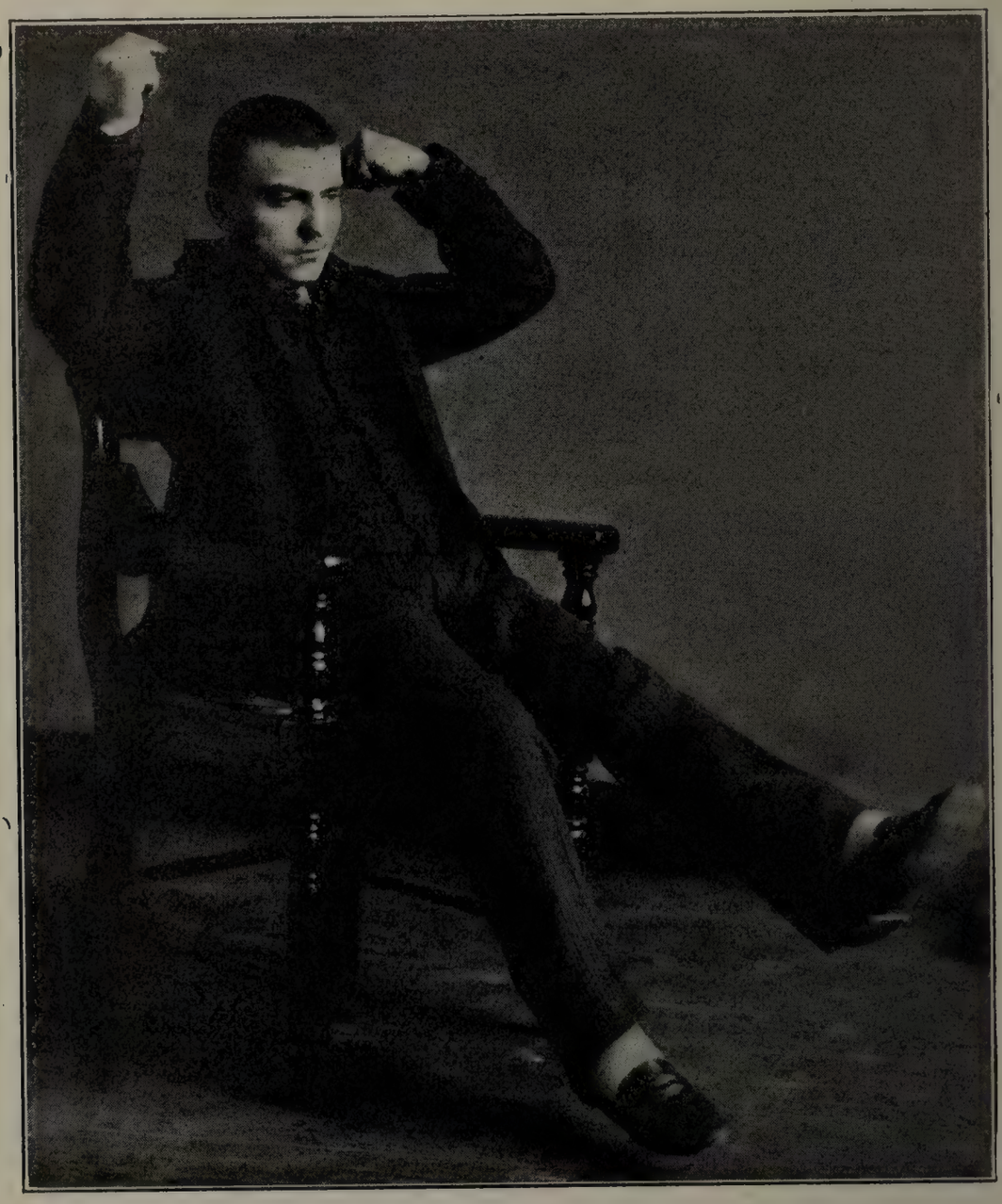


Fig. 447.—Catalepsy; flexibilitas cerea.

the repetition of movements which are made in their presence. On the other hand, there may be a marked degree of *muscular tension*, the patient maintaining fixed attitudes with the muscles thrown into a condition of rigid contraction. The limbs and body are stiff and immobile and resist any effort at bending. Patients in this condition are positively negativistic, withdrawing from all approaches, refusing any coöperation with the nurse in attempting to dress or undress or feed them, and show a tendency to react by doing the opposite of

what is expected of them. In this condition of stupor the patients may appear to be quite disoriented and have no knowledge of what is going on about them. If they are watched, however, they may be seen at times to show evidences of paying attention to their environment, and not infrequently when the stupor passes they are able to give a fairly good account of the things that happened during it, but show no capacity for explaining their strange conduct.

In the opposite condition of *catatonic excitement* there are marked degrees of activity, constant talkativeness and noisiness, sometimes destructive and impulsive tendencies manifested by breaking windows or attacking those about them, but in general showing, as does the speech, a marked lack of coherence. Both the productivity and the activities of the patient fail to show any clear goal, although some patients show what very closely approaches to the flight of ideas of the manic. Catatonic excitement may reach a very high grade, manifesting itself by wildly delirioid reactions, constant motor unrest and sleeplessness, a rapid failure in nutrition, a veritable *status catatonicus*. Some of these catatonic cases show hysteriform or epileptiform seizures, and death occasionally results in these higher grades of excitement.

The chronic conditions tend to show well defined mannerisms, such as the peculiar attitudes of the body, especially clumsy ways of holding the spoon or the fork in eating, meaningless grimaces, odd ways of walking, such as sliding the foot back and forth two or three times before starting off and other ceremonials for initiating movements. They are characteristically stiff, awkward, clumsy and inaccessible, and usually indifferent to their surroundings and apparently emotionally dull, though given at times to emotional outbreaks without apparent cause. All of these psychical symptoms of course must be considered as having some psychological meaning. In harmony with what has been said before both the types of increased suggestibility and catatonic rigidity are ways of shutting out the world of reality, whereas the peculiar automatisms can sometimes be traced to their meanings, as in the case before mentioned of the young woman who kept constantly pounding her hand with her clenched fist.

Physical Symptoms.—The general appearance of the catatonic forms of precox indicate more than in the other varieties the presence of definite somatic accompaniments. Exaggeration of the tendon reflexes, lowering of cutaneous sensibility, vasomotor disturbances, cold and cyanosed extremities, widely dilated pupils, disturbances of secretion, and loss of weight are frequently observed. It is this group of cases that have led most distinctly to a toxic theory of etiology and caused the precox cases to be thought of in connection with disturbances of metabolism due to changes in the internal secretions.[1]

Recent suggestions have taken the form of an attempt to hitch up

[1] Jelliffe: Dementia Precox and the Vegetative Nervous System. N. Y. Med. Jour., May, 1917.

the catatonic reaction with disease at the basal ganglia level, particularly the corpus striatum.

Fraenkel[1] does not believe that the cortex must always be indicted when the psychic life is altered, but believes that disease of the subcortical ganglia is associated quite constantly with mental symptoms. Pseudosclerosis and Wilson's disease show mental changes, as do athétosa double, which is dependent according to O. Vogt on a status dysmyelinisatus, and torsion spasm. In strangulation and coal-gas poisoning thus is found softening of the lenticular nucleus with speech and motility disturbances, stupor, and Korsakow's symptom complex. In catatonic precox the disturbances of vasomotor and vegetative functions resemble those found in neurological diseases of the subcortical ganglia. Catatonic symptoms may be expected in



Fig. 448.—Dementia precox; mannerism.



Fig. 449.—Dementia precox; mannerism.

many states. They are found not infrequently in the toxic-infectious group of psychoses, in paresis, in lethargic encephalitis, etc. Catatonia is not, therefore a specific type of reaction which belongs to precox solely.

IV. *Paranoid Forms.*—In the paranoid cases there is a much more efficient effort at creating a coherent and logically connected series of delusions and associated hallucinations. In these cases there is much less outward evidence of the dilapidation and emotional indifference that have led to the belief in the presence of a permanent mental impairment, a dementia.

These patients, originally, many of them at least, were grouped

[1] Ueber die psychiatrische Bedeutung der Erkrankungen der subcorticalen Ganglien und ihre Beziehungen zur Katatonie, Ztschr. f. d. ges. Neurol. u. Psychiatr. 1921, vol. **70.**

with the paranoias, but under the influence of Kraepelin that group of paranoid conditions which showed a tendency toward progressive deterioration were included in the dementia precox classification. All degrees of intervening possibilities, however, occur, so that one may get a fairly well knit delusional system in a patient who deteriorates quite rapidly, whereas other patients maintain their intellectual integrity over a period of years. The délire chronique of Magnan is generally considered to be dementia paranoides and this group of cases in general has been recently included by Kraepelin under the designation of *paraphrenia*. Some authors today, more particularly Bleuler, are inclined to think that perhaps the so-called true paranoias are only attenuated forms of dementia precox.

The delusional system in these cases must be conceived as a compromise formation and as essentially wish-fulfilling and is characteristically sexually colored. One such patient, a middle-aged woman, was persistently persecuted by a man who accused her of leading an immoral life to such an extent that she finally went to a physician to be examined to prove her virginity. All sorts of vulgar remarks were constantly made about her, she was referred to by the voices, as the widow of this man. The patient was a devout Catholic and the man was a Protestant, a perfectly understandable reason for a severe emotional conflict, which in her delusional system unloads itself upon the man rather than acknowledge its true origin. This whole conflict arose at the time of the death of her brother and it is significant that in her delusions she believed that she had received letters accusing her of incestuous relations with her brothers, of having become pregnant by them and of destroying the pregnancy. These letters were addressed to her as the wife of her several brothers. Here one sees an infantile determiner for the delusional system in the love which she entertained for her brothers and which later was transformed, under the influence of the conflict, into delusions of an incestuous nature. Finally, there was a whole crowd of people who were conspiring against her and whose object it was to kill her. Such delusional systems can without much difficulty be seen to be expressions in distorted form of the conflict. She has never been able to get away from her infantile attachment to the members of her family and establish herself upon her own feet, and these attachments hold her back, produce a withdrawal from the outer world, a regression into the world of fantasy, with a tendency toward what may be termed psychic death, or an absolute lack of efficient reaction to reality, and this is symbolized by the gang of persecutors who are bent upon her destruction.

V. *Mixed and Atypical States.*—All of these forms of dementia precox so far described are simply variants, in accordance with the present viewpoint, of one disease trend, and so it is not strange that it should be found that there are no hard and fast lines separating one from the other. In consequence it is not infrequent to see transitory or mild motility disturbances in cases that are essentially hebe-

phrenic or to see fairly well defined paranoid delusional formations in either the hebephrenic or the catatonic varieties.

As to the atypical forms, there have been mentioned and described under dementia simplex the larvated and abortive types, the "formes frustes." These are probably much more frequent than ordinarily supposed. For example, Wilmanns, in a study of 127 vagabonds, found 66 cases of dementia precox, while undoubtedly it is not infrequent for these abortive forms to be considered as cases of constitutional defect. This difficulty becomes very much more aggravated when it is remembered that attacks of dementia precox may occur in childhood, according to Vogt as early as five years of age, while a little later, nearer to the period of puberty, they are not so uncommon. In these cases of course mental development is interfered with and remaining upon a lower level gives the impression of imbecility. The same difficulty arises in a somewhat aggravated form when dementia precox develops in young persons already of defective make-up. Here, of course, the differentiation is no longer possible, because it must be realized that both conditions of defect and precox are present in the same individual.

Pathology.—There is a considerable pathology of precox which, however, is not always clearly correlated with the clinical picture. It is probable that during the life of the individual disturbances that are shown in the general physical manifestations of the disease occur at the biochemical level. The pathology of the central nervous system is essentially parenchymatous, there being little evidence of changes in either the vessels or the neuroglia. Tuberculosis is quite frequent, but is easily understandable as being dependent upon the general lowered physical condition and inactivity of these patients, coupled with their bad habits and the necessity of their rather close confinement, although it has been suggested that the connection is much more intimate. In fact Wolfer[1] thinks that tuberculosis is a factor of prime etiological significance. Such changes as are found in the nerve cells are of a degenerative character, with evidences of neuronophagia and with perhaps amœboid glia cells. The acute cases of catatonic excitement which end fatally, the so-called catatonic "Hirntod," show a certain amount of evidence of degeneration, more particularly, however, evidences of a chemical nature in the form of certain lipoid elements in the cortex, which seem to be products of disintegration.

Southard has found certain anomalies in precox brains which appear to be of the nature of aplasias or ageneses. These agenetic or aplastic areas appear to be grouped more or less in correspondence with the three main types of the disease. The frontal region is characteristically involved, while he has described a cerebellar group corresponding to the catatonic variety, while the profound emotional disturbances he thinks are due to lesions in the deep layers of the cortex which have no direct motor, sensory or perhaps associational

[1] Die Tuberculogenese der Dementia Præcox, Zeit. f. d. ges. Neurol. u. Psychiat., 1919, vol. **52.**

relations. These changes and perhaps many others will probably be illuminated by an understanding of the mechanisms at the vegetative nervous system level.

Mott[1] has found, in a large proportion of testes of precox cases examined, a complete arrest of spermatogenesis and in all cases a more or less regressive atrophy. In spermatogenesis active nuclear proliferation is going on with abundant nucleic acid formation. The failure of nuclear activity in the testicle is considered as important in view of Nissl's and Alzheimer's demonstration that the essential change in the nerve cells in precox is one of nuclear decay. Mott traces the chemical results of this lack of nuclear activity and seeks to explain the pathological findings such as atrophy and granulo-pigmentary degeneration, on this basis which can be broadly expressed as a process of impaired oxidation. He seeks also to correlate these changes with the clinical picture. These findings are significant in view of the deficient potency of so many of these patients and the therapeutic effects of sodium nucleinate as recommended by Lafora.[2]

These findings of Mott are in line with what has been found by other investigators. For example, Hauck[3] has found general infantilism relatively frequent and genital infantilism in female patients especially in association with the catatonic form. Lewis[4] has found marked aplasia of the circulatory system manifested by very small hearts and aortic arches.

The most extensive investigation into the histopathology of the central nervous system in dementia precox that has been made since Alzheimer's time is that of Josephy.[5] His material consisted of 50 brains, some of which, however, had to be rejected as unfit for study. His control material consisted of the brains of 5 mentally normal persons, dead of phthisis. Josephy believes that dementia precox is not only a mind disease, but that it is also a brain disease. He comes to this conclusion because of the practically invariable presence of pathological findings. Briefly he concludes as follows:

The brain of precox shows evidences of a disease process occurring in an organ which, so far as can be discovered, was previously healthy. He remarks upon the infrequency of anomalies of development and states that anatomically the process is a purely parenchymatous one without inflammatory component. It is the ganglion cells which are principally attacked. Fatty degeneration, sclerosis, and even cell atrophy are found, while glia proliferation of a peculiar type plays a role in rare cases. While these changes are considerable, it is not pos-

[1] Normal and morbid conditions of the testes from birth to old age in one hundred asylum and hospital cases. British Med. Jour., November 22, 29 and December 6, 1919.

[2] Nuevo tratamiento causal de la demencia precoz y concepcion de su patogenia, El Siglo medico, 1918.

[3] Gynäkologische Untersuchungen bei Schizophrenen. Monatschr. f. Psych. u. Neurol., 1920, vol. **47**.

[4] At Saint Elizabeth's Hospital, Washington, D. C.

[5] Beiträge zur Histopathologie der Dementia precox, Ztschr. d. f. ges. Neurol. u. Psychiat., 1923, **86**, Nos. 3 and 4, 391.

sible to make a diagnosis with a microscope because they are largely, undoubtedly, end-products rather than signs of a process still in a state of activity. The location of the changes was most marked in the anterior portions of the brain, especially in the frontal and temporal lobes and in the third and fifth cortical layers. Changes in the cells of the basal ganglia were found and he believes that disease of the pallidum is somehow related to catatonic symptoms.

Just what relation the anatomical findings have to the mental picture Josephy is unable to state. He is rather inclined to believe, however, that they indicate a toxic condition.

Extra-neural Pathology.—If the distinctive characteristic of psychological reactions is that they are *total reactions*[1] as compared to various organ reactions which are therefore partial reactions then it certainly would not be strange if we should find an extensive pathology of mental disease in organs other than the cortex. This is in harmony with certain pretty generally accepted descriptive characteristics of dementia precox, namely, (*a*) that there is an organic, probably hereditary factor present, (*b*) that the majority of onsets occur early in life, and (*c*) the high incidence of pulmonary and other forms of tuberculosis which is the terminal lesion in 85 to 90 per cent. of cases. When the individual comes to function as a whole the inadequacy of his several parts to the demands made upon them must find expression in the final result, the end-product, as expressed in his thinking, feeling, and acting. Lewis's findings in the Saint Elizabeth's Hospital material[2] strongly support this view, based upon a study of over 600 cases. These findings are briefly as follows:

The average weight of the dementia precox heart is one-third less than the normal or average, the small size being independent of race, age, sex, size of body, duration of psychosis, or lethal lesion. It stands out in considerable contrast with the other mental disease groups. The decrease in size is of the nature of an aplasia or arrest in growth since the well known pathological signs of atrophy are as often absent as present, the valves are normal developmentally and free from lesions, and although occasionally secondary atrophy in the muscle is present the original arrest in growth is indicated by the small aorta with its correspondingly small branches. This type of circulatory system retains its small proportion throughout life and lacks the capacity to develop a true compensatory hypertrophy. The walls of the congenitally small aorta are always thin, smooth, free from lesions and usually hyperelastic. This state of affairs along with the widened bed of the lymphatic stream, although not associated with all of the features of the status lymphaticus syndrome, may suggest the larval state of this disorder.

In the gonads he found the same three stages of regressive atrophy

[1] I have set forth this view of the nature of psychological reactions in my "Foundations of Psychiatry," Nerv. and Ment. Dis., Mon. Se., No. 32.

[2] The Constitutional Factors in Dementia Precox, Nerv. and Ment. Dis., Monograph Series, No. 35.

described by Mott and his associates, but differs with them in one respect in that he found no correspondence between the degree of regressive atrophy of the testes and the duration of the mental symptoms, since some of the most profound changes were in the organs of those with psychoses of short duration.

In these cases he has found pronounced alterations in the adrenal cortex to be fully as constant a feature as regressive atrophy of the testes. In general the cortex is thin, the three histological zones being narrow and ill-defined. The stroma is always abundant with definite sclerosis of the zona glomerulos. The medulla does not always share the atrophy, but may be normal or hypertrophied. In 50 per cent. of the cases there was a definite arrest in the cortical development. Accessory adrenals are frequently found in the dementia precox group.

The thyroid gland was found altered in its connective tissue and colloid structures in such a manner as to suggest a functional association with the genital glands. Various degrees of sclerosis with marked losses in colloid, which have been proven to be independent of other factors, is a characteristic finding, and moreover the scleroses do not conform to descriptions of the "senile type" of gland.

Directing attention now to the *paranoid development*, it is obvious that we are dealing with a much smaller group of individuals, and one in which an opportunity is rarely available to investigate the physical components of the condition in its early stages, since these individuals usually live for a number of years, apparently living out the life span and deteriorating late in life, a deterioration which is perhaps slightly presenile in the average case, therefore, when the opportunity comes for a postmortem there is the probability of finding such changes as were once prominent to be partly or wholly obscured by senile changes, however, in a group of cases comprising 18 paranoid developments among the diagnostic appellations of which were paranoid state, alcoholic paranoia, and paranoid dementia precox, there were tissue changes quite characteristic of the group differentiating it from seniles and arteriosclerotics on the one hand and from the dementia precox group on the other.

In the first place very few of the paranoids died from tuberculosis and none in the group were seriously affected with this disease, but they do succumb from vascular accidents and so prevalent are these accidents among the paranoids that almost all cases die with cerebral hemorrhage, ruptured aneurysms, or cardiac decompensation. The circulatory systems of these individuals are markedly in contrast with those of dementia precox. The heart weight is greater because of the incidence of compensatory hypertrophy; and also the arterial system as a whole is larger in caliber.

Associated with these compensatory reactions through the circulatory system there was found in the thyroid gland an atrophy with colloid destruction and a fibrosis which is patchy in type, these patches very often alternating with areas of compensatory glandular reaction, frequently to the extent of adenomatous proliferations. In some of the

other thyroids of this group there was very little colloid reduction, but very marked hyperplasias with general overgrowth of the epithelioid cells.

The adrenal glands are quite in contrast to the dementia precox cortex, a widened structure which is developmentally large and which later may have undergone secondary disease alterations, but still patches were found of compensatory glandular reactions in certain areas in that there is an increase in the number of cortical acini, sometimes to the extent of an adenoma. The medullary portions were also occasionally hyperplastic.

The gonads showed some interesting features in contrast to the dementia precox testicle so well described by Mott and others, in that they presented areas of tubular atrophy, alternating with normal areas, with hyperplastic tubules, and sometimes with hyperplastic areas of adenomatous nature. Thus it appears that all through the endocrine glands of the paranoid group there are reactions on the part of the tissue to compensate for focal defects probably indicating attempts to maintain a certain level of functional existence. Frequently in the other tissues of those dying in paranoid states we find evidence of compensatory reaction, this being particularly well shown in the tendency to blood vessel proliferation, miliary angiomata, prostatic hypertrophy and carcinomata, and liver cell hypertrophy.

Since the so-called paranoid dementia precox presents the reaction mentioned above it would seem that this type should be placed definitely in the group of paranoid developments and the term "precox" dropped, as this constitution is in no way like that of the hebephrenic and catatonic type first mentioned, where the secondary tissue reactions are regressive in nature and where there is a primary hypoplasia of the circulatory system which is quite in contrast with the compensatory type of circulatory system of the paranoid developments which usually ends in rupture from overcompensation, or in cardiac decompensation.

In contrast to the type of conduct and regression to the autoerotic level of development found in the majority of hebephrenic and catatonic precox states with their aplastic circulatory apparatus and defective endocrine glands, are the paranoid compensatory reactions (including "paranoid dementia precox") where there is a better preservation of the personality, an interest in the outside world, more harmony between ideas and mood and less tendency to deteriorate—the tissues of the body participating in this preservation by compensatory growth changes.

Nature of Dementia Precox.—From the description of dementia precox up to this point it will be seen that it presents correlations on the one hand to the more distinctly so-called psychogenic types of disorders, such as the psychoneuroses, and on the other hand to the more distinctly somatic diseases. It has been seen that it was possible to formulate all of the mental symptoms in the same way that they are formulated in the psychoneuroses. In other words, the mental

symptoms are capable of interpretation solely at the psychological level. On the other hand, for an understanding of the whole disease process it must not be lost sight of that recent investigations are tending to show more and more that there are distinct biochemical disturbances during life and pathological changes are being found after death. In any case the acute cases that lead to death must be conceived as having profound bodily changes correlated with psychic symptoms.[1]

The formulation of the disease from either standpoint alone must of necessity be unsatisfactory. For example, the theory which makes the etiology toxic fails utterly to give a comprehensive idea of why the mental manifestations take the particular form they do, why for example, in the case already cited, the hallucinatory voice should be that of a young woman that the patient had seduced telling him to lead a decent life. This is surely the patient's self-critique appearing in hallucinatory form.

At any rate, it seems quite evident that the great difficulty of conception here is due in part to the quite arbitrary separation of the individual into two distinct and mutually exclusive parts, namely, mind and body. The dualistic hypothesis that conceives of the mental and the physical going side by side without mutual interference or interaction is responsible for such a conception. From the medical standpoint such a concept seems entirely unnecessary, for one is met at every turn with the intimate relations constantly maintained between the two, and therefore comes naturally to consider the human being as a biological unit presenting types of reactions that at one extreme are predominantly psychic, while at the other they are predominantly physical, but which present every grade of intermediary type. The severe mental disturbance of the nature of anxiety that goes with certain cardiac and pulmonary diseases is well known, as are also the physical upsets, particularly the gastro-intestinal, that are associated with certain mental conditions such as worry. The interrelations between the mental and the physical are a matter of daily observation. Occasionally, however, they are very pronouncedly emphasized. A chronic patient who had been for many years in a hospital, working daily at outdoor labor, was suddenly seized with a violent impulse in which he attacked all about him. He seized a heavy iron bar, killed two people and injured another and ran headlong and wildly without direction into the woods. He was finally cornered, and in the process of securing him he was shot by a farmer with a load of buckshot, none of which, however, penetrated farther than through the skin and produced no serious wounds. He was brought back to the hospital, incoherent, mumbling and trembling, showing all the evidences of a tremendous emotional upset. The shot were picked out of his skin, the wounds were dressed and he was put

[1] Jung: Psychology of Dementia Precox; Spielrein: Analysis of a Case of Dementia Precox; Binswanger: Dementia Precox. See Abstracts in Psychoanalytic Review, vols. 2, 3, 4.

to bed. Up to that time he had been a strong, physically healthy negro. He never left his bed again and approximately a year afterward he died, having developed an acute tuberculosis. Such cases as these demonstrate the necessity of considering the human being as a unit and not endeavoring to draw hard and fast lines of distinction between the mind on the one hand and the body on the other.



Fig. 450.—Fantastic paranoia.

In the present state of knowledge, however, one is often unable to make any specific correlation between the physical findings and the mental symptoms, while on the other hand it is quite possible to express the symptomatology of the disease, to describe it, to, so to speak to reconstruct the psychosis purely in psychological terms. For the present, therefore, the disease must be described psychologically, and the explanation of the mental symptoms must be sought psychogenetically, without, however, forgetting that there are certain somatic changes which are pretty generally attached to the symptomatology of the disease process and which must ultimately be made to fit into the general rubric before a complete understanding of the entire situation is had. It will be seen, too, that this method of procedure helps to an understanding of many of the physical symptoms which otherwise stand alone and cannot be assimilated to the general concept.

On the psychological side, then, dementia precox is seen to be a certain type of reaction to a mental conflict, resulting in a splitting of the psyche and the outcrop of unconscious mental trends to the surface of the mental life. The patient is confronted with a situation to which he cannot adequately adjust, which is absolutely inacceptable and impossible, and he is therefore driven away by his incapacity to assimilate it and cast back upon himself. The battle of the opposing forces produces the disease picture which is the outward evidence of the effort on the part of the individual to reach a solution of the difficulty. The symptoms are the results of the appearance of the unconscious trends distorted and disguised as they are in dream formations. Jung has especially noted this similarity to the dream state and would consider precox as a

sort of waking dream or dream from which the patient does not awake, the dream picture being fixed, as it were, by another element in the situation, the toxin, which for the sake of analogy solely, may be said to act like the fixing agent in the photographic process. It is seen that the conception of the disease as being from the beginning and fundamentally a dementia must be modified, if by dementia is meant a permanent mental impairment, a mental loss, which is thought of in the same terms as a loss of tissue from a wound. It would appear from the description of the mechanisms that have been given that at least in the early stages of the disease there is nothing at all corresponding to such a permanent loss; that what has happened has been a disintegration, a falling apart of the component parts of the psyche, a shifting of emphasis to the unconscious factors in the conflict, more particularly a shifting, or to use the technical term, a displacement of the emotional content of certain idea constellations. The dementia, therefore, which has been described as such, is at least to this extent a misnomer. From the point of view, however, that this disintegration and resulting impairment is either permanent or tends to be, it has a meaning. If instead of trying to express what is happening in terms of mind and body the form of the presentation be cast in terms of energy then what is happening may be described by saying that the patient withdraws his interests (charge of energy) from the outer world of reality, like an amœba withdraws its pseudopodia, and that this energy comes, therefore, to be projected upon, to charge the individual himself. This energy reanimates progressively lower levels in proportion to the depth of regression. The energy which is redirected from reality to self does not alone possess the possibilities for interest but has a libidinous component which, if directed without, is the root of the capacity for object love. Directed within it not only produces interest in self to the exclusion of the world of reality, but a love of self, which has a distinct erotic element. The libido removed from the outer world of interests and turned upon the self causes that enlargement of the ego which is the basis of the grandiose delusions. This is the autoerotic and narcissistic component of dementia precox which explains so much of its symptomatology and which also accounts so largely for its malignant character.



FIG. 451.—The fetal attitude assumed in severe grades of regression. This attitude was maintained most of the time for years, the patient seeking the darkest corner of the ward.

The psychological side of the situation, however, as may be seen, is not all. There are certain physical changes in the course of the

disease and certain pathological findings. So far as observation goes, however, the etiological factors lie almost, if not quite entirely, in the mental sphere and one must therefore conceive of the physical changes as superadded. This is a possibility which was well illustrated by the case already quoted of the man who died after a tremendous emotional explosion during which he killed two people. When the psychic splitting is profound and when it is of considerable duration it is quite understandable that it should unloose bits of physiological mechanism and thus produce the physical changes found. From the descriptions of the mechanisms in the psychoneuroses, taken in connection with the discussion of the vegetative nervous system, it may be seen that constantly operative psychic disturbances are capable of producing physical changes. Compare Crile's study of the emotions and Cannon's work on the relation of anger and fear to the gastro-intestinal functions where it may be seen that both surgeon and physiologist are forced to put the psychic factor in the foreground. Or to put it in terms of energy change, the energy has been withdrawn from its occupation with objective reality and is now turned back upon the individual where it expresses itself as muscular tensions, visceral tonicities, or in other words as physiological function or to use the terminology developed in the Introduction, it expresses itself as organ libido.

This withdrawal of interest (energy) from occupation with the world of reality and the consequent redistribution of the withdrawn energy (libido) involves still further implications. Whereas the energy charge of certain organs may be increased, that of others may be diminished. The occupation of certain zones or organs with a libido charge may be so painful that the only defense is to so raise the barriers to this territory that the energy will be kept from overflowing them. Just as the hysteric pushes, so to speak, an arm out of the field of consciousness by rendering it anesthetic, so here certain organs, zones, or autonomic segments are sidetracked. Small, inferior, or aplasic organs may therefore be considered not only congenitally or hereditarily defective, but as possibly arising as a result of this withdrawal of energy—a sort of atrophy by disuse as it were. Either hypothesis seems to be tenable. At present there seem to be no adequate criteria for deciding between them in a particular instance.

As already indicated, dementia precox is an introversion psychosis, that is, instead of running away from his conflict into reality as does the manic-depressive, the patient turns back and attempts to retrace his footsteps upon the path of development. Introversion, however, is a characteristic of many types of mental illness, some of the neuroses, for example, and occurs in the course of other illnesses such as paresis.

Introversion to some degree is also a quite common mechanism in otherwise healthy people, especially when confronted by difficulties, while whole races, the Orientals, seem to be characterized by an emphasis on the introversion side of their mental mechanisms.

To describe precox then, as an introversion psychosis merely desig-

nates its most characteristic quality, it does no more than this. Why does introversion become so malignant in these cases? That is the question to be answered. A number of suggestions only can be offered, any one or all of several factors may be active. The depth of the introversion is probably important but the stratigraphy of the psyche is not well enough known to decide this question. Many patients do, however, get worse in proportion to the distance on the regressive pathway that their difficulties force them. Then again the strength of the fixation is of undoubted importance but perhaps of greater importance to prognosis for many patients with profound regression get well, often rapidly, which would indicate that the strength of the fixation was not great. The factors which make for possibility of rebound are not known though, in general, when the etiological factors are largely exogenous and can be removed recovery is more to be expected as in the war neuroses and the prison psychoses. The quantity of libido that regresses as compared to the quantity remaining attached to reality is undoubtedly a factor of the first importance. Finally, as has been indicated, regression may involve bits of physiological mechanism and so tend to fix the regression and prevent the recovery. This latter element is bound up in the conception of inferior organs. It is often impossible to say whether an organic difficulty has been produced by the regression or whether it conditioned the regression in the first instance. In other words, whether the etiology is primarily psychogenic, or organic, or whether, on the other hand, organic and psychological manifestations are not both the outward manifestations of an inherent developmental anomaly.

The general tendency at present is to think of the psychological conflict as primary and the various symptoms of disordered function at the physiological level as secondary. Circulatory and glandular imbalances and functional disturbances of various organs are thus explained as results of the primary emotional disturbances and the organic changes as results when such functional disorders are maintained for a long period and are severe in character. In many instances primary defects of the personality undoubtedly found in organic defects and such defects as well as those that are more obviously secondary, would appear to be mediated through the vegetative nervous system. They would include glandular (gonadal, etc.) deficiencies and imbalances, gastro-intestinal, cardiovascular and other organic inadequacies, and organic deficiencies such as the aplasias of the central nervous system. There is a wide field here for correlating generalizations which will undoubtedly show precox to be the result of relatively severe processes which in less severity occur in prognostically less serious conditions such as certain of the neuroses.

From this point of view then precox is an especially severe manifestation of the individual's inadequacy to meet the biological demands made upon him to fulfil his biological destiny. The theory of a toxemia does not help, as such, to understand the results but on the other hand the toxemia can be understood, if it exists, as resulting from

the general functional disturbances incidental to his unsuccessful struggle with his problems. Only under some such comprehensive formula as here indicated is it possible to arrange any of the multitudinous manifestations of this type of mental illness.

A study of the dominating visceral tonicities and postural tensions in individual cases will demonstrate the nature of the psychological conflict because it will show the organs that are in a state of preparedness for carrying out unconscious trends which, however, are not permitted to express themselves but are kept inoperative by the force of repression.

Ways of Getting Well.—With the concept of the disease process which has been previously elaborated, what is the significance of the three main types of precox which are found clinically? Bertschinger[1] has made an admirable study of the process of recovery in precox and his discussion of the nature of the conflict and the ways of adjusting is particularly illuminating.

The three clinical types of the disease are the expression of the interplay of the two factors, the conflict and the reaction, the severity of the former and the efficiency of the latter determining the outcome in the individual case. The degree of confusion in the acute onset would then be an expression of the completeness with which the patient was driven back from reality and the dominance of the unconscious trends. Conditions of moderate confusion with capacity for adequate reaction to reality at times, or under the special stimuli of, for example, questions, show that the patient still has a certain grip upon the real world and is making an effort at least to retain it. Certain other cases of quite clear consciousness with complete orientation show a very adequate grasp upon reality and these patients, to the casual observer, often seem quite natural. In such patients, however, one will notice interferences of thought, hesitations in the course of conversations, stutterings and stammerings over certain points, the evidences in other words, of complex interferences and the patients will complain that from time to time their minds seem to be absolutely blank. These *saltatory associations* and *thought deprivations* are the expressions of reactions to buried complexes, so that in these patients there is a fairly adequate grasp upon reality for ordinary purposes at least, with only spasmodic influences from relatively restricted areas.

In general, then, the hebephrenic type may be seen to be a reaction to the conflict which is essentially inadequate and inefficient. In the acute stages the patient may be absolutely overwhelmed by the conflict, disoriented and confused. Later on the progressive disintegration and dilapidation of thought indicates the slowly progressive conquering of the capacity for adjustment to reality by the invasion into consciousness of the unconscious trends.

The catatonic type represents a somewhat different form of reaction.

[1] Heilungsvorgänge bei Schizophrenen, Allg. Ztschr. Psychiat., Band 68, Heft 2. Translated in Psychoanalytic Review, April, 1916, iii, No. 2.

Here the patient is oftentimes suddenly overwhelmed by the conflict, as under circumstances of accident or sudden and severe shock. No attempt at adjustment is made at first, but the whole situation is actively and definitely shut out. Here there is an active effort on the part of the individual to exclude the offending tendencies, and when this succeeds recovery takes place as the result, so to speak, of the encapsulation of the objectionable material and its exclusion from consciousness. This form of the disorder is the most acute and the recovery is equally most apt to be prompt, and it will be seen from this explanation why this is so.

In the paranoid form of the disorder the reaction is much more efficient than in the hebephrenic variety and in some respects less efficient than in the catatonic. Here the individual takes a flight into a psychosis and the delusions are the expression of a compromise between the opposing psychic trends. Unable to live in the real world, the patient succeeds in inventing a world in which he can live, and having invented it he succeeds in getting along fairly well without noticeable deterioration. The conflict in these cases tends to become stationary after the development of the delusional world. The mechanism here is the same as in paranoia. (See Paranoia.)

Bertschinger has more especially defined the ways of getting well by pointing out that the patient in recovering may, as the result of his conflict to which he cannot adjust, find a compromise by changing himself and interpreting the world of reality in terms of his morbid fantasy, or by translating the world of his fantasy into terms of external experience. And so one would find on the one hand delusions of grandeur which are a compromise formation and serve to change the individual so that he may be better satisfied with life and on the other hand one finds delusions of persecution, the delusions of influence from the outer world, that serve to change the outer world in conformity with the patient's complexes. These outside influences are but the reflections back upon the patient of his failures to get from the world what he wants and they are consequently felt as malign and destructive influences. Another method of getting well is that already described of the catatonic, the shutting out and encapsulation of the conflict in a circumscribed amnesia.

In many cases conversions into bodily symptoms, such as are found in hysteria, are found. Another method of getting well is by living through a series of imaginary experiences, which brings the complex to a logical conclusion. For example: A young Japanese woman was overwhelmed by the sad news that five members of her family had been killed in battle. She passed instantly into a dreamy state of consciousness, went on with the work of the household just as if all five were members of it, made their beds, set their places at table and acted in every way as if they were alive and present. Finally she, so to speak, let one of them die and then another and another until finally she had compassed the death of all five, after which she awoke from her dream-like state and was well. She had succeeded in an

efficient reaction to the situation by its attenuation, extending it over a considerable period of time. Finally, a certain number of patients get well by the final domination of the reality motive, with a resulting correction of their delusional fantasies.

Recently Mayer-Gross[1] has made a very interesting study of the wide range of borderland conditions lying between complete recovery with free insight and the more or less recognizable persistence of the psychic disturbances. There are various psychological points of view which patients may assume as their condition improves. The following are possible attitudes which they may take toward the schizophrenic psychosis which has run its course. (1) "Despair" because of the disturbance of the life values with the tendency to suicide. (2) "New life," a sort of resignation in a discouraged psychic existence with conscious renunciation of the past; a shutting off of the self in an atmosphere of small interests or giving up to aimless rambling or sinking into purposeless apathy. (3) "Rejection," a denial of the experiences of the disease itself. Even in thought these experiences are repressed as much as possible and there is a constant striving against the appearance of any after effects. To these more or less negative attitudes may be added two positive ones. (4) "Conversion," the life values are reversed. That which occurred before the psychosis has little worth. The psychosis itself was the critical moment, the beginning of illumination and regeneration. (5) "Amalgamation," the experiences during the illness are ceaselessly mingled with the continuance of the development of the personality. It will be seen from this that the author regards the terminal stages, not simply as passive biological occurrences, but as reactions of the preserved remnants of the personality to the endogenous catastrophe.

Course and Progress.—As will be seen from the description of the disease the catatonic form is more apt to be acute in onset and it has the best prognosis, while the hebephrenic form and the simple dementing varieties tend to progressive deterioration and the paranoid form tends to remain stationary without material deterioration. When the paranoid precox deteriorates it is because his compensation breaks down. A few of the hebephrenics get well, more of the catatonics recover, but all of these cases are liable to recurrent attacks. According to a study of Zablocka[2] of 515 cases, 60 per cent. proceeded to light, 18 per cent. to medium and 22 per cent. to severe deterioration.

In the catatonic cases that recover there is usually an appreciable change in the individual. The encapsulation of the conflict means that the recovery has taken place by a sacrifice of a certain portion of the personality. The portion of the personality in which the conflict is resident, so to speak, has had to be cut out or walled off, and the

[1] Ueber die Stellungsnahme zur abgelaufenen akuten Psychose. Eine Studie über verständliche Zusammenhänge in der Schizophrenie, Zeit. f. d. ges. Neurol. u. Psychiatr., 1920, vol. **60**.

[2] Zur Prognosestellung bei der Dementia Precox, Allg. Ztschr. f. Psychiat., Band lxv.

energies from this region are no longer available to the individual. This loss is quite characteristically shown in certain changes of character, indicating that the individual has gotten well, but has paid a certain price for that result.

The cases that do not get well tend to regress to lower levels, and quite characteristically, in institutions at least, they finally reach a level on which they can maintain themselves without sinking further. In this condition they remain stationary for long periods of time. Quite often patients brought to an institution in a very much disturbed condition settle down quite promptly under the simpler conditions of institution life, while in the outer world the precox cases tend to gravitate into the ranks of the hobo, the prostitute, and the petty criminal. Here they finally find their level and get along after a fashion.

Acute and severe grades of regression, in which the splitting goes to the very foundation of the personality, often express themselves by suicidal attempts and sometimes by homicidal attempts. The preparation for such attempts may sometimes be foreseen in the dreams.

Remissions are quite the rule and come about under circumstances which reanimate the conflict. Often patients get along very well in an institution, but become upset shortly after going back to the conditions under which the conflict developed. This is especially true, Bertschinger has shown, if the conditions to which the patient is returned have materially changed during his internment. The patient does better if returned to a home that has remained comparatively unchanged. If, on the contrary, the family has moved into a new neighborhood, acquired new associations, or if a parent has died meantime, conditions requiring new adjustments on the part of the patient, he may not be able to meet the demands and so relapses.

Malignancy of the Reaction.—The ways of getting well show that, for the most part, the method of dealing with the conflict is the opposite of that in the extraversion type of psychosis. It is the method of introversion that demands that the individual change in conformity with the demands of the complex, the delusional system. This method, because it involves a flight away from rather than into reality is a determining factor in the malignancy of the precox type of reaction. It is because the patient cannot escape from the mechanism of introversion, because he cannot project his interests into the world of reality (extraversion) that he so frequently fails to make those adjustments that spell recovery. He is incapable of socializing his instincts and remains shut in, infantile, solitary, asocial.

The course of the disease is irregular and prediction is quite impossible. In a general way, however, one gets clues from keeping in mind the fact that the conflict is between the reality motive and the fantasy formation, and by watching the interplay of these two motives one gets an idea as to whether the reality motive is gaining or losing ground.

The question of a recovery is a mooted one, but in general it is

conceded that the patients that recover do present to careful analysis certain residuals. Of course, however, these residuals may be of any degree and may be so slight as not to necessarily impair the individuals in the position in life which they may occupy. It is possible that true recovery may take place with a resolution of all the symptoms, but if this is so it is probably the exception.

Diagnosis.—The diagnosis of dementia precox, while comparatively easy in the well defined and the advanced cases, becomes a matter of great difficulty during the early history of the illness or in cases in which the symptoms are mild.

The manic-depressive psychosis presents one of the characteristic difficulties. In this psychosis there is usually a history of repeated attacks without deterioration. It must not be forgotten, however, that precox itself frequently presents a similar history and that if deterioration is present it may not be prominently in evidence. The depression which so frequently ushers in a precox attack may easily be mistaken for a depression of the manic-depressive psychosis. The manic-depressive retardation is similar in its outward appearances to the indifference and perhaps lighter degrees of negativism, particularly the inaccessibility of the precox. The manic-depressive is more apt to have delusions of a self-accusatory type than the precox and the latter's delusions are more apt to be grotesque. They more frequently are evident distortions and symbolisms. The pressure of activity of the manic resembles the activity of the catatonic. In the former, however, the activity, although rapidly changing as to its object, is characteristically addressed to some purpose, while in the catatonic the activity is more diffuse and has less direction. It is more incoherent. The distinction here between the extraversion of the manic and the introversion of the precox will aid in the differentiation.

It is still a mooted question whether there are not conditions intermediate between precox and manic-depressive psychosis which partake somewhat freely of the characteristics of both. There are a considerable number of cases in which the difficulties of diagnosis are very great. Flight of ideas may be quite typical in precox, for example, while the depression of the precox may resemble very closely that of the manic-depressive. The difficulties are greatest with that class of manic-depressive psychoses known as the mixed states.

If we will consider the individual and not the disease we can understand how in the same patient extraversion and introversion mechanisms may replace one another. In precox, therefore, we may expect to see temporary efforts to escape from the conflict by a flight into reality—thus producing manic symptoms which, however, cannot be kept up for long and then the patient lapses into regression. In this way the characteristics of the two psychoses may be present in the same illness and only represent efforts at dealing with the conflict. Such facts indicate the danger of attempting to adhere to a cut-and-dried scheme of diagnosis.

In the early stages the mild depression of the precox may simulate

that of a neurasthenic, or the agitated depression may simulate that of an anxiety neurosis. In both instances the precox is more apt to show grotesque delusions and conduct disorders of a bizarre nature, such as tearing his clothes, self-mutilation, or, on the other hand, characteristic negativistic symptoms, such as retaining the saliva or the urine, withdrawing from efforts addressed to assist him, refusing to coöperate in changing his clothes, the refusal of food and the like. It must not be forgotten, however, that the etiological factors of the actual neuroses may operate in the same person who breaks down as a precox, and that therefore neurasthenic and anxiety symptoms may be present as expressions of these etiological factors. It is important to keep such a possibility as this in mind when it comes to the matter of treatment.

Epileptiform and hysteriform episodes may lead to a diagnosis of epilepsy or hysteria. It must not be forgotten, however, that it is possible to have precox complicated with epilepsy and that in dementia precox all of the symptoms and characteristic mechanisms that are found in hysteria may be found.

In the infection and exhaustion psychoses the differentiation may be very difficult and it is necessary in such patients often to wait for a considerable time until the subsidence of the infection and then see whether the case clears up, as it usually does if it is a simple infection psychosis. One has to be very careful in making a prognosis in cases of this character. Not infrequently cases of so-called puerperal insanity are really cases of precox which have been precipitated by the circumstances of the puerperal period, loss of blood, prolonged labor, infection, or the mental stress incident to an illegitimate pregnancy.

From paresis the differentiation can now be made by the laboratory methods at our disposal. It must not be overlooked that the precox may have syphilis and therefore a positive Wassermann in the blood serum. In fact, not a few do show this reaction, and it is quite readily conceivable that the presence of an uncured syphilis may well be a precipitating factor in the outbreak of the psychosis in a certain proportion of cases.

From the toxic psychoses, particularly from alcoholic deterioration the differentiation is often quite difficult. It must be borne in mind in this connection that the relatively normal man deteriorates very slowly from the use of alcohol, while one finds in the records of precox cases who have indulged in alcohol that the deterioration has come very much earlier. In addition to this it will be found that the amount of deterioration in the precox case is very much greater than could reasonably be explained by the alcoholic indulgence of the patient. When this discrepancy in the history is found one is justified in suspecting that one is dealing with a fundamentally more serious condition than mere alcoholism. These two types of cases show a characteristic type of defence which serves to separate them, the one from the other. In both instances the patients tend to minimize the true etiological factor and to exaggerate the unimportant one. For example,

the precox case will always exaggerate the amount of alcohol he has been taking, while the alcoholic will always minimize it. This is of course only roughly true but it may be helpful in sizing up patients.

The whole question of alcoholic deterioration is by no means settled. Bleuler[1] is inclined to include the alcoholic hallucinoses in the dementia precox group and at least it must be acknowledged that many alcoholic patients after repeated attacks of acute alcoholic psychoses undergo a deterioration which is precox in character.

The difficulty of differentiating between defect due to precox and congenital defect has already been mentioned in discussing precox in children, and precox on a defective basis. A material help in making this differentiation is an inquiry into the school knowledge. The school knowledge will be reasonably well retained in the precox, while it will not have been acquired in the defective.

As was insisted upon in the beginning, the hebephrenic and simplex types frequently complain of numerous minor ailments. These are frequently treated by the general practitioner or the specialist on the basis of their physical rather than their psychical character. Such patients repeatedly go through complicated systems of treatment until the true character of their illness has become evident, when often much valuable time has been lost.

Treatment.—Dementia precox has generally been considered to be a hopeless condition for which little or nothing could be done. This is at least not an attitude with which to approach a patient, and when we bear in mind the considerable number of recoveries that take place in the disease it is hardly an attitude that is warranted. The treatment, however, must of necessity be very difficult, because conditions that have to be met are multitudinous and range all the way from disturbances at the lower physiological levels through distinctly psychological problems to the relation of the individual to his social milieu. These matters may be taken up in their order.

Treatment of Physical Conditions.—Here the treatment must be practically entirely symptomatic. The bladder and rectum must be carefully watched where there is negativistic retention. The mouth must be kept clean, swabbed out with listerine or some other antiseptic mouth wash, if there is retention of saliva or food particles, otherwise putrefactive changes may occur, infections of the gum with ulcerations may result, with possible complications, such as pneumonia and death. Other such conditions as these have to be met in a practical and common sense way and need little special comment; for instance, surgical injuries, such as self-mutilations, bruises, and the like have to be met in the usual way, while it is especially important to get these patients out of doors and not permit their seclusive tendencies to further the development of tubercular disease.

[1] Dementia Precox oder Gruppe der Schizophrenien. Franz Deuticke, Leipzig u. Wien, 1911. This work is by far the most comprehensive and complete treatment of the whole dementia precox problem extant. An excellent review of the work by August Hoch will be found in the Review of Neurology and Psychiatry, June, 1912.

At the present time a good deal of attention is being paid to the internal secretions. These may be investigated in the individual case, but as yet their beneficial action is unproved.

Treatment of the Mental Condition.—The treatment of the mental condition resolves itself into the treatment of disturbances at lower and higher levels. In the disturbances of the lower levels we have characteristically the excitements and the stupors. In general the treatment of the excitements should be by hydrotherapy, the continuous bath or the pack, according to the individual experience of the physician or the convenience of the hospital. Restraint, either physical or chemical should be avoided if possible, resorting to drugs only in so far as may be necessary to produce a sufficient amount of sleep. It must be borne in mind that all of the drugs that are used for chemical restraint, more especially those belonging to the belladonna group, are delirium-producing and therefore tend to interfere with the adjustment of the patient to reality. Small doses of atropine are not contraindicated to obtain vegetative nervous system control of certain annoying somatic symptoms.

In conditions of stupor the general health has to be carefully looked after. The patient must often be tube-fed, bowels and bladder require special attention, and the position of the body should be changed sufficiently so as to prevent pressure upon any portion of the skin surfaces or a tendency to hypostatic congestion of the lungs in weakened patients. Cleanliness and regular and sufficient feeding are the essential things, and it is very desirable in addition to wheel the patient's bed out upon the open porch where he can have abundance of fresh air, if this is possible.

Treatment at the higher psychological levels has the same thing to be said for it as psychotherapy has for the psychoneuroses. While it cannot be expected that patients can be cured by psychoanalytic treatment as they are when suffering from the psychoneuroses, still it must be remembered that all of the symptoms of a precox case are not necessarily at the same level, and while the patient may not be susceptible of a cure, still many of the symptoms may be largely relieved, if not dispersed altogether. Psychoanalysis therefore should be used and even where the therapeutic effect may not be especially great, still it furnishes that sort of information about the patient which it is essential to have in order to deal with him intelligently. One must know something about the factors that produced the breakdown, one must know something of the nature of the complexes, in order to go at the problem of regulating the life of the patient, not only intelligently but with any possibility of accomplishing anything. To approach the problem in any other way is to approach it blindly.[1]

In the old cases of precox that have become considerably dilapidated, that are relatively inaccessible, it would seem that the best

[1] Jelliffe: Treatment of Dementia Precox, International Clinics, 1917. Campbell: Modern Treatment of Nervous and Mental Diseases, White and Jelliffe, Lea & Febiger, 1913.

method of approach was through the agency of industrial training. If an effort be made in this direction intelligently after a sufficiently careful analysis, so far as possible, of the particular condition so that it will appear what is the best method of approach in order to arouse the interest and fix the attention of the patient, a great deal can be accomplished in making this class of patients generally useful about the hospital and in limiting their destructive and filthy tendencies and adapting them to a much healthier series of adjustments than if they are merely left to themselves.

The Treatment of the Social Relations.—The endeavor to modify the environment of the patient must be guided by what has been found as the result of psychoanalysis. The precox splitting goes back to the early infantile situations, the time when the love of the child was given out in its entirety to the immediate members of the household, father and mother, brothers and sisters. Later on if the child is to become an efficient adult he must emancipate himself from the thraldom of this affection. This, however, is what the precox has not been able to do, and the affection which binds him to his infantile loves is quite truly felt as a destructive force that prevents his onward progress in the world. There frequently results, therefore, all sorts of antagonisms addressed to the members of the immediate household which are variously expressed and variously symbolized and distorted. The love of the little child for the parent or the brother or the sister when it breaks through from the unconscious into the conscious life of the adult, is not understood at its true value, but becomes a hateful thing, and so characteristically there arise all sorts of incest fantasies. Recently one such patient in the hospital struck and stunned another patient and when called to task about it he instantly protected himself by the statement that "they" were accusing him of incestuous relations with his sister. These are the commonest of ideas among precox cases. Psychoanalysis will orient the physician with relation to these ideas and enable him to adjust the patient intelligently. Such ideas frequently require the removal of the patient to an institution.

Although a wholly pessimistic attitude is not warranted in approaching the problem of precox in an individual case, still it must be realized that after all one can hardly expect a complete recovery. One can only hope to readjust the situation so that the patient may get along comfortably and perhaps lead a useful life, probably upon a slightly lower level. The tendency of the disease is essentially to limit the individual in the mental sphere, and this limitation means of necessity that life has to be led at a correspondingly lower and simpler plane of adjustment. This is exactly what the institution provides for the patient, but there is no reason, in the absence of dangerous tendencies and if it is desirable from other standpoints, why an attempt should not be made in this direction outside of an institution. We know this can be done, for we see not infrequently patients getting along very well under the solicitous care of some relative, for example, and when

this relative dies and the care is removed and their world is no longer carefully arranged for them, then they find their way into the hospital.

A careful dealing with all of the conditions surrounding the patient, which may have to include the other members of the family, more particularly dealing with them intelligently as the result of such psychoanalysis as can be made, will enable the physician, in a very considerable proportion of cases, if the means are at his disposal, which of course they frequently are not, to so adjust the situation as to bring about a state of relative calm and quiescence, with the preservation of a considerable degree of efficiency.

Prophylaxis.—The prophylaxis of dementia precox is a most difficult problem, and in the first instance of course should be met from the eugenic standpoint. Marriage should be very carefully supervised where the individual comes from badly tainted stock. Such general principles may be borne in mind, as for example, the liability to mental disease in children from tainted stock is greatest among the earlier born and falls off rapidly, as Heron has shown, particularly after the fourth child, while Mott in working out his Law of Anticipation claims that if the individual passes the twenty-fifth year the liability to a mental break-down is very materially lessened. This Law of Anticipation, which has been rather generally accepted, has, however, been seriously questioned. Karl Pearson[1] pointed out that a person who develops mental disease at an early age is not so apt to become a parent and therefore, the parents constitute a group selected on the basis of age. Heron has also indicated what he believes to be other statistical fallacies.

The possibilities of prophylaxis before the outbreak of the psychosis are not known, yet it would seem that it would be rational to endeavor to deal with those character anomalies that we know favor this type of disorder. The method of approach will of course resolve itself into an attempt to define the lines along which frank, open reactions do not seem possible to the individual, particularly along lines of definite sex conflicts. In this particular the whole matter of sexual education has to be gone over and its value as a prophylactic measure determined.[2]

[1] Holmes, S. J.: The Trend of the Race, New York, 1921.

[2] See Jelliffe: Predementia Precox, Am. Jour. Med. Sc., August, 1907, for a study of these early features which antedate the appearance of the disease, also chapters on the treatment of Paranoid States and of Dementia Precox, by Adolf Meyer and Macfie Campbell, Modern Treatment of Nervous and Mental Diseases, White and Jelliffe, Lea & Febiger, 1913, vol. i.

CHAPTER XX.

INFECTION-EXHAUSTION PSYCHOSES.

The Toxic-infectious Group in General.—In connection with the similarity of the manifestations in this group, although the ultimate etiological factors are widely different, it is worth while to bear a few considerations in mind. In the first place it has been suggested that after the manner of thinking regarding the syphilitic manifestations and the alcoholic psychoses, that the mental picture is not dependent upon the immediate infecting agent or upon the toxin directly elaborated by the infecting microörganism, but on the contrary is due to a general disturbance in the metabolism, the result of the infection; in other words, that there is an intermediate agency at work, a metatoxin. On the other hand, it has been suggested that the gamut of symptomatology which the neuron may produce in the process of its destruction is necessarily confined within certain relatively narrow limits and that therefore inimical agencies that act at the biochemical level can only produce relatively few groups of symptom-complexes. This seems to be true in the main. On the other hand, while it will be found that if the individual cases are carefully studied the general course of the malady and the mechanisms involved are quite similar in the different cases; in other words, that the patients fall into one of very few groups; still the content of the delirious or delusional experiences and the minor variations in the manifestations in the malady must have another explanation. This explanation is naturally the make-up of the individual. A destructive agency at work in tearing down can only tear down what has before been built up. It is constrained, in other words, to deal with the material at hand, and therefore personal variations must be expected; for example, the delirious patient will weave expressions into his productions that refer directly to his own experiences.

This group of infection-exhaustion psychoses is somewhat of a heterogeneous group, including all of the mental disturbances dependent upon the various infections, as well as certain conditions which are tentatively supposed to be dependent upon exhaustion and which give similar clinical pictures. For the most part it contains the psychoses dependent upon all the febrile diseases, for up to the present time at least these different diseases cannot be differentiated by means of the mental picture alone.

In this whole group of toxic and infectious origin it has long been the aim if possible to define the psychotic symptoms as specifically as the other symptoms, to determine, for example, whether a given infection

could be diagnosed as such by the mental symptoms alone. On the whole this effort has failed and resort must be had to neurological and laboratory findings to effect an accurate diagnosis;[1] though in certain instances, particularly when there seems to be associated an organic element as in encephalitis lethargica and in paresis the psychic symptoms do seem to have a certain specificity. This is true also in the alcoholic reactions, though it is always difficult to differentiate the true individual components contributed by the personality make-up. Instead of specific types of psychic reaction there is found a generalized type of response which varies within considerable limits and which seems also to vary from similar reactions observed in the better defined psychoses. Delirium or confusion in some form is the most generalized type of reaction found to which are added affect and mood disorders, schizoid reactions (catatoniform rigidities), convulsive reactions, Korsakow-like reactions, distressed perplexity,[2] paranoid trends and perhaps many others. Here as elsewhere the affect fluctuations are indicative of benign personality trends as over against the more omnious schizoid types.

Prefebrile, Febrile, and Postfebrile Psychoses.—The same thing may be said regarding fever and infection which is said regarding alcohol, namely, that it is a measure of the mental stability of the individual. While some persons may remain mentally clear with a temperature of a 106° F., others may become delirious with hardly more than a degree of temperature. This difference is a well known one, and in a disease like typhoid fever, for example, the general consensus of opinion is that the prognosis is most serious in those cases that become delirious early and show from the beginning marked and exaggerated nervous symptoms.

Infection and Initial Delirium.—Under this head are included the mental disturbances which develop early in the infectious diseases before there has been any rise in temperature. In fact, the delirium in these cases may disappear when the fever is fully developed, although this is not the rule, the usual course being for the delirium of this period to go over into a fever delirium. This infection delirium is also found in conditions which are essentially afebrile, as for example rabies, and is there due of course to an overwhelming of the body with toxins, or perhaps to a bacteremia. This type of mental disturbance occurs characteristically with the onset of typhus, in the period previous to the eruption in smallpox, and has been observed in connection with influenza, acute chorea, especially the chorea of pregnancy, and in malaria.

Symptoms.—The symptoms of infection delirium are the usual symptoms of delirium, which may present any degree of severity, from

[1] Krisch, H.: Die Symptomatischen Psychosen und ihre Differentialdiagnose, Abhandl. u. d. Neurol., Psych., Psychologie u. ihren Grenzgebieten, No. 9. 5, Karger, Berlin.

[2] Hoch, A., and Kirby, G. H.: A Clinical Study of Psychoses Characterized by Distressed Perplexity, Arch. Neurol. and Psychiatry, April, 1919.

mild confusion to *delirium acutum*, or collapse delirium, ending in death. The diagnosis of initial delirium previous to the appearance of the typical signs of the disease of which it is a symptom is of course practically impossible.

Fever Delirium.—Fever delirium is the psychosis which accompanies febrile movement and which in general varies in severity hand in hand with the variation in the severity of the fever.

It may be described in four stages according to the degree of its severity. In the first stage headache, irritability, sensitiveness to noises and light, restlessness, and disturbing dreams; in the second stage hallucinations appear, especially in the visual field; the hallucinations are of a dream-like character, and the patient may still be made to react clearly; in the third stage the motor disturbance is greater and takes on the character of jactitation; in the fourth stage there is profound dulling of consciousness, uncertain and ataxic movements, ending in coma and death. Of course this regular progress of the delirium may be interrupted at any point by an improvement in the symptoms and recovery.

The onset and the severity of the delirium, as already set forth, show to an extent the mental stability of the patient. The delirium develops or becomes severe much more readily in the unstable and poorly organized than in the stable. The course of the delirium may be interrupted by an acute excitement followed by a stuporous condition, which Bonhoeffer[1] describes as an *epileptiform excitement*, which is followed by a dream state, for which latter he retains the name of Ziehen, *infectious dream state*. With these conditions confusing symptoms may arise which makes the diagnosis difficult for a time. Thus, along with the disorientation there may be flexibilitas cerea confabulation, perseveration.

In certain cases the orientation is less disturbed, while the hallucinations are more prominent, and there is an outward semblance of a hallucinosis.

Here, as elsewhere in psychiatry, the various forms of the psychosis are frequently designated by the prevailing symptoms. Thus, the symptoms found may be epileptiform excitement, dream states, stuporous conditions, hallucinosis, catatonic and confusional states, and the delirium may be designated by using any one of these descriptive terms.

Postfebrile Psychoses.—These conditions either develop as a result of the passing over of the delirium of the febrile state into the period of convalescence, or they may take their origin from the first, during the postfebrile period. In the latter case they would be considered as belonging more properly to the exhaustion psychoses, and yet it must be understood that the term exhaustion is a very vague one and that in all probability it implies at least the accumulation of toxins.

The characteristic picture of this psychosis is associated with the

[1] Die symptomatischen Psychosen.

great physical debility which follows the subsidence of the fever in an infectious disease. The patient is weak, tremulous, exhausted, and complains of being tired. The mental state is fundamentally one of weakness. There is no confusion, but the capacity for attention is very much reduced, and in that way there come about apparent memory disturbances. The patient is unobservant of his surroundings, usually somewhat depressed, and may be very much occupied with his own bodily feelings to the extent of having hypochondriacal ideas, and fleeting hallucinations are not infrequent. In more severe cases there may be more evidence of mild delirium or confusion, the mood may be more definitely anxious and fearful and there may be delusions characteristically of the persecutory type. The patient is apt to be irritable, cross, and complaining. Occasionally there may be considerable motor excitement of an epileptiform character, and sometimes actual delirious dream states. Occasionally the picture of a mild expansiveness is found. A certain number of cases present a well marked Korsakow syndrome.

Exhaustion Psychoses.—The term exhaustion in this connection is not intended to be used in other than a tentative sense. The whole question of fatigue, except with reference to very specific problems, such for example as muscle fatigue, is still little understood. In general, however, it may be said that there are two factors in the problem, the negative and the positive. The former is the result of the actual wearing out of a substance in the body, as for example muscle, while the second is the result of the formation of certain poisonous substances which result from the breaking down of tissue. In the conditions which ordinarily are observed it would seem evident that both of these factors, the positive and the negative, are in evidence, and exhaustion is considered where they appear to be the predominating factors. For example, exhaustion is spoken of when coming on acutely as the result of a sudden loss of a considerable quantity of blood, of coming on more slowly as a result of the debilitating effects of a chronic disease such as carcinoma. In this latter case, however, it will be easily seen that in all probability the toxic element must enter. Similarly in convalescence from acute illness where the fever has been very high and the illness has been prolonged, conditions accompanied by great physical prostration are seen in which it seems fair to assume that the element of direct exhaustion is responsible very largely for the mental picture. Under the head of exhaustion psychoses two main types will be described—collapse delirium and acute hallucinatory confusion (amentia)—but it must be understood that these two psychoses are not necessarily peculiar but only conditions in which the exhaustion element appears to predominate. Either or both of them may be found during the period of acute infection in the febrile diseases, and conditions that begin with infection and fever and give the picture of an infection or of a fever delirium, may go over into the severer type as the patient's general condition becomes worse and the organism is overwhelmed with the poisoning.

Collapse Delirium.—This is the *delirium grave*, or the *acute delirious mania* of the older authors.

The disease may present a prodromal period of restlessness, irritability, and insomnia, after which a condition of mild confusion may develop with only a slight degree of perplexity and perhaps fleeting hallucinations, slight clouding of consciousness, disorientation and dreamy delusions; psychomotor excitement is common at this time, the patient being active and perhaps inclined to actual violence and destructiveness. Often associated in the early symptoms are accesses of anxiety, amounting at times to actual terror. This condition gradually becomes worse, and finally the degree of excitement becomes very great, exceeding anything that we usually see in the other psychoses. When this extreme form of excitement is in evidence the outcome is usually apt to be serious, and the older writers believed it to be uniformly fatal.

In these severe cases the incoherence becomes absolute, the disorientation complete, the clouding of consciousness profound, the temperature generally runs high, perhaps as high as 106° F., gastro-intestinal symptoms are common, there is usually almost complete anorexia, coated tongue, offensive diarrhea, a high grade of indicanuria, and rapid emaciation which results in a high grade of exhaustion, with typhoid symptoms. There may be a certain amount of catalepsy, with stereotyped movements, grimacing, and echolalia, and stuporous conditions; coma and death not infrequently result.

This condition occurs with all degrees of severity, and while the most severe cases die, the milder ones make good recoveries.

It may often be quite impossible to make a diagnosis of the physical condition of the patients while in their excitement. It must be remembered, especially if the patient has temperature, that these conditions are pretty apt to be dependent upon some acute physical illness, particularly an infection, and that not infrequently a deep-seated pneumonia, concealed from the usual approach by percussion, and auscultation, is at the basis of the difficulty, while an infection such as grip, rheumatism, or the like may also be an etiological factor.

That the element of exhaustion is after all not the only element, and perhaps not the most important element, will be appreciated if one stops to consider that the great majority of patients do not react in this exaggerated way to acute toxemias or infections. In all probability the fundamental factor at the bottom of such a reaction is the individual make-up, just as an ounce of whisky will disorganize one individual as much as a pint will another. The personal factor is the important one.

Acute Hallucinatory Confusion (Amentia).—This psychosis is less acute in its characteristics than the former. The symptoms are those of a mild confusion with incoherence and a considerable degree of perplexity. There are usually fleeting hallucinations in the various sensory areas, sometimes delusions, which, however, are not characteristically fixed, with a more or less changeable emotional attitude

varying with the content of the delusions. The patient is characteristically in a condition of mild motor unrest.

The duration of the illness is relatively long. It may be prolonged over several weeks, is usually from one to three months in duration, and may be considerably longer. The course of the disease is not infrequently interrupted by lucid intervals, during which the patient is quite clear. These may last anywhere from a few minutes to a day or two, and then the patient will lapse back into his previous condition of confusion. This is an important point to bear in mind.

A very marked degree of perplexity is rather characteristic with these patients. There is considerable disturbance in their perception of their environment which they do not seem to understand, things about them appear to be changed, they appear to be in some strange place, things are not right, they do not understand the meanings of things, they get mixed up, get the wrong bed, and act in similarly stupid ways.

Bonhoeffer[1] describes hallucinatory and psychomotor catatonic forms, in the latter of which flight of ideas and incoherence predominate.

In addition to the previously described psychoses, Bonhoeffer[2] describes, as occurring late in the course of the infectious diseases, and therefore it would seem dependent to a certain extent at least upon exhaustion, a *hyperesthetic emotional state of mental weakness*, associated with physical symptoms, such as severe headache, paresthesias, and pains in the joints, great prostration, oversensitiveness to noise and light, easily frightened, troublous dreams. He also describes an *amnesic variety* resembling Korsakow's psychosis. Acute delirium may also develop and one may see a meningitic form with very severe delirious reaction.

The possibilities of local injuries to the brain, such as brain abscess and local meningitis must be held in mind. When these develop in children they not infrequently leave sequelæ, such as mental defect and epilepsy.

Treatment.—The treatment of all of these conditions is, of course in the main the treatment of the underlying disease. In general however, it may be said that for the excitement the continuous bath or the wet pack, with perhaps the occasional exhibition of a hypnotic should be chosen rather than the constant drugging of the patient, so frequently employed.

When there is marked lack of desire for food and the condition is serious and approaching one of profound exhaustion there should be no delay in resorting to artificial feeding. The rectum, of course, may be used if the stomach is very irritable, but should be a last resort. It is preferable to give small quantities by the stomach frequently, and endeavor in that way to deal with the situation than to give rectal feeding, with the usual result of being satisfied with the giving of an amount of food which is really very inadequate.

[1] Op. cit.

[2] Op. cit.

In the extreme exhaustion of the later stages, especially when there is marked dehydration, hypodermoclysis often gives most excellent results.

Typhoid Fever.—An initial delirium in typhoid fever is generally conceded to be of most ominous import, while an early delirium which is continuous and severe is also of bad prognostic significance. With this disease the good effects of the bath treatment, as used in the Brand method, is especially well seen. Cold baths for the reduction of temperature have as one of their most important results their sedative and calming effect upon the nervous symptoms.

The possibility, in all such diseases as this, of the localization of the infection in the meninges should be thought of, and in cases of marked delirium, therefore the possibility of a meningitis should be borne in mind. Lumbar puncture may be valuable under these circumstances, both for diagnostic purposes and for the relief of pressure.

In the late stages, during convalescence, special efforts should be made to make the dietary as full and liberal as possible.

Erysipelas, the various *exanthems,* and the several types of *malaria* are also not infrequently complicated by mental symptoms, while *influenza* often produces profound depressions associated with great physical exhaustion hanging over during a long convalescence. We must remember in all of these conditions, particularly in the exanthems, the possibility of meningeal focalization. Influenzal infections sometimes also produce meningeal inflammation.

Acute articular rheumatism is especially important in this connection because of its relation to acute chorea and the so-called *chorea insaniens.*

Pneumonia is frequently associated with mental symptoms. Very many of the cases of delirium tremens owe their severity to a pneumonia, usually a masked form of this disease, either a central pneumonia or one located in the upper lobes, and therefore not so readily diagnosed.

In connection with all this class of diseases the many complications must be borne in mind which may arise in their course and upon which the mental symptoms may depend other than the meningitides, as for example, the acute types of nephritis in connection with scarlet fever, the middle-ear complications of measles, and the endocardial complications of rheumatism.

Influenza.—Influenza, because of its wide prevalence, has presented the evidences of the relation of infection to the origin and modification of the psychoses in a manner which is perhaps less equivocal than is the case with any other infectious disease. It acts in three ways:[1] to create psychoses, to precipitate psychosis in predisposed individuals and to augment or alter their form when already existing. Delirium is the most frequent mental picture produced by influenza, as by other infections, and the outstanding fact of its greater frequency than other

[1] Menninger, Karl A.: Psychoses Associated with Influenza, Archives of Neurol. and Psychiatry, 1919, vol. 2,

types of reactions argues, here as elsewhere, against the qualitative psychic specificity of various infectious and toxic factors.

Menninger gives the following examples of the three groups above mentioned. The conditions created by influenza: Delirium (simple, errant, schizophrenic), apoplexy, atypical psychoses, senile psychoses, schizophrenia, cyclothymia, hysteria. The conditions precipitated by influenza: delirium tremens, schizophrenia, cyclothymia, psychoneurosis. The conditions altered by influenza: defectiveness, epilepsy, psychopathy, apparent normality, neurosyphilis. In this last group he reports the case of a moron who was reduced, following influenza, to the level of imbecility; and the case of an apparently normal person (latent syphilis) who developed paresis following influenza.

CHAPTER XXI.

THE TOXIC PSYCHOSES.

Alcoholism.—It is generally conceded that alcohol is a powerful poison and as such if taken in large quantities or over a long period of time produces serious damage to the individual. There is, unfortunately, associated with this view the opposite view that alcohol is a valuable medicine in certain conditions, particularly that it is a valuable stimulant, while among certain peoples it is generally accepted as an essential article in the daily dietary.

As a matter of fact the toxic properties of alcohol far outweigh any possible beneficent effects that it may have. In fact, it is questionable whether alcohol should be considered in any other sense than as a poison. It has no special medicinal properties that are of value, it is not a stimulant, and at most might be considered as a hypnotic, especially in old people with some arteriosclerosis. Other drugs can be used, however, quite as well and more safely. It is probable that the widespread belief in its efficacy for all sorts of conditions is based upon an effort at justification for its use.

The part that alcohol plays in the production of mental disorders is extremely difficult to express in definite terms. It has been claimed that 12 to 15 per cent. of the psychoses are dependent upon alcohol as the principal etiological factor, and yet any such figure as this is extremely misleading, for it is really not known how alcohol brings about its results, and especially it is not known whether it is primarily the alcohol which is to blame or whether it is secondarily the metabolism disturbances which are produced by its continuous use. It is of the highest significance that of the cases that come to autopsy a very considerable number of them in the general hospitals and poor-houses are found to suffer from cirrhosis of the liver, while in the hospitals for the insane this condition is of rare occurrence. The indication is very clear that the psychoses are dependent upon some peculiarity of make-up of the individual which is affected in an exaggerated way by alcohol or of which alcoholism is the expression. If this is true, then the alcoholism is only a surface indication, and the true etiological factors lie deeper.

Psychology.—There are many types of persons who drink and there are many reasons for drinking. The usual reasons given refer to social conditions. One is rather taught to believe that the social demands are of such a nature as to require a certain amount of drinking, and that this drinking once started tends to perpetuate itself and to finally become a fixed habit. This way of looking at the situation, like the

belief in the beneficent effect of alcohol, is very largely at least an effort at justification. The social conditions cannot be changed. They have to be submitted to, and therefore the drinking is inevitable and not the fault of the individual. This really offers no adequate explanation, and in all probability there is very little truth in the statement. People do not drink simply and solely because they have acquired a habit of drinking. The habit element is the least important in the whole situation, and if that were the only thing to be dealt with the problem of alcoholism and of other habituations would be relatively simple. People drink because of definite returns which they get from drinking. A given number of persons all placed under the same conditions, social, etc., do not all drink. It is only some of them who drink, and those who drink do so not only because they get definite desirable results, but because those results are practically necessary for them. In other words, far more important than the question of habit formation, is the question of the individual psychology. Here, again, as has been seen already in dealing with the psychoses, the question of mental conflicts is most important. When the individual is confronted by situations to which he cannot adjust adequately, when the world of reality makes demands which are too great for him to meet, one of the ways in which the individual reacts to such a condition is by narcotizing himself and so withdrawing from the whole situation. Alcohol then becomes an agent which helps the patient to get away from the conflicts thrust upon him by reality, it helps him to withdraw within himself, helps him to live in the world of fantasy where things come true as he wishes them. Under these circumstances it can be seen why what appears to be a habit is formed. The moment the individual, harassed by the absolutely inacceptable demands of the world, finds an avenue of escape in which he can rest from their harassings, finds the possibility of peace, of repose, he finds it equally impossible to resist the temptation to avail himself of it and of course he usually continues to avail himself of it. He is rendered more and more incapable of meeting the conflict efficiently. Therefore a vicious circle is established and the individual is hopelessly involved.[1]

From this psychological point of view alcohol is seen to be an agent that breaks down sublimations and permits regressions. The drinking of alcohol is therefor a concession to the demand for regression and the acute alcoholic psychoses are thus more properly of psychogenic rather than of toxic origin. Alcohol in this sense can hardly be considered a toxic agent at all. This concept of the action of alcohol is borne out by the fact that not infrequently the same symptoms that led to the diagnosis of an acute alcoholic psychosis (see alcoholic hallucinosis) have been seen to supervene without alcoholic indulgence.

Aside from the class of individuals described above, it must be constantly borne in mind that indulgence in alcohol is oftentimes the expression of a neurosis or a psychosis. For example, the recurrent

[1] See Jelliffe: The Mentality of the Alcoholic, N. Y. Med. Jour., April 7, 1917, for discussion of psychoanalytic principles concerning the deeper psychology of alcoholism.

attacks of manic-depressive psychosis may be ushered in by alcoholic indulgence, and if one is not keenly observant he may easily suspect that he is dealing with an alcoholic psychosis rather than with a manic-depressive. The same thing of course may be said of dementia precox which is often found associated with alcohol, and with paresis, which not infrequently has a history of alcoholism in its early stages. Then it must be remembered that in certain post-traumatic conditions, that is, cases following, especially, head injury and sunstroke, and in arterio-sclerotic and senile conditions the patient may react in an exaggerated way to alcohol and that very small doses may produce very pronounced effects. This is true also in connection with certain other conditions, particularly imbecility and epilepsy. Here pronounced reactions to alcohol are found, and it is necessary to evaluate the importance of the alcohol in the entire situation.

Finally, there are certain psychoses which appear to be essentially alcoholic; to depend upon prolonged indulgence in alcohol. Of the acute conditions due to alcohol drunkenness is the most typical, while of the chronic conditions dependent upon alcohol, delirium tremens, alcoholic hallucinosis, and Korsakow's psychosis are the most important. These latter because they appear to be dependent upon something other than simple alcohol, as they never occur as the result of single large doses, but can only occur in persons chronically addicted it has been proposed to call, after the manner of the psychoses due to syphilis, the meta-alcoholic psychoses. With this introduction a short description of each of the mental pictures dependent directly or indirectly upon alcohol will be given.

Drunkenness.—The general phenomena of drunkenness are too well known to require detailed description, except that perhaps the less evident manifestations are not generally thought of. The progressive disturbance of coördination of the motor centers, the higher being thrown out of adjustment first, and the progressive disturbance of the sensory apparatus in the same way produce disturbances, the latter of which are entirely subjective, and so are not generally observed. The *mood* of the drunken man is variable. Each man reacts to alcohol in a way peculiar to himself, but in a general way there may be considered to be two classes into which cases of drunkenness may be divided, the *exalted* and the *depressed*, the former closely resembling the manic phase of the manic-depressive in his extreme loquaciousness and hyperactivity, while in the latter the patient withdraws within himself, being sullen, morose, and disinclined to associate with others.

One of the important considerations under this head is the degree of ease with which the individual is seen to react to alcohol. Alcohol, like fever, may be used to express the measure of cerebral resistance; unstable and defective individuals reacting to both in an exaggerated way.

Pathological Drunkenness.—Drunkenness that exhibits unusual features, which leads the individual to perform strange acts or acts of violence or which produces serious physical symptoms is known as

pathological drunkenness. Persons in this condition may develop well marked hallucinations or delusions to which they tend to react, while convulsive attacks are not infrequent, and the whole period is often obliterated from the memory by an amnesia.

Chronic Alcoholism.—Uncomplicated habitual drunkenness, that is, inebriety not dependent upon some definite underlying condition, such as manic-depressive psychosis or paresis, has the earmarks of a psychoneurosis and is therefore dependent upon the character make-up of the individual for its cause. These earmarks, which the alcoholic well shows, are the feelings of inefficiency as a result of which the alcohol is taken as an effort at finding safety—it is the means of a flight from reality.

It must be recalled that many psychoneurotics whose compulsive flights are alcoholic—and such is usually the flight from an unconscious homosexual conflict—are very superior types of people.

The life history of many an alcoholic shows him to be an inefficient individual. He is incapable of meeting reality efficiently every day. He may be able to deal with the problem of reality for a greater or lesser length of time, but continuity of effort, day in and day out, is foreign to the alcoholic character. He can stand the strain only about so long, longer in some cases than in others, but the principle is the same. This is the inefficiency Adler believes is dependent upon organ inferiority, or to use an older and more tried expression, it is constitutional. The reaction to such a feeling of inferiority drives the inefficient individual to find some way of escape from the horrid facts, the overburdening oppressions of reality. This he finds in alcohol which dulls his perception of reality and permits the world of fantasy to reign supreme. In this fool's paradise the alcoholic finds temporary surcease from the burdens he is but poorly equipped to bear.

The effects of alcoholic poison may be exhibited in any organ of the body, more particularly the central nervous system, the stomach, the liver, the kidneys, and the bloodvessels. The effects on the nervous system are shown in various disturbances of sensation, such as the paresthesias, amblyopia, amaurosis, dulness of hearing, of touch, etc., while in the motor realm we find tremor and epileptiform attacks, with general motor enfeeblement characteristically. The mental changes are gradual and progressive, the intelligence is blunted, the judgment is impaired, the moral sense dulled, while actual delusions not infrequently develop.

While all of these changes may occur in chronic alcoholism, it is usual to see in individual cases one organ more especially selected out by the alcohol for its destructive effects. With the beer drinkers who absorb several liters of beer each day, cardiac hypertrophy is quite common. Some patients develop serious kidney complications, while others are able to drink over long periods of years without any material impairment of the kidneys. The same remark applies to the liver, while the effects upon the nervous system and the bloodvessels are equally varied. It is characteristic, too, that the individual as a

whole varies in the destructive effect which alcohol has upon him, some individuals apparently being able to use large quantities over considerable periods of time without noticeable impairment. The general outward appearance of health, however, which many indulgers in alcohol show is sometimes rudely dissipated when they are attacked by an acute illness, for however well they may have been able to go on with the routine day's work, their weakness is shown when they are subjected to the special stress of, for example, a pneumonia, and the death-rate in this class of persons is much higher than in abstainers.

Delirium Tremens.—Delirium tremens, while an acute manifestation of alcoholism, can only occur in a person suffering from the effects of chronic alcoholic poisoning. It may manifest itself sometimes as the result of a prolonged debauch, sometimes as the result of an illness or injury in a chronic alcoholic. It has often been maintained, and is still believed by many, that the delirium is not infrequently the result of a sudden withdrawal of alcohol, as for example when a patient is taken to the hospital for an injury nothing is thought of his alcoholic tendencies until he develops a delirium. In these cases the delirium is presumed to be due to the fact that the patient did not get his usual supply of alcohol. There is no good reason for this opinion, and it must be remembered that in the prodromal period of delirium tremens not infrequently the patient has experienced a disgust of liquor for a number of days. A pretty effective negation of the theory of an *abstinence delirium* is derived from the English prison statistics, which show that in the year 1907 there were 63,000 inebriates who were suddenly deprived of alcohol by confinement, and in this number only 246 developed delirium tremens, less than one-half of 1 per cent.

Symptoms.—The symptoms of delirium tremens may come on slowly being preceded by several days of general physical upset, with transient sensory falsifications and perhaps delusional interpretations, which show a special tendency to come on at night or under conditions in which accurate perception is interfered with. On the other hand, the delirium may come on very rapidly. A case is recalled of a longshoreman, a man of perhaps thirty-five, a giant in physique, who walked into the hospital at noon with a sprained ankle; the ankle was strapped, and he was put to bed. That night he was in the wildest delirium, and the next morning he was dead.

Following these prodromal symptoms the delirium appears in its complete manifestations. It is a typical toxic delirium with multiform disorders of the sensorium. The hallucinations predominate in the visual sphere, although tactile hallucinations are very common. The patient is manifestly very sick, he is greatly depressed physically, he is tremulous, the tremor being so constant as to have given the name to the condition, and his mood is characteristically one of apprehension, anxiety, and fear. He is disoriented, mistakes the people about him; not infrequently his delirium is an *occupation delirium* and he believes himself back at his accustomed work, but it is characteristically filled with fearsome sights, and he is in constant terror from the invasion of the numerous animals that he may see about him.

In the somatic realm the pulse is rapid, the tremor constant, the skin bathed in perspiration, the tongue foul, the appetite *nil*, and there may perhaps be a marked grade of albuminuria. The temperature is characteristically either normal or below normal, but it may be high, in which case we have what is known as the *febrile type* of the disease, which is generally fatal. All sorts of complications may naturally occur. Acute cardiac dilatation sometimes causes death, while one of the most frequent of the complications in severe cases is "wet brain." In this condition the patient sinks into a low muttering delirium, the temperature falls to subnormal, the face is pale and bathed in cold perspiration, the pupils dilated, there may be some rigidity of the neck with a tendency to a bending back of the head; the patient sinks into a comatose condition and dies.

The psychosis runs an acute course of about three days and usually terminates by a long sleep in recovery. Ten to 15 per cent. die.

Treatment.—The treatment of delirium tremens should be supporting, liquid concentrated food, predigested if necessary. The bowels and the kidneys should be freely flushed, heart stimulants are necessary to prevent cardiac failure, and hypnotics are often required to produce rest. For the excitement, hydrotherapy in the form of the continuous bath, or if this is not available, cold packs are preferable to drugs. The constant thing to be kept in mind is the support of the strength of the patient, and the logical way to accomplish this end is by feeding. If the patient refuses food no time should be wasted. He should be immediately fed with the tube, preferably as often as three times a day in small amounts, watching the stools and governing the quantities given in each feeding by the amount the patient is able to digest. If the patient is unable to retain the food owing to acute irritability of the stomach and constant vomiting, feeding by the rectum should be resorted to, while if the prostration is extreme and there is emaciation and the deprivation of fluid considerable, hypodermoclysis is a valuable adjunct.

Korsakow's Psychosis.—This psychosis is found typically in association with alcoholic polyneuritis, although the same mental state may be found with a polyneuritis of different origin, as for example the metallic poisons, some of the infections such as tuberculosis and influenza, and some of the endogenous toxins as in diabetes. The syndrome occurs also in connection with general organic changes of the central nervous system as in paresis, arteriosclerosis, and senility. In the latter case it is a part of the clinical picture of presbyophrenia.

While this psychosis occurs typically in connection with polyneuritis, the evidences of a polyneuritis may be very slight. They should be carefully examined for, especially by pressure over the large nerve trunks, which will often elicit painful points, rather typically at the points of exit through the bony foramina. The Lasègue sign should be sought for.

Korsakow's psychosis is sometimes designated in contradistinction to delirium tremens, which is spoken of as an acute alcoholic delirium,

as a chronic alcoholic delirium. In fact the attack may begin with a typical delirium tremens which merges into the chronic delirium of Korsakow's psychosis instead of clearing up, although this is not the usual mode of onset. Not infrequently, however, a history of previous attacks of delirium tremens may be elicited.

The mental symptoms are the result of a combination of attention and memory disorders. The memory disorder is of the nature of a lack of impressibility. The result of this combination is a special type of amnesia. There is defect in the recording of current events. The patient is usually disoriented, to some extent at least, and the things that have recently happened cannot be recalled. These defects of memory are characteristically supplied by fabrications of all sorts of degrees of probability and grotesqueness, which are usually related by the patient with a composed bearing and with every outward appearance of relating facts, or at least occurrences which he himself believes. These fabrications do not correspond if the patient is asked at intervals about occurrences covering a certain period, and they may often be guided by suggestions from the questioner. Not infrequently the patient in his fabrications invents occurrences which account for his symptoms, for example, a patient who is suffering from a certain amount of pain in his legs as a result of his polyneuritis, even though he may have been confined to his bed for weeks, will say that he was out that morning and climbed a long hill and tired his legs out, and in that way he accounts for their aching.

The experiences which these patients relate are of a delirious character and not infrequently it is rather difficult to distinguish the fabrications from the dreams, and it would appear that the two often merge into one another.

Physically the patient usually presents the signs of a polyneuritis, although these may be very mild. When the neuritis is severe, foot-drop and wrist-drop are characteristic signs, as the nerves of the extensors of the forearm and leg are most characteristically involved in alcoholic neuritis.

The serious complications of the disease are either dependent upon intercurrent affections such as pneumonia or upon the involvement of important nerves such as the vagus, vagus involvement of course being pretty apt to be fatal. A serious complication is a neuritis of the phrenic nerve with paralysis of the diaphragm on one or both sides with resulting tympanitis and serious embarrassment of cardiac and respiratory activity.

Pathology.—The pathology of the disease shows that it is by no means confined to the peripheral nerves nor even to the motor areas of the central nervous system, but that there is a very general involvement of the entire central nervous system, the cord, basal ganglia, and the cortex all showing lesions, although there appears to be a tendency to the focalization of the pathological process. As a result certain focal symptoms as aphasia, apraxia, hemianopia, etc., are found. This wide distribution of the pathological disturbances and their ten-

dency to focalization is due to the fact that the disease involves the bloodvessels. The smaller vessels proliferate and present evidences of endo-, meso- and periarteritis with frequent ruptures. When this condition is focalized in the midbrain and gives rise to various ocular palsies the syndrome of *acute hemorrhage polioencephalitis of Wernicke* is present. This is not infrequently observed in chronic alcoholics. The condition is typically ushered in by severe and prolonged vomiting, which is followed by marked confusion with delirium, vertigo, sometimes headache, and somnolence. Optic neuritis may be present. Death usually occurs in from one to two weeks.

With a disease of this character having a pathology so widely distributed throughout the nervous system and dependent upon chronic toxemia it can be understood how very many *types* have been described. These types or clinical forms are nothing more than descriptions of the disease in which certain symptoms are especially prominent, and so there are amnesic, confusional, delusional, anxious, demented, delirious, stuporous, and hallucinatory forms described by different authors.

Treatment.—The treatment of the condition is of course the withdrawal of alcohol and the treatment of a general toxic state along supportive lines. The deformities which are due to the paralysis should be dealt with by recognized orthopedic measures. It should be remembered that while the patient is in bed and delirious the weakened extensors of the forearm and leg should not be stretched by the weight of the limb, but should be supported, particularly the weight of the bed clothes should be removed from the feet.

Alcoholic Hallucinosis.—This condition is also an expression of chronic alcoholism and may be preceded by attacks of delirium tremens. It is characterized by hallucinations, auditory predominating, in this respect strongly contrasted to delirium tremens, and delusions of a persecutory character which harmonize and are explanatory of the hallucinations. It is quite characteristic that the hallucinations and the delusions deal with sexual matters, the patient frequently being abused by "the voice" for committing some sexual crime or is accused of sexual perversions. The delusions are practically invariably persecutory, although occasionally expansive elements may enter the picture. Not infrequently, too, the patient is very much frightened, as when he thinks he is being closely pressed by his persecutors who are determined upon taking his life. There are not a few borderline mixtures of this psychosis and delirium tremens.

The condition is essentially an acute paranoid state and as such its explanation is the same as the explanation of paranoia. In other words, there is an unconscious fixation at the homosexual level, and this accounts for the very great frequency of the sexual character of the hallucinations and the references to sexual perversions. The alcohol which is taken by the individual because he cannot deal efficiently with reality causes him to turn back upon himself and produces a reanimation of this early fixation, and then the mechanism

of projection comes in as a distortion to make it appear that the suffering has its origin from without.

A recent analysis of cases by Schneider[1] has brought out the make-up of the individual in a type of psychosis which has too often been thought to be sufficiently explained by alcohol. Aside from the fact that hallucinosis occurs aside altogether from alcoholic indulgence he found that repeated debauches may terminate without hallucinosis, that this only supervenes as the result of a special precipitating factor. The precipitating factor is of psychogenic nature, mental shock followed by worry and the content of the hallucinosis is determined by the nature of the experience.

The delusional system is rapidly systemized so that in certain cases the patient might give the impression of a paranoiac. The course of the disease is usually prolonged over a number of weeks, and not infrequently over a number of months. It generally ends in recovery, but sometimes merges into chronicity. The disease is not fatal and its pathology is therefore the pathology of chronic alcoholism.

Nothaas[2] reports a condition which he calls chronic alcoholic hallucinosis and believes that after years of alcoholic indulgence a chronic psychosis may develop the course of which is uninfluenced by cutting off the alcohol. The nature of these psychoses is still a matter of controversy. As already pointed out the alcoholic excesses may easily have been exaggerated as the etiological factor and the make-up of the individual may be of prime importance. Whether the psychosis is of the nature of schizophrenia is also debatable. It certainly presents schizophrenic characteristics and so would seem to indicate that alcohol may release certain schizophrenic tendencies or that alcoholic hallucinosis and dementia precox may be combined in the sense perhaps of what Binswanger[3] would prefer to call alcoholism on the foundation of a schizoid personality.

Alcoholic Pseudoparesis.—In a few patients prolonged alcoholic intoxication produces a picture closely resembling general paresis. In these cases we find an expansive delirium combined with the signs of alcoholism, such as ataxia, speech defects, and tremor. It must be remembered, too, that pupillary anomalies quite regularly occur in severe grades of intoxication and even an Argyll-Robertson pupil has been observed. These observations should be taken with a certain amount of reservation because it is quite impossible often to tell, without the aid of instruments, exactly what the pupillary reaction may be and especially whether it is entirely lost, and it is generally conceded that toxic conditions will produce a slowing of the light reflex. On the other hand, it has been thought by some that

[1] Schneider, Carl von A.: Studies on Alcoholic Hallucinoses, Psychiatric Bulletin, January, 1916.

[2] Ueber chronische Alkoholhalluzinosen, Sitzung v. 1920, **22**, 1. (Deutsche Forschungsanstalt für Psychiatrie in München).

[3] Ueber schizoide Alkoholiker, Zeitschr. f. d. ges. Neurol. u. Psychiatr., **60**, 127

where these marked pupillary disturbances are found it indicates the presence of syphilis. Therefore these observations should be correlated with the serological findings. A case has been reported by Nonne,[1] however, in which the Argyll-Robertson pupil was demonstrated to be due to alcohol: lues was excluded by negative four reactions, optic neuritis was excluded by careful ophthalmological examination, and the light reflex returned upon withdrawal of the alcohol. These cases clear up promptly upon the removal of alcohol.

Alcoholic Pseudoparanoia.—In some patients with chronic alcoholism a fairly circumscribed delusional system may develop which characteristically takes the form of delusions of marital infidelity. In endeavoring to interpret this delusion the paranoia mechanisms must be borne in mind. Quite commonly, however, the delusion when it occurs in a man is a defense reaction to impotence, which has been largely produced, probably, by the alcoholic indulgence. Instead of realizing his own impotence, which is an inacceptable thought, he blames his wife for being untrue to him. These cases are essentially chronic and persist at least as long as the alcohol is indulged in, while even when it is removed they may be a long time clearing up and may perhaps go over into a chronic delusional state largely because of an unconscious homosexual fixation.

Alcoholic Epilepsy.—In a chronic alcoholic toxemia it is not strange that epileptiform convulsions should occasionally develop and recur from time to time. The outward characteristics of the convulsion are in every way the characteristics of an epileptic attack. If the person is not essentially epileptogenic the convulsions will subside on the removal of the alcohol.

Dream States.—In conditions of pathological drunkenness it is not infrequent for the patient to have no recollection of the period during which he was intoxicated. If, during this period, he has been engaged in some sort of occupation, business transaction, travel, or what not, or perhaps engaged in the performance of criminal acts, he may still wake up with no recollection at all of this period, although during it he outwardly appeared to be in a normal state. Some people are especially liable to these dream states. It would seem that they present a tendency to the doubling of their personality and that the alcohol helps to produce these somnambulistic episodes.

Dipsomania.—Dipsomania is a term applied to a periodical impulse to drink. Drinking is only the outward manifestation of the trouble. It is a recurrent neuropathic attack which demands the narcotizing result of alcoholic indulgence and is not infrequently a manifestation of the manic-depressive psychosis or of a compulsion neurosis.

Interpretation.—Read[2] sums up the psychology of alcoholism, so far as it relates to the psychoses, about as follows: Alcohol is taken to promote the social instincts and to alleviate and narcotize the many mental conflicts to which we must all to some extent be victims. He

[1] Neurol. Centralbl., 1915, Nos. 7–8. Also see section on Eye Reflexes for other cases.
[2] The Psychopathology of Alcoholism, Jour. Ment. Sci., July, 1920, p. 233.

thinks it thus becomes a psychological necessity in modern civilization. In excess it tends to destroy sublimation and aid regression and in this way may precipitate a psychosis. The regression may be of various degrees and may bring into conflict with the personality different impulses and desires previously more or less successfully repressed. Of these the homosexual impulse is found on analysis to be the most frequent, the resulting conflict being very liable to result in paranoid states. A deeper study of the so-called alcoholic hallucinoses and paranoid psychoses reveals psychogenic factors which should be looked upon as the real pathological basis of the abnormalities. Dipsomania most assuredly has a psychogenic basis. It must be noted, too, that with the aid of alcohol the psyche defends itself against mental pain, pleasure is gained by the freedom from inhibitions and compensations occur, though often at the expense of sanity. If these views are in any way true, many of these psychoses should be differently classified.

Course.—Chronic alcoholism, whether interrupted or not by any of the forms of mental disturbance described in this chapter, tends to an ever-increasing dementia, *alcoholic dementia.* Mental enfeeblement is a symptom from the outset and is noticeable at first in the esthetic and moral sphere. The previously proud, well-dressed man become slovenly in his habits and unkempt in his appearance. Incapable of the close and continuous mental application of former years it becomes impossible for him to meet the requirements of his business or professional life and lying is resorted to in finding excuses. This is followed by moral obliquities of a more serious nature in which the sexual element is apt to predominate and result in medico-legal complications. Memory is early and noticeably affected. The everyday affairs of life are forgotten, so that the subject of alcoholism neglects to keep appointments, forgets important business engagements, etc. Judgment and the reasoning faculties are similarly enfeebled, until finally the most profound degree of dementia is reached, hastened perhaps by apopletic insults which are not uncommon.

Pathology.—The gross pathology of alcoholism has already been indicated. Cirrhotic liver, chronic nephritis, fatty heart, chronic gastritis, arteriocapillary fibrosis, cerebral arteriosclerosis and cerebral hemorrhage.

The principal lesions found in the brain are pachymeningitis, edema, congestion, thickening and opacity of the piarachnoid, atrophy of the convolutions, sclerosis of the vessels, degeneration of the cells and increase of neuroglia.

The mistake should not be made of attributing everything found at autopsy to the alcohol. Alcoholism is a symptom of an underlying psychopathic state, the stresses of which need to be considered as factors in their own right in interpreting any terminal pathological conditions.

Treatment.—The treatment of *delirium tremens* and the other *acute* alcoholic psychoses should be supporting; liquid concentrated food predigested if necessary. The bowels should be kept free and the kid-

neys kept flushed by a goodly supply of fluid. Heart stimulants are often necessary, digitalis, caffeine, strychnine, to combat cardiac failure, and hypnotics to induce sleep and give rest. The latter should be carefully selected with reference to the patient's condition, depressing agents, such as chloral, giving place to safer ones as trional if there is much heart embarrassment. The after-treatment consists of abstinence from alcohol, tonics, nourishing food and regulation of the emunctories.

For the excitement especially hydrotherapy in the form of the continuous bath is valuable. The thing to be kept constantly in mind in these cases is the matter of nourishment. If the patient does not take sufficient food tube feeding should be begun at once without any delay in temporizing. Food alone will often ameliorate in a remarkable manner the excitement and the insomnia.

The medical treatment of *chronic alcoholism* should be tonic and supporting. Strychnine for a general nervous and cardiac stimulant, ergot if there be symptoms of "wet brain," capsicum and bitter tonics for the gastric condition of anorexia; attention to the emunctories, moderate exercise, baths, massage and electricity for their general tonic effects; sedatives and hypnotics with caution; a modified "rest treatment" if there is marked neurasthenia, and later a sufficient amount of mental and bodily exercise to keep the patient healthfully occupied.

The matter of isolation is an important one. In cases in which the habit is firmly fixed isolation is highly desirable, if not imperative, as in these cases the patient is unable to resist temptation and as soon as opportunity presents itself, will lapse. After confinement for a few months, during which the patient is restored as far as possible to physical health, he is in condition to abstain if he wants to and is able; if he does not wish to or if he suffers from too great weakness of will, he will return to his old practices. If he does wish to stop drinking, however, he has been given the best possible opportunity, an opportunity which should be early extended in all cases and not delayed until by long-continued indulgences the case is of necessity hopeless.

A psychoanalytic approach to the patient's problems is of course indicated and can be carried out to the best advantage during the period of isolation.

Opium.—The habitual use of opium in some form has become common among all classes in society. The same thing may be said with reference to the reasons for taking opium as has been said with reference to alcohol. The opium habitué is a person primarily of neuropathic taint, the mere opium taking or the symptoms it produces being but surface indications of the real trouble.

Symptoms.—The general symptoms of its use are well known. In small doses it is mildly stimulating and produces a pleasant euphoria, while if it is taken in large doses and continuously it leads to a mental blunting, a general feeling of malaise, inability to make effort, with

marked physical disturbances such as tremor, muscular weakness, constipation, paresthesias, etc. It is not infrequent for opium in some of its forms to be combined with addiction to alcohol or cocaine. Continuous use may simply produce a thoroughly crippled individual who is alternately in a mildly euphoric state or in a condition of stupidity, malaise, and indolence. When a psychosis develops, it is characteristically a hallucinated state usually with paranoid coloring, or it may be distinctly delirious. The prognosis is of course not good unless the underlying neuropathic taint can be dealt with.

The use of opium has much less tendency than that of alcohol to produce tissue degeneration.

Treatment.—The treatment is usually the prompt removal of the large doses. The abstinence symptoms which result refer particularly to the heart, the nervous system, and the gastro-intestinal system. These symptoms according to Bishop[1] are uneasiness, depression, apprehension, yawning, sneezing, excessive secretion of tears and mucous from the nose. The extremities get cold, there is clammy sweating, nausea, vomiting, purging, tremors and muscular twitchings. These symptoms are followed by intense abdominal pains, shooting leg pains, cramp, and a feeling of abject misery. The pulse may be slow or rapid. Usually there is a marked drop in blood-pressure. Sometimes profound collapse and actual death may occur with the withdrawal of the drug. Cardiac conditions should be watched and stimulants administered if there is any sign of weakness. For the diarrhea opium should be avoided if possible.

The method of rapid withdrawal is the method of preference if the patient can be relied upon to coöperate or if he is interned. Otherwise the method of gradual reduction. In the rapid method, hyoscine, scopalamine and atropine are used, with pilocarpine as a corrective, to still the craving. In the gradual reduction method codein and dionin are used to replace morphine and strychnine is used as a stimulant. Rapid withdrawal and treatment by atropine for the sympathetic collapse, and pilocarpine or eserine, for autonomic stimulation, may be practised to advantage.

Cocaine.—Cocaine is taken for the same reasons that alcohol, morphin and other drugs are taken, but it is much more dominating in its influence and more difficult to escape from than either of the others, while it is more disintegrating than morphin or opium.

Symptoms.—The symptoms of cocaine intoxication are those of marked stimulation. The patient is extremely active and very talkative, full of all sorts of schemes, ambitious, tells what he is going to do, unfolds plans, and in general is in a manic condition of mind. Following this hyperstimulation there is of course profound malaise and exhaustion. Associated with the habitual use of cocaine there is more apt to be marked and persistent sensory disturbances in the form of paresthesias, while in the mental sphere the disintegration of the indi-

[1] The Narcotic Drug Problem, New York, 1922.

vidual is much more marked than with opium, as a rule. The moral sense is blunted, he lies readily, the judgment is impaired, and not infrequently they develop distinct delusions of which the delusion of marital infidelity is not infrequent. Chronic paranoid conditions, hallucinatory states, and deliria are seen in cases of long-continued habituation. In addition to these systemic effects addicts not infrequently show a perforation of the nasal septum as a result of its local effects when used as a snuff.[1] The drug may be withdrawn much more rapidly than morphine since it does not contribute so largely to produce an infantile regression as does morphine.

Miscellaneous Intoxicants.—A large number of drugs are taken habitually, particularly the whole group of pain-relieving and sleep-producing drugs. Heroin has particularly come to be largely used and is regarded as in some respects the most dangerous of the group especially from the standpoint of habit formation and the creation of new addicts.[2] In general the mental condition due to these drugs varies all the way from delirium as the result of an acute intoxication to chronic paranoid conditions. The main feature of the *drug deliria* is their dream-like character. The patients have all sorts of grotesque experiences which they weave into a more or less consistent description, elaborating the details here and there as may be necessary. The delirious experiences pass like moving pictures before the patient's mind, and they not infrequently are perfectly composed as they recount them, no matter how grotesque or unusual or even terrifying some of them may be. The chief underlying motive for the taking of these drugs is to escape from reality as much as possible and to get into a world of fantasy.

Bromides.—The possibility of bromide delirium from the taking of large doses of bromides for a long period of time should be borne in mind. Patients are not infrequently admitted to hospitals supposedly suffering from some psychosis but with a bromide delirium, the bromide having been administered to quiet the nervousness of a neurosis, or perhaps given in the treatment of an epilepsy. The average practitioner does not appear to realize the possibility of chronic poisoning that prescriptions of this sort present.

The symptoms of bromide delirium as summarized by Karpman[3] are as follows:

Physical.—Marked emaciation and weakness bordering on collapse; cutaneous manifestations, usually an acneform eruption, occasionally severe ulcerations and gangrene; habitual constipation, poor appetite, foul breath, coated tongue, sordes; circulatory disturbances are evidenced by weakness, lowering of tension; genito-urinary disturbances are manifested by renal disturbances, retention or diminution of urine, menstrual irregularities, and lowering of sexual functions. Neurological disturbances are often extreme and may simulate organic

[1] Traffic in Narcotic Drugs, U. S. Treasury Report, June, 1919.
[2] Treasury Report, Loc. cit.
[3] Bromide Delirium. The State Hospital Quarterly, August and November, 1921.

disease. Ataxia is marked, the knee jerks and other tendon reflexes are almost always changed, usually exaggerated, unequal, and irregular; ankle clonus, pseudo-ankle clonus and Babinski have been observed. Tremors of the hands, face, tongue and other parts have been noted and the whole body may be tremulous. The pupils are frequently widely dilated although contracted pupils have been observed; they are often irregular and unequal; most always the light reflex is either sluggish or absent. The speech is thick and indistinct. Sensory disturbances are manifested by anesthesias, parestherias, and hyperesthesias.

Mental.—There are alternations of excitement and stupefaction. The general appearance is of deep confusion. There are active hallucinations, delusions and dream-like experiences are prominent and are mostly of a persecutory and terrifying nature. The mood is variable, usually depressive, although euphoria has been observed. The patients appear completely dissociated from reality, do not understand their environment, and misidentify persons and places; their response to intelligence tests is practically *nil*, attention poor, response to questions often irrelevant and incoherent; nevertheless, if their attention can be gained, the response is often unusually good, although the patient will often hallucinate while conversing, thus showing marked variations in levels of consciousness.

The underlying factors responsible for bromism are the excess of bromides in the body and the deficiency of the chlorides which they displace. The withdrawal by salts of the fluid from the mucous membranes will explain in part the digestive disturbances, the other factors being gastric hypomotility and hyposecretion. The depression of the circulatory apparatus is due to the action of the bromine ion as well as of the basic element. The cutaneous manifestations may be conceived as being due to the profound disturbance of the salt balance in the body which causes defective renal elimination and a corresponding increased elimination through the skin upon which it acts as an irritant. There may be an angioneurotic factor due to the effects upon the vasomotor nerves. The effect on brain and cord is that of depression. The alterations of metabolism and the progressive intoxication produce catabolic and toxic conditions with resulting toxemia.

The total condition above described, bromism, occurs more frequently in those of neurotic and unstable make-up and the delirium is found, not infrequently, to have been preceded by certain psychogenic factors of a precipitating character.

Carbon Monoxide.—Carbon monoxide delirium is of special importance in connection with the frequent attempts at suicide with illuminating gas that one meets with in our large cities. The principal thing to be thought of in connection with carbon monoxide poisoning is that after the initial effects of the poisoning have sudsided and the patient comes out of the coma there may be quite a rapid return to normal and the patient be in an apparently normal condition for a week or ten days and then a relapse occur with marked mental disturbance.

The patient should never be discharged from the hospital until after this period of danger has been passed. Delirium is characteristic of this form of poisoning, and an amnesia for the period usually follows, often associated with fabrications and pseudoreminiscences, producing a Korsakow syndrome. Disturbances of speech are also common, as are disturbances in the emotional field, such as, for example, causeless laughter. Pathologically there appear to be thrombotic occlusions and hemorrhages which characteristically are found in the basal ganglia, and in this region may account for the disturbances of mimic.

Lead.—In chronic lead poisoning we may find a condition of pseudo-general paresis or Korsakow's syndrome in addition to the ordinary hallucinatory and delirious episodes.

Mercury.—This poison produces characteristically a condition of great irritability associated with insomnia and anxiety. This condition may become more pronounced and develop into a well marked delirium. Subacute mercurial poisoning may bring on a typical vagotonic condition with the physical symptoms of general or local vagotonia, the somatic symptoms of visceral vagus alterations or the psychical analogies of an anxiety hysteria, or a manic-depressive psychosis.

There are, of course, other poisons, but these are the principal ones.

General Considerations.—With regard to all of the habit-forming drugs, particularly alcohol, opium, cocain, and the analgesics and hypnotics, it may be said, as already indicated in discussing them separately, that the true cause of addiction lies in the make-up of the individual. This cause would appear to be a narcissistic or homosexual fixation. This fixation and its results are by no means simple matters psychologically and differ very materially in different individuals. The only hope for the treatment of this group of cases, however, is an ability to modify this character anomaly. The treatment, of course, therefore can only be tentative and symptomatic during the periods of acute disturbance, and it is in the interval, when the patient is free from the psychosis, that the treatment should be applied. This is, of course, the psychoanalytic treatment and should be addressed to discovering the underlying etiological factors. Unless something can be accomplished in this direction very little is to be hoped for. The mere palliative treatment by drugs is largely a medical illusion, sometimes, as in specially vaunted systems of cure, a delusion or quackery.

As regards the metallic poisons associated with dangerous trades, of course, the treatment here is purely symptomatic, eliminative, and expectant, while the real effort that is to be of material help should be expended in prophylaxis.

Uremia.—In acute uremia the convulsion is one of the most typical signs, and cannot be distinguished in its outward manifestations from the ordinary epileptic attack. Similarly with epilepsy, too, there may be quite well defined delirioid experiences or dream states in which there is disorientation and noticeable disturbance of the sensorium,

usually associated with more or less constant activity. There may be an anxious affect, or the patient may, as is quite usual in delirium, merely be interested in hallucinatory experiences. Acute uremic symptoms, in the mental sphere, of this character may accompany exacerbations of the physical condition in a patient suffering from chronic nephritis.

In chronic uremia one finds not infrequently marked mental symptoms. The mental symptoms are usually of the character of defect, a general stupidity with a feeling of physical weakness, desire to sleep, with perhaps complaints of headache, associated with irritability. Along with the mental symptoms may go tremor, speech disturbances, eye muscle palsies, pupillary differences, sluggish or failing light reaction, which point to an organic disease of the brain If along with this condition there are Jacksonian attacks followed by mono- or hemiplegia with perhaps aphasia and disturbances of vision, the similarity to general paresis on the one hand and to brain tumor on the other may be very great. Sometimes along with these symptoms the mood is distinctly euphoric, which still further suggests the possibility of paresis. A certain number of the cases of uremic psychoses show close relationships to dementia precox showing negativism, incoherence, catatonia. Probably this group is more serious in its prognosis.

Diabetes Mellitus.—The mental disorder which is associated with diabetes is usually of a mild depressive type, often with ideas of ruin and sin. Anxiety states have also been described. Occasionally the picture gives somewhat of the impression of general paresis. With the chronic depressed condition paranoid ideas sometimes develop.

In general the severity of the mental symptoms may alternate with the severity of the physical disease, as indicated by the sugar eliminated. When the intoxication is very great the patient may be irritable, sleepy, stupid, complain of weakness, and headache.

The diabetic state itself is an etiological factor in the production of arteriosclerosis, and evidences of arteriosclerosis may be found associated with the condition. It should be remembered, however, that both the arteriosclerotic condition and the senile state interfere with the metabolism of the sugars, and, therefore, may themselves be associated with glycosuria.

Gastro-intestinal Diseases.—In general it is well known that with diseases below the diaphragm there is associated on the mental side a depressive mood. The relation between diseases of the gastro-intestinal tract and associated glands to mental states is extremely difficult to interpret. Many of the gastro-intestinal disturbances, rather than being causes of mental disease, are effects. This is particularly seen in the realm of the neuroses and the psychoneuroses, and is discussed in the chapter dealing with these conditions.

Certain cases of acute confusion develop associated with profuse and offensive diarrhea, a high grade of indicanuria, vomiting, low fever, and perhaps mild albuminuria. Some of these cases go on to acute delirium, with typhoid state, profound exhaustion, coma, and death.

Just exactly where these cases belong and what they mean is not altogether known.

Pellagra.—There has been very little study of the mental symptoms of pellagra of late years. From the few cases observed and from study with those who have had it under observation there seems to be a variety of symptom pictures.

Many cases present no nervous or mental symptoms at all. In those who do there seems to be a tendency toward a variable localization of the disease process. There seem to be patients in whom the spinal cord suffers most and others in whom the brain suffers most. In this latter group a condition of very *acute delirium* may be developed



FIG. 452.—Skin lesions of pellagra.

running a rapid course to fatal termination and reminding one of the acute forms of paresis. The more frequent condition, of which we have seen a number of cases, seems to be more in the nature of a *simple retardation*. The patient moves slowly, or not at all, and answers questions after a long delay in a low tone of voice and in monosyllables. There does not go with this retardation, however, a corresponding emotional depression as in melancholia. *Pellagraphobia* has been observed in an infected territory.

With this disease, as with many others, it must not be forgotten that it may be associated with various psychoses without having any specific relation to them. This is peculiarly so in this country, as the large

groups of cases which have occurred have been in hospitals for the insane.

Gregor[1] has made a most careful clinical study of the mental symptoms. He considers his cases under the following seven categories: (1) Pellagrous neurasthenia, (2) stuporous group, (3) mental aberration, (4) acute delirium, (5) katatonia, (6) anxiety psychosis, (7) manic-depressive group. It does not seem clear, however, just what is the connection in all of these cases between the pellagra and the psychosis.

[1] Beiträge zur Kenntnis der pellagrösen Geistesstörungen, Jahrb. f. Psych. u. Neurol., 1907.

CHAPTER XXII.

PSYCHOSES ASSOCIATED WITH ORGANIC DISEASES.

Apoplexy.—Immediately following the apoplectic insult the patient is quite commonly unconscious. As the symptoms subside and the patient begins to "come to" there may be marked disorientation to the extent of a mild delirium, especially if fever be present. This symptom of a slight rise in temperature with onset of mild delirium sometimes occurs a few days after the original insult and is then of bad prognostic omen. In severe attacks the unconsciousness may pass on into profound coma and death.

Ordinarily following an apoplectic insult and after recovery from the acute symptoms a condition of more or less impairment is left, and when it is borne in mind that the patients who suffer from apoplexy are in the main in the senile or arteriosclerotic period it will be understood that the symptoms of the defect take on characteristically symptoms of these two conditions, which perhaps become aggravated materially following the insult. The defect, however, may appear to be very much greater than it really is, because of the inability of the patient to express himself owing to incident aphasia or apraxia. The emotional attitude of the patients is usually one of irritability, although indifference also enters into the picture. Such patients often lie quietly and apparently indifferent until an attempt is made to communicate with them. Under these circumstances if they have serious aphasic or apraxic disturbances and know, for example, what they wish to say, they may become very much excited and quickly fatigued and emotional as a result of their repeated efforts and failures to enter into communication. This is also quite characteristic in the younger patients with apractic disturbances and relatively clear intelligence. It is, so to speak, "maddening" not to be able to control any longer the power of expression. Some of these patients, especially those who suffer from sensory forms of aphasia, being out of communication with their fellows, and not being able to understand what goes on about them, may acquire a suspicious attitude which gives a paranoid coloring to their mental condition. It can be understood that the apoplexy which produces disorders of expression and interferes with the patient's keen contact with his fellows will not only precipitate the deterioration of the senium, but will make that deterioration much more rapid.

The neurological signs of hemiplegia are present, also often the signs of senility, and not infrequently of arteriosclerosis, while a certain few of these patients develop epileptic attacks. (See Chapter XII.)

Traumatism.—The effects of trauma may be divided into the immediate or acute effects and the more remote or chronic results,

into which the former may or may not merge. The ultimate results are divisible into changes in constitution and defect conditions, while a certain proportion of cases develop psychoses following an injury without there being any special connection between the two or in which the injury can only be conceived as a precipitating or contributory factor. Oftentimes the injury is the result rather than the cause, as is so frequently seen in paresis, the injury serving to call attention to the presence of a disease up to that time not recognized. Many shell shock cases have to be interpreted in this broad manner.

The usual immediate result of a head injury, either direct or indirect, is a certain degree of unconsciousness which may vary all the way from a light stupor to profound coma. When the unconsciousness is not profound the patients are quiet, but if disturbed they become very irritable and resistive. Quite typically, associated with the stupor, is a mild delirium of hallucinatory character which is especially pronounced and apt to be more violent in patients who are the subject of alcoholism. Associated with this may be a slight rise of temperature, but if the rise is pronounced meningitis should be thought of. Alternating conditions of apparent clearness and marked confusion may occur. Usually the stupor is of only short duration, but may last several days and finally disappear. In severe cases the patient becomes comatose and dies in that condition. In a few cases the delirious reaction is unusually prolonged and may be associated with Korsakow's syndrome.

After recovery from the immediate effects of the injury certain constitutional changes may slowly develop, the most typical of which is *Friedmann's complex*, which is attributed to a vasomotor disturbance in the brain. The symptoms of this complex are headache, dizziness, irritability, insomnia, physical and mental fatigability, a certain change of character, and intolerance of alcohol, with which may be associated a memory defect of the type of retrograde amnesia, which, however, is not a part of the complex, although there may be a certain memory defect which is quite common to all highly nervous worried people. The headache is apt to be complained of as a feeling of pain or a peculiar feeling of constriction of the head and is associated with feelings of fulness, especially on stooping over. The dizziness is also characteristically intensified by stooping or by sudden movements. The irritability of temper may be so marked as to lead to explosive outbreaks, which simulate psychic epilepsy. Kaplan has named this condition the *explosive diathesis*. They are much aggravated by alcohol. Sleep is liable to be disturbed, physical and mental endurance is very much lessened, and the patient may become depressed, moody, and in various other ways show a subtle change in character which is appreciated by all of his close associates. The intolerance to alcohol is very characteristic of this post-traumatic constitution. Sometimes associated with the explosive diathesis, but also occurring alone, are temporary dream states. Various other hysteriform and epileptiform manifestations may also occur.

Finally, certain defects are left as a result of the traumatism, depend-

ing, of course, upon the nature, the extent, and the location of the injury. As regards the more characteristic results of head injury of a distinctly psychotic nature, it should be borne in mind that the change in character, transformation of the personality, as it might be called, is often a very subtle process and one extending over a very considerable period of time. It might be practically impossible to evaluate the situation at all if one were dependent upon a cross-section of the patient's mental life. When a longitudinal section, however, is available one finds in typical cases an individual who up to a certain point in life has gotten along well, showing efficient reactions and developing by steady progress in some chosen line of work. Such an individual receives a head injury and from the time of this injury on there will be noted in the history a gradual falling off in efficiency. It may be quite impossible to put one's finger upon any specific thing in the situation and set it down as a pathological type of reaction, but the individual reaction has changed in character, and from efficiency there is evolved inefficiency. Naturally, it is a long time before the true explanation of such a change is reached. In fact, it is a long time before it is realized that any change at all has taken place, and it is because of this fact that the older psychiatrists used to speak of traumatic insanity as sometimes eventuating many years after the injury. Not infrequently this falling off in efficiency has going with it a gradual deterioration in morale, and with the heightened susceptibility of the patient to alcohol, which trauma produces, it is natural that alcohol should enter into the picture very largely and often be regarded as the cause of the whole trouble.

Meyer's[1] classification of the effects of traumatism in the nervous system is as follows:

1. The direct focal and the more diffuse destruction of the nerve tissue or of parts of it; and the reaction of the tissues.

(*a*) The immediate effects—edema.

(*b*) The scar formation.

2. The distinctly diffuse commotions in which the general reaction and the psychic elements preponderate, including the remote reactive results of exaggerations of vasomotor and emotional responsiveness.

He classifies the psychoses developing as follows:

1. *The direct post-traumatic deliria with the following subdivisions:*

(*a*) Preëminently febrile reactions.

(*b*) The delirium nervosum of Dupuytren, not differing from deliria after operations, injuries, etc.

(*c*) The delirium of slow solution of coma with or without alcoholic basis.

(*d*) Forms of protracted deliria usually with numerous fabulations, etc. (with or without alcoholic or senile basis).

2. *The post-traumatic constitution.*

(*a*) Types with mere facilitation of reaction to alcohol, grippe, etc.

[1] The Anatomical Facts and Clinical Varieties of Traumatic Insanity, Am. Jour. Insanity, January, 1904.

(*b*) Types with vasomotor neurosis.

(*c*) Types with explosive diathesis.

(*d*) Types with hysteroid or epileptoid episodes with or without convulsions (such as most reflex psychoses).

(*e*) Types of paranoiac development.

3. *The traumatic defect conditions.*

(*a*) Primary defects allied to aphasia.

(*b*) Secondary deterioration in connection with epilepsy.

(*c*) Terminal deterioration due to progressive alterations of the primarily injured parts with or without arteriosclerosis.

4. *Psychoses in which trauma is merely a contributing factor.*

(*a*) General paralysis with or without traumatic stigmata.

(*b*) Manic-depressive and other transitory psychoses, catatonic deterioration and paranoiac conditions, with or without traumatic stigmata.

5. *Traumatic psychoses from injury not directly affecting the head.*

This group of traumatic psychoses includes also a certain number of the *postoperative* psychoses, psychoses following operations upon the eye and residence in a dark room, the so-called *ophthalmic* psychosis, the psychoses of *insolation* and many shell shock cases seen under war conditions. Of course this statement applies in general only to such acute psychoses as do not belong in other groups, as the shock of surgical operations, etc., may well be a precipitating factor in the onset of such psychoses. When, however, they are essentially psychoses the result of shock they have a symptomatology and history quite the same as the traumatic psychoses. They can be understood if the hypothesis of Friedmann is borne in mind that the complex named after him is due to vasomotor disturbance. If this be true it can be understood how severe emotional traumas, as well as physical traumas may produce a vasomotor imbalance, although, of course, it must never be forgotten that whatever may in the last analysis be the actual cause, the cause operates upon a certain kind of individual and the symptom picture is necessarily modified accordingly.

A trauma may act solely as a precipitating cause in a type of personality already set to fall into the pattern of a neurosis. In such cases the trauma is of only incidental significance.[1]

In some shell shock cases concussion and actual destruction of tissue takes place.

The above symptomatology refers to those traumatisms that are accidental. Some traumatisms, however, clearly belong in the symptomatology of the neuroses and psychoses. The injury in such cases is the expression of an unconscious wish, often a symbolic punishment inflicted for sinful acts, the outgrowth of which has been termed a castration complex. Some patients actually castrate themselves, others do so symbolically by putting out their eyes, others

[1] Coriat, I. H.: Recent Conceptions of the So-called "Traumatic Neuroses," Boston Med. and Surg. Jour., July 22, 1926, **195**, No. 4.

bring about apparent accidents, such as a broken leg, with the same unconscious wish as the motive.

Acute Chorea (Sydenham's).—Quite usually a patient with acute chorea is irritable, somewhat emotionally unstable, fretful and impatient, a state of mind one would expect from the nature of the illness. While this condition is quite usual it may become somewhat more aggravated, the restlessness and emotional instability be more pronounced, with evidences of transitory disturbances in the sensorium, and perhaps slight apprehensiveness and suspiciousness. The hallucinatory disturbances may become very much aggravated, together with clouding of consciousness and marked delirium in connection with the febrile movement. Under these circumstances, of course, we are dealing essentially with a fever delirium. Along with these milder manifestations of mental disorder one occasionally finds symptoms of a hysteriform character.

Chorea insaniens is generally considered as a distinct form of acute chorea. It is associated with high temperature and marked mental disturbances, usually beginning early in the disease. The mental disorder is essentially of a delirious character with hallucinations and clouding of consciousness. The hallucinations are quite apt to be terrifying and the patient consequently apprehensive and fearful, although the opposite condition of elation has been described. The disease is quite frequently associated with pregnancy and is not infrequently fatal.

Korsakow's psychosis is sometimes seen in patients suffering from acute chorea as a result of polyneuritis resulting from overtreatment with arsenic. (See section on Chorea.)

Chronic Chorea (Huntington's).—It has always been recognized that this disease was associated with mental symptoms, and it has generally been considered that there was a tendency to progressive deterioration ending in well marked dementia. While in a general way this may be true, it is well to be cautious in estimating the mental condition of chronic choreics. They are extremely inaccessible in many instances, especially those patients whose speech apparatus is affected by the disorder, and it requires great patience to make a satisfactory examination of their mentality. On the other hand the patients themselves find extensive explanations so difficult to communicate that they are content with the shortest possible responses, so that they frequently mislead the examiner into the belief in an intellectual poverty which does not really exist.

With these warnings it may be conceded that in general the chronic choreic is of somewhat enfeebled mentality with a tendency to emotional depression and to a mood of suspiciousness. The emotional depression can be readily understood, as the patient realizes quite well his plight, namely, that he is affected with a chronic disease which practically isolates him from his fellows. Not infrequently there is a marked emotional instability and irritability and such choreic patients may have great difficulty in getting along in wards

where they come in contact with other patients. Finally, with the increasing deterioration there is a tendency to indifference, to emotional deterioration. The suspicious mood, which so many of these patients have, we believe has at least in part its explanation in the patient's isolation as a result of the nature of the disease. We find suspicious moods in all persons who are cut off from active contact with their fellows, more especially in people who are closely confined, either voluntarily or otherwise, those who suffer from pronounced varieties of aphasia, which make it impossible for them to communicate their thoughts or to receive communications from others and predominantly among the deaf. To a certain extent, too, the dementia is probably due to this cutting off of active contact with life by impairing the means of communication. The choreic finds it extremely difficult to talk to others. He may find it equally difficult to read, perhaps because of defect of vision which is liable to be present, as it must be remembered that this is a disease of later life, and in various ways he loses touch with what is going on about him. Finally, when he has long passed any ability to pick up the thread of events he becomes indifferent, and when it is realized that he is frequently in the arteriosclerotic period, often approaching the senium, it can be seen that this removal from acute contact with reality tends to produce inactivity, as it were, and the appearances of dementia, if not actual dementia, and so the chronic choreic shows emotional defects, attention disorder, lack of impressibility, defect of recall and in other ways demonstrates that he has lost interest, as it were, in life.

A certain few cases show distinctly more pronounced psychotic symptoms, occasionally showing well marked persecutory ideas. (See Chapter X.)

Paralysis Agitans.—It is probable that the majority of cases of paralysis agitans show a certain amount of mental disturbance, although in a great number of cases this disturbance is so slight, compared with the physical, and so easily seen to be dependent upon it that it is quite overlooked. This simple disturbance is in the emotional sphere and for the most part is one of slight depression but rarely one of euphoria. Occasionally delirioid episodes supervene, with marked confusion, but in general consciousness is clear and the patient remains well oriented, except, of course, in the terminal condition when arteriosclerotic changes and the deterioration of the senium have set in. Occasionally there is a well marked psychosis, in which instance it is most apt to be of a depressive hypochondriacal character, often with paranoid coloring. The depression may be sufficiently great to result in efforts at suicide. In these cases disorders of the sensorium may also be present.

As intimated above it must be remembered that we are dealing with a disease of later life and that not infrequently arteriosclerotic and senile changes show themselves by changes in the mental sphere.

Multiple Sclerosis.—The outward manifestations of the mental disorder associated with this disease are mainly in the emotional

sphere. It has been stated both that the majority of patients were depressed and that the majority were exalted. Both these statements are based upon the observation of very few patients. Although exaltation and depression may be manifest and there may also be spasmodic laughing and crying, due probably to lesions of the basal ganglia, which are unaccompanied by emotion, there is a certain amount of deterioration which is progressive and which depends, of course, upon the destructive changes. When the distribution of the sclerotic patches is quite similar to the distribution of the lesions in general paresis the outward symptoms of the two conditions may be very much alike and lead to confusion in diagnosis. The diagnostic problems, however, can be cleared up by the laboratory findings.

Encephalitis Lethargica.[1]—As might be expected, from an analogy with paresis, an infectious-organic disease of the nature of lethargic encephalitis gives a wide variety of symptom groupings at the psychological level. These have been variously worked over, described and classified by the different observers who, as yet, have reached no uniformity in their description of types which is as yet comparable to that attained in paresis. There are indications thus far that the qualitative specificity of the psychic symptoms is greater than for the general run of toxic and infectious states and perhaps as great as for the analogous infectious-organic disease, paresis. While delirium is an outstanding feature in many cases, as in the various toxemias and infections, lethargy seems to be a more definitely specific symptom but one which is in need of much detailed clinical analysis to the end of appreciating its different degrees and its intrinsic nature.

During the acute stages the symptoms are those of a toxic-infectious psychosis with delirium, various grades of sleep disturbances running from simple drowsiness to lethargy, stupor and coma, the Korsakow syndrome and various other reactions. Hyposomnia may also occur. Mood changes may precede and there may be euphoria or depression. In the lethargic and stuporous states there is apathy and lack of affect. In the unrecovered cases there is evidence of alteration of character and mood shown by depression, exaltation, irritability, explosive reactions, stubbornness, apathy, etc., which Kirby and Davis think indicate lasting damage in the emotional sphere. This is most characteristic and they have found little evidence of organic defects nor even of a characteristic catatonia such as found in dementia precox.

Tilney and Riley[2] on the other hand describe a cataleptic type as well as a lethargic, an epileptico-maniacal, and an acute psychotic type. Hohman[3] classifies his cases into four reaction types (*a*) depressive, (*b*) psychoneurotic-like, (*c*) delirious, and (*d*) organic and gives nine symptoms of frequent occurrence (*a*) push of talk without distortion, (*b*) surprising alertness on arousal from stupor, (*c*) feeling of

[1] Kirby, G. H., and Davis, T. K., Psychiatric Aspects of Epidemic Encephalitis, Arch. Neurol. and Psychiatr., 1921, vol. **5**.

[2] Epidemic Encephalitis, Neurol. Bulletin, 1919, vol. **2**.

[3] Epidemic Encephalitis, Arch. Neurol. and Psychiat., September, 1921.

well-being and happiness, (*d*) behavior oddities out of keeping with general activity or mood, (*e*) emotional instability and irritability, (*f*) organic type of delirium at times accompanied by fear, (*g*) stupor states, (*h*) depression, frequently the dominant mood, and (*i*) characteristic memory defect.

Wolfgang[1] has recently called attention to a sequel that should not be lost sight of, namely, ethical defects manifested by lying, stealing, and cruelty which may be of the psychic compulsory sort or associated with akinesis or hyperkinesis. He thinks this condition more usually follows the non-lethargic form of encephalitis and recommends iodine therapy, especially if the ethical changes are less in the form of compulsions than of weakness resulting in irritability as a psychic correlate of hyperkinesis.

Treatment.—The treatment is the treatment of the underlying infection which is moving in the direction of serum and vaccine therapy.

Polyneuritis.—See Korsakow's Psychosis.

Heart Disease.—Depressive states are most in evidence here. Mental symptoms, however, are most apt to be associated with failure of compensation. With precordial distress goes typically a mental state of anxiousness. Transitory confusions with dreamy hallucinations occur with compensation disturbances and edema.

Various other diseases have from time to time mental symptoms associated with them. The great majority of such diseases, if not all of them, have elements of infection, or toxemia and exhaustion combined, with all or part of them. The mental symptom-complex of confusion arises most typically. In some cases, especially the less acute, paranoid conditions occur and hallucinosis is of occasional occurrence.

Head has shown that certain visceral diseases, especially of cardiovascular and pulmonary origin, often have associated mental symptoms, although they may not appear except on the most careful examination. The symptoms found are: (1) Hallucinations of vision, hearing and smell; (2) moods, either of depression or exaltation and (3) suspicions usually occurring when a depression has persisted for some time.

These conditions take their origin in part as a result of reflected visceral pains. Each spinal segment has both a visceral and a cutaneous representation. Disease occurring in the visceral area is referred to the cutaneous surface supplied by the same segment. The cutaneous distribution of the fifth nerve corresponds to the visceral distribution of the vagus, so pain occurring in the vagus territory will be referred to the scalp and thus occur points of tenderness in this region with which the hallucinations are associated. The mood of exaltation is essentially transitory and arises as a contrast phenomenon

[1] Psychische Veränderungen bei Kindern im Gefolge von epidemischer Encephalitis, Archiv f. Psychiatrie, 1923, vol. **69**.

of the depression and as a result of the disappearance or lessening of the reflected somatic pain.

Still more light may be thrown upon the psychoses associated with these various chronic diseases when the unconscious factors are better known. A disease like paralysis agitans or chronic chorea for example seems to be due to a wearing out of certain mechanisms, a, so to speak, localized senility which may be conceived as due to having too constant and too severe demands made upon these mechanisms over a long period of time. It may be that here we see a chronic tension of a given mechanism, a chronic state of preparedness, as it were, for a form of expression which the repressions do not permit to come to pass and so the machinery is worn out in futile efforts. A deeper study of the unconscious factors is needed in all these conditions.

CHAPTER XXIII.

PRESENILE, SENILE AND ARTERIOSCLEROTIC PSYCHOSES.[1]

The grouping of the presenile, senile and arteriosclerotic psychoses together in one chapter is a matter altogether of convenience, for obviously one would expect all sorts of admixtures, particularly as between the senile and arteriosclerotic groups. There are many pictures here which are not distinctive, especially in the presenile period, which Kraepelin begins his discussion of by saying it is the darkest region of all psychiatry today. This whole region is exceedingly obscure and the factors which enter into and are causative of the psychoses of these periods are little understood. The difficulties of coming to any clear understanding of the various psychotic pictures here included are due, in the main, to (*a*) the lack of any clear comprehension of the nature and meaning of the conditions included in such concepts as involution and senility; (*b*) the multiplicity of the factors involved; (*c*) and the impossibility of adequately correlating the anatomical findings and the clinical picture.

To the classical trio of factors usually considered as of etiological significance; namely, the climacterium (in men as well as in women), arteriosclerosis, and the senium must be added various exogenous and endogenous (including psychogenic) factors, biogenic factors (including organ inferiority), the extinction of certain gonadal functions and the resulting endocrine and other physiological readjustments, and the operation of various psycho-physical coefficients and their dynamic interplay at a time of failing capacities for compensatory readjustment.

The last series of factors would seem to offer the largest possibilities for a logical dismemberment of this complex group, especially if coupled with a better understanding, from a biological point of view, of the nature and significance of the involution period (climacterium and senium). It would seem that, only by a life history study of the personality of the individual which would disclose the nature and direction of his strivings and the consequent stresses to which he had subjected the machinery of his body (soma), coupled with a fair comprehension of his organic integrity or otherwise (organ inferiority) could an understanding be reached of the nature and meaning of his failure, as exhibited in the symptoms. If this theoretical approach proved correct then the symptoms would prove to be the symbolic representation of his life problems writ large because of his failure longer to adequately handle them.

[1] Kehrer, F.: Die Psychosen des Um- und Rückbildungsalters, Zentralbl. f. d. gesamte Neurol. u. Psychiat., 1921, Bd. **25**.

This point of view is already helpful in understanding certain of the affective and delusional symptoms of the involution and arteriosclerotic groups and also of some of their physiological and later anatomical correlates. These functional expressions can be seen, if adequately probed, to belong to the patients' life history way of reacting and often to merge by insensible degrees into the more stable picture of actual anatomical changes. Thus vascular hypertension with its hyperthyroidism and metabolism accompaniments may be correlated with an anxiety based upon a feeling of inferiority and may be seen to merge into more stable physico-chemical fixations such as arteriosclerosis, arthritis, nephritis and the host of associated and dependent processes.

The psychic would seem, therefore, to be the logical point of attack for getting hold of those guiding lines that would ultimately lead to a logical grouping of signs and symptoms into reaction types based, not upon supposedly distinctive pictures at some particular level, such as miliary plaques at the anatomical level or the peculiar Korsakow-like symptoms at the symbolic level, but to an understanding of the process as a whole. While the existing facts may warrant certain speculations of the relation, for instance, between vascular hypertension and subsequent arteriosclerosis, it goes without saying that there is no such warrant for connecting up the finer and more remote anatomical changes upon any such basis. The impossibility, at present, at least, of doing so is the reason why the anatomical findings cannot as yet be brought into unambiguous correspondence with the symptoms at the psychological level and why also, of course, various other symptoms (metabolic, the peculiar character of certain delusions) have not been adequately assimilated to a comprehensive concept.

The Presenile Psychoses.—In the presenile period there are a number of psychotic pictures the exact significance and nosological placement of which is not at all understood. There is no doubt, however, that in this period depressions are much more in evidence than excitements, the depressions seeming to mount up in frequency during the period of involution. Hübner found, for example, 21 cases of single attacks of melancholia after the fiftieth year, but only 2 cases of single attacks of excitement.

Involution Melancholia.—*Symptoms.*—The Kraepelin school for a considerable time considered that certain depressions of later life, characterized by an anxious apprehensive agitation, with profound emotional depression, composed a nosological unit to which was given the name involution melancholia, or more briefly melancholia, the term melancholia being limited in its application to this particular type of depression.

The symptoms of this psychosis are generally preceded by a considerable period during which there are vague head symptoms, such as pressure, pain, vertigo, together with anorexia, irritability, insomnia, mental insufficiency, and a mild neurastheniform state with perhaps some emaciation. This condition becomes progressively worse and the patients develop an agitated depression, with anxiety, apprehen-

sion, fear of impending danger with quite usually delusions of sin. The depression is characteristically very profound and the motor agitation may be quite considerable. The patient may go about wringing his hands, moaning and groaning, perhaps repeating over and over again such phrases as "Oh, my God! Oh, my God!" "It is a fearful thing. Good Lord help me!" One patient constantly repeats "Doctor, will I be done away with tonight?" and "Then will I be here tonight just the same as last night, and will I be here tomorrow just the same as today?" Another patient believes herself very sinful, refuses to eat because the food should be used for others, walks about in an agitated manner picking her fingers and attempted suicide because she was afraid she was to be put to death.

Even in these cases of quite extreme agitation and profound depression consciousness remains unclouded, orientation is little if at all impaired, and the form of thought is well maintained.

Another type of case presents symptoms of retardation. Many of these cases may be so retarded as to be almost stuporous and some

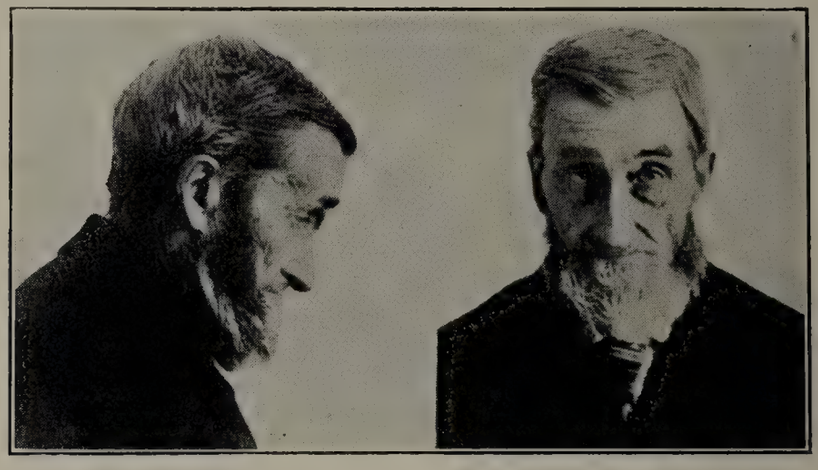


FIG. 453.—Facies of involution melancholia.

of them present an amount of resistance which reminds one of the characteristic negativism of dementia precox. This latter group was originally described by Kraepelin as "late catatonia." Its nosological status is at present in doubt.

Certain cases of anxious depression may be highly agitated with insomnia, refusal of food, rapid emaciation, clouding of consciousness, hallucinations, self-inflicted injuries, attempts at suicide, a rapid course ending in death.

In the marked cases of apprehensive and agitated depression there is frequently a considerable degree of precordial distress with perhaps tachycardia and often a sense of oppression over the chest with a feeling of difficulty of breathing. These symptoms are apt to appear in attacks, at which times the depression is more pronounced.

The danger from suicide is very great in this psychosis, particularly because the patient is not so frequently rendered inactive by marked retardation.

The delusional content of consciousness varies widely, but hypo-

chondriacal delusions, nihilistic delusions and the feeling of unreality are common. Occasionally the delusions take on bizarre, absurd and fantastic forms, and there may be delusions of grandeur late in the course of the disease. There may be a strange mixture of depressive and grandiose ideas, as in the patient of Weygandt's, who believed she was going to be boiled in a silver kettle.

The statistics of this disease show that about 40 per cent. got well, the remaining 60 per cent. terminating in various ways; some by suicide, some by death from intercurrent disease, which they were ill adapted to withstand; some by death from general marasmus or the development of tuberculosis; some lapsed into chronicity; and finally, a few improved sufficiently to leave the hospital and get along at home, though still somewhat depressed. A certain number of this class may get worse under home surroundings and have to be returned to the hospital.

This group of involution melancholia, so-called, was studied with great care by Dreyfus.[1] He studied the life histories of 81 cases, 34 of which were personally investigated. Eight were not personally investigated and 39 were deceased. As a result of his study he concluded that with the exception of 2 cases undiagnosed, 2 cases in which a mistake in diagnosis had been made, and possibly 2 more doubtful cases, all were cases of manic-depressive psychosis. This conclusion was reached by finding the fundamental symptoms of this disease present. Of these cases 66 per cent. were recovered or were recovering at the time of death, 8 per cent. developed arteriosclerosis, 25 per cent. died unrecovered of intercurrent disease or suicide.

The conclusion reached by Dreyfus that the involution melancholia of Kraepelin is really a form of manic-depressive psychosis was accepted by Kraepelin himself. A study of this group of cases shows quite characteristically the presence in the history of periods of affect fluctuation; and quite usually periods of slight depression which were not severe enough to attract attention seriously to the mental condition, or perhaps were not at all understood at the time, being accounted for in various other ways.

Even admitting that this group of involution melancholias really belongs to the manic-depressive psychosis, still the problem is not wholly solved and it must be admitted that in all probability the involution period has certain modifying effects upon the psychosis.

In the first place the depressions as noted heretofore are very much more frequent than the excitements and they are very much longer in duration than during earlier life. This of course can be easily understood by the failing resistance of the involution period. A psychosis that has manifested itself only by mild and hardly appreciable evidences throughout the youth of the individual may well get a firm hold when the powers of efficient reaction are failing.

With the recent work that has been done on the ductless glands

[1] Die Melancholie, ein Zustandsbild des manisch-depressiven Irreseins, Jena, Gustav Fischer, 1907.

and with the somewhat characteristic mental pictures that go along with disturbances of the internal secretions one must bear in mind that perhaps many of the changes of the involution period are, in part at least, determined by changes in these glands, particularly as the result of atrophy of the uterus, the ovaries, the prostate, the testicles and the adrenals, with the possible result of an imbalance being brought about in the relationship between them. This, of course, if it is so, would only be one of the modifying factors of the involution period which one might expect to see reflected in the diseases at this time of life.

The following case illustrates this type of psychosis: The patient, a woman, was admitted to the hospital at the age of fifty. She was very much depressed, said that she was very unworthy and should be hung, that she had committed the unpardonable sin and would burn forever in the flames of Hell. A little later, in addition to these ideas that she had committed the unpardonable sin and that her soul was lost, she said that she was dead, what existed of her now was her spirit, that her body had passed away, and because she was dead it was useless to attempt suicide. This condition had its origin some two years before, following an operation, when she had cleared up, and as a result, so stated, from overwork, had broken down again and developed these ideas and subsequently was admitted to the hospital after having made a suicidal attempt. In addition to the delusions she had visual hallucinations, saw spirits and heard them talking to her.

This patient gave the general impression on her admission to the hospital of a patient suffering from involution melancholia. A further inquiry, however, developed the history of numerous previous attacks of depression, which suggested a manic-depressive reaction type. Just before her discharge from the hospital, however, when she had become much more accessible, we learned that in her early life, before she was twenty, she had had a love affair. This love affair had been a very profound emotional experience, particularly because she had found it necessary to give up the young man because of his drinking habits. That this was not willingly done, however, was shown by her subsequent life, which was taken up by constant activities, largely of a social nature and apparently for the purpose of side-tracking her disappointment. She was an attractive young woman and had many admirers, but did not permit herself to become attached to any one of them. She led a very active life and was able to sublimate the energy of her suppressed emotions until the period of the menopause, when she broke down with self-accusatory delusions and suicidal tendencies. She believed she had committed the unpardonable sin; this sin was having committed adultery in her mind with the young man with whom she had been in love in her youth. The psychosis lasted somewhat over five years, at the end of which she finally succeeded in reaching a compromise by developing tendencies diametrically opposed to her delusional system. She became talkative, humorous and particularly facetious, and just before her discharge

she made the remark that she was born dead, but made it with a smile, and with the added comment that she had gotten along pretty well all her life for a dead person. This condition of relative equilibrium enabled her to go back to her family and in the absence of disturbing factors she will probably get along all right, at least for a considerable time. Nothing has been heard of her for some years. The compensation, the balance struck, was a fairly efficient one.

Such a case as this illustrates a common type of depression which occurs in the involution period. It is a type which if dealt with solely from the descriptive standpoint would have been considered an involution melancholia, while a little more careful history indicates that it is a manic-depressive reaction type, while a still more profound probing into the facts of the life make the whole thing quite understandable in psychological terms. It is manifestly a psychogenic depression, but whether it should be called manic-depressive or not is a question. Now that psychoanalytic methods are showing the presence of endopsychic conflicts in the manic-depressive psychosis, such a case as this shows the close connection between a pure psychogenic psychosis and the manic-depressive reaction type, and the further relation of these conditions to the depressions occurring during the involution period.

Diagnosis and Prognosis.—In a recent study of this group Hoch and MacCurdy[1] have endeavored to differentiate the cases which can be properly grouped with the manic-depressive depressions from those that are of more malignant course and to which the term involution melancholia is more applicable. The former group show feelings of sadness, hopelessness, wickedness, incapacity and unreality on the subjective side, with objective evidence of physical and mental sluggishness and an appearance of dejection in attitude and facial expression. Delusions are concerned with the moral obliquity of the patient. Hallucinations are extremely rare. In the melancholias, on the other hand, on a general background of such symptoms appear fearful delusions and marked anxiety, often with terrifying hallucinations; a stray tendency to hypochondria, sometimes leading to fantastic delusions; gross anomalies of conduct with much irritability, perversity and sometimes filthiness; and finally some patients show a loss of adequate emotional response to their environment or to their own ideas. Between these two types there are innumerable transitions but the extremes are in marked contrast. These two types may be described as the benign and the malignant, the former related to manic-depressive psychosis, the latter to schizophrenia.

As a result of the study of 67 patients in whom the final outcome was determined in all but 1 case, Hoch and MacCurdy concluded that patients with involution melancholia recover unless they show as dominant symptoms, marked insufficiency of affect, peevish or autoerotic behavior, or ridiculous hypochondriac delusions which usually

[1] The Prognosis of Involution Melancholia, Am. Jour. of Psychiatry, January, 1922.

are concerned with the alimentary tract. These prognostically bad symptoms may be present for a brief period in the psychosis of women at the menopause without omnious significance. All patients who recover show some improvement within four years from the onset, the others run a chronic course or die unimproved.

The Nature of Melancholia.—In a recent study of melancholia Freud[1] has contributed many illuminating observations that help to an understanding of this condition. In the first place he differentiates melancholia from normal grief in which the desire to live finally triumphs over the wish to die and to share the fate of the lost love object and in so triumphing the love is gradually released from its former attachment and becomes free to make new ones. Melancholia differs from this normal process both in its outcome and in certain of its manifestations. Painful depression, abrogation of interest in the outer world, loss of capacity to love, and inhibition of effort are common to both, but, in grief it is the world that is felt to be poor and empty while in melancholia it is the self that feels poor, worthless, and pervaded with the conviction of (especially moral) inferiority. The loss in grief is a conscious one, in melancholia it is unconscious. Observation shows that the abuse which the melancholic heaps upon himself really represents complaints directed against a person he formerly loved. This explains why he is not ashamed of his expressed deficiencies and does not behave as if they were true, but, on the contrary, acts as though he had been unjustly treated. The mechanism is as follows: As a result of some disappointment or injury to his love he has withdrawn it from its object, but instead of transferring it to a new object as is the normal mechanism, or introverting it on to unconscious phantasies, as does the neurotic, or applying it to the ego as the paraphrenic does, he replaces it by a narcissistic identification of the self with the former object. The libido is withdrawn from the object and the object is built up within the ego itself, that is, it is, so to speak, projected upon the ego. The explanation of this mechanism is that it probably represents regression to the original narcissistic way in which he fell in love. The ego thus becomes split and one part to which consciousness adheres can thus criticise, hate and abuse the other part formed by a fusion with the idea of the object. In other words, one part of the ego can treat the other part as object. Three conditions seem to be necessary for this outcome; first, an actual loss as in grief, though the loss is more often due to disappointment than to death; secondly, the combination of a strong narcissistic fixation on the loved object with a lack of resistance in its investment; and thirdly, a marked ambivalency, as in the obsessional neurosis. Thus suicide becomes understandable as an attack upon that aspect of the ego that is identified with the object both loved and hated (ambivalence).

In melancholia interest in the outer world, object cathaxis, is given up

[1] A General Introduction to Psychoanalysis. Jones, E.: Recent Advances in Psychoanalysis, The British Jour. of Psychology, Medical Section, October, 1920.

and as a result their is megalomania, which the psychoanalysts believe can always be found by the analytic technique, and hypochondria. The megalomania may be manifested by such beliefs as that the patient is the greatest sinner in all the world or the worst criminal that ever existed. The self-love and egotism are symptomatic also of a degree of narcissism that make these patients almost inaccessible to therapeutic approach. The refusal of food is indicative of the importance of the oral-sadistic factor. This sadistic tendency is turned against the self (that part which represents the introjected object with which the patient is no longer in harmonious adjustment) and by the severity of self punishment an effort is made to be rid of the sense of guilt. The psychoanalyists believe the sense of guilt is oral-sadistic in origin consisting of cannibalistic impulses.

Pathology.—An increased neuroglia formation in the deeper layers of the cortex has been described, and in this disease we find most often a termination in the condition described by Meyer as *central neuritis.* This is essentially a parenchymatous degeneration with swelling of the cell body, a disappearance of the chromophile substance and eccentricity of the nucleus. In rapidly fatal cases there are evidences of extensive destructive changes in the cortex. There are also found a certain amount of disintegration products.

Treatment.—One of the very important considerations in this group of depressions, as shown by the symptomatology, is the prevention of suicide. It is therefore very much more frequently necessary to intern these patients so that they can be properly watched. Insomnia, agitation, failure of nutrition with suicidal tendencies are the conditions that have to be met. For the agitation and the insomnia hydrotherapy is the best means of treatment. As few drugs as possible should be used. For the suicidal tendencies constant watchfulness, which should have as its ideal an unobstrusiveness which does not suggest the suicidal idea constantly to the patient's mind. Failing nutrition must be met by tube-feeding if necessary.

An analysis of the patient's mental symptoms with the view of understanding them is of course highly desirable, but this class of patients do not lend themselves easily to analytic technic, at least until they have begun to quiet down, oftentimes not until they are convalescent.

Other Psychoses of this Period.—Various other types of psychoses have been described as belonging in this period. Types of depressive Wahnsinn, some of them associated with anxiety and proceeding to deterioration, while Kraepelin has described a very small class of so-called presenile delusional psychoses, affecting especially women, in which delusions of infidelity are usually prominent, together with hallucinations. The delusions are variable and do not seem to at all logically control the conduct. Farrar has described three types of psychoses belonging to this period: *melancholia vera, anxietas præsenilis* and *depressio apathetica.*

In *melancholia vera* there is in the main an autopsychosis. The delusions are auto-accusatory, with ideas of sin but with clear consciousness; there is no defect of orientation. The patient believes his soul is lost, that he is to suffer eternal torment hereafter, and about these beliefs there is no doubt, but on the contrary a marked "*subjective certainty.*" There may be some slight tendency to somatopsychic delusions, insight is defective and slight anxiety may be present.

The following attempts at formulation indicate the outcome of further efforts directed toward the dismemberment of the confused mass of symptom groups found at this period of life.

Involution Paranoia of Kleist.[1]	Involution Paraphrenia of Serko.[2]
Beginning.—In close connection in time with the climacterium.	Acute.
Climax.—Years after this.	Chronic progressive.
Occurrence.—Ninety per cent in women; seventy per cent, living alone.	Principally women.
Foundation.—Grows out of the personality, out of a hypoparanoid constitution, and finds expression in exaggerated consciousness of self, desire to dominate obstinacy, sensitiveness and suspicion.	
Symptoms.—1. Elementary: Suspicious or expansive depressive mixed affects, misinterpretations, illusions, falsifications of memory.	*Primary.*—Insane ideas of insult and injury, persecution; secondary: the demands that go with the overvaluation of self.
2. Peculiar disturbances of thought: Retardation and perseveration; flight of ideas (difficulties in performing certain mental tests, restriction of associations with flight of ideas in the free play of thought; loquacity in regard to past experiences.	*Content.*—The sexual factor is never absent (jealousy, trickery with sexual point, sexual threats, insults, rape, indecent assaults, insane ideas with regard to marriage.)
Resultants.—Insanity of injury with or without ideas of grandeur in all forms (connected with reality or without any connection whatever with reality, unchanging or varying, systematized or non-systematized.	*Form.*—Trivial trend, common place scope, lack of elaboration (complexes without logical elaboration as the expression of the general narrowness of spirit and whimsicality).
Very Great Role.—Sexual affects (jealousy, fear of sexual insults, indecent conduct).	Visual phenomena rare.
Illusions; especially optical are never absent.	Pseudomemories are often decisive; also phonemes of sexual coloring.
Hallucinations.—In 50 per cent there is belief in thought-hearing, and the insane ideas take the form of sense presentations.	True hallucinations questionable. On the other hand loquaciousness and cyclothymic variations.
Course.—Insidious onset; when the climax is reached the curve proceeds at an even level, terminating in chronic invalidism without destruction of consciousness, or dementia.	In the course of years a chronic progressive mental regression in the sense of a reduction of affective interest (absentmindedness in expression, relative inaccessibility, but without schizophrenic features).

[1] Kleist: Ueber chronisch wahnbildende Psychosen des Rückbildungsalters usw. Allg. Zeitschr. f. Psych, 1912, **69**. Die Involutionsparanoia. Ebenda, 1913, vol. **70**.

[2] Serko: Die Involutionsparaphrenie Monatschr. f. Psych. u. Neurol., 1919, vol. **45**.

INVOLUTION PARANOIA OF KLEIST.

Nature.—Autochthonous intensification of the hypoparanoid constitution as result of non-destructive changes in the brain of inner secretory nature, following involution of the generative organs. The somatic endogenous reactive factor outweighs considerably the psychogenic reactive factor. The physiologic or normal change of the personality at advanced age in the direction of obstinacy, etc., is more marked because the individuals have been hypoparanoid during their entire life.

In the main lines all the cases cited conform to the symptomatology both in course and origin, as given.

INVOLUTION PARAPHRENIA OF SERKO.

Reflextion of the normal psychic peculiarities of the individual in intensified form, in the involutional years.

PRESENILE PARAPHRENIA OF ALBRECHT.[1]

Beginning.—Somewhat later than the climacterium, forty-five to fifty-five years.

Occurrence.—Two-thirds in women.

Foundation.—Hereditary taint. Hypoparanoid character not proved.

Symptoms.—Suspicion. Misinterpretations. Ideas of reference and persecution, often in the sense of tricks, mocking, poisoning and with sexual content (paramour, incest, impregnation, etc.).

Secondary delusions of grandeur.

After months hallucination of senses in the direction of the insanity, which, becoming more numerous, are finally the principal symptom.

No flight of ideas. Good humored excitability not specially injured.

Course.—Chronic condition of insanity with hallucinations. Even after years no considerable mental weakness.

Nature.—"Identical with involution paranoia, perhaps only distinguished from Kraepelin's paraphrenia in involutional years for practical reasons, being a subform thereof."

PARANOID PSYCHOSES IN ADVANCED YEARS OF SEELERT.[2]

Years after the climacterium, between fifty and seventy-four.

Five-sixths women; nearly all women who have lived alone in want and in bad social conditions.

From youth on, abnormally lively affectivity (extreme feeling of self-importance, tendency to dominate, suspicion, incompatibility, excitability) with good intellectual endowment.

Insane ideas of being insulted and injured through exhalations, smoke, powder persecutions; numerous bodily disturbances, sensations of taste and smell; hallucinations of hearing (telephone, crackling noises) never hallucinations of sight. Insanity of interpretation. References to self, falsifications of memory, retrospective misinterpretations. Isolated delusions of grandeur.

Material.—Like the subjective perceptions of organic brain disease.

Form.—Slight systematization. Endogenously conditioned individual reactions to an organic cerebral arteriosclerotic process, of slow course, and proved to have actually existed in two-thirds of the cases (brain involution). "Identical with presenile insanity of injury, senile insanity of persecution, involution paranoia." Sharply distinguished from paraphrenia, but with transitions to, and connections with, melancholia and the characteristic features of manic-depressive insanity.

Insidious development with only occasional variations in emotions. Uniformity of picture with transitions to intellectual amnesic defective conditions of light form.

[1] Albrecht: Die funktionellen Psychosen des Rückbildungsalters, Zeitschr. f. d. gesamte Neurol. u. Psych., 1914, **22**.

[2] Seelert: Paranoide Psychosen im höheren Lehensalter, Arch. f. Psych, 1915, **55**. Verbindung endogener und exogener Faktoren in dem Symptomenbild und der Pathogenese der Psychosen. Beiheft 2, Monatsschr. f. Psych. u. Neurol., 1919, **6**.

In *anxietas præsenilis* there is, on the contrary, in the main an allopsychosis. There is a very marked "*subjective uncertainty*" which gives an unreal tinge to the outer world and out of which grows the fear of things unknown, culminating in the marked anxiety which is characteristic of this form of the psychosis. Remorse or dread of the future are not elements in the depression; on the contrary, it is the great unknown and overwhelming present that seems about to destroy them. These cases occur later in life than the former, show more evidences of senile decay, such as arteriosclerosis and present such symptoms as verbigeration, rhythmical movements, suggestibility; the prognosis is less favorable.

In *depressio apathetica* there is simply a let-down, a stopping on the part of one who has been leading an active life. Interest abates, the struggle is drawn away from and we have a picture of mild depression with clear consciousness and no disturbance of orientation. There is some "subjective uncertainty" delusions and sensory fabrications play little part. The symptoms are negative rather than positive; the prognosis is relatively good.

Finally one must remember that a true anxiety neurosis may develop, and it not infrequently does, at this period of life.

In conclusion then it will be seen that we have during this period of life a group of psychoses of widely different forms, although tending toward a certain amount of uniformity in their outward expression in that depressions are so much more frequent at this period. In the first place there are frank attacks of manic-depressive psychosis with here as elsewhere marked differences, depending upon the severity of the constitutional taint upon the one hand, or its absence and the presence of a reactive type upon the other. Apprehension and anxiety appear frequently at this period of life, and a true anxiety psychosis is not uncommon. Paranoid conditions occasionally occur and disorders of the sensorium are not infrequent. In a certain group of cases negativistic tendencies are sufficiently marked to make the outward semblance to catatonia quite pronounced.

Certain of the psychoses of this period tend toward more or less physical changes. In other words, the physical side of the disease has to be kept constantly in mind and must be considered to be the most important prognostic feature. The changes incident to vascular degeneration, while of course not usually prominent in the fifth and sixth decades, may well be in evidence, while the changes in the ductless glands are to be borne in mind. Other changes undoubtedly occur, but their nature and their bearing upon the psychosis are not at all understood. The psychosis must, nevertheless, be expressed in psychological terms and the explanation of the mental symptoms must be sought through psychoanalytic study. We have here, then, diseases which have a marked physical side apart from the mental manifestations, the physical side being more prominent than in dementia precox and less prominent than in general paresis.

The Senile Psychoses.—The normal course of life leads to a certain amount of gradual mental and physical deterioration during the later years. When this occurs or not probably depends upon many factors, for we see some men at sixty as old as they should be at seventy-five and other people at eighty presenting a wonderful degree of elasticity and enthusiasm without any apparent falling off in mental powers or interests. This variation was wont to be expressed by saying that "a man is as old as his arteries," but it is certain that there are many other factors besides the condition of the bloodvessels that lead to senile deterioration. In fact senile deterioration may take place and lead to very profound dementia without material disease of the bloodvessels at all. In fact it were better, as Marie[1] suggests, that old age, a physiological condition, should, from an anatomical and clinical standpoint, be distinguished from senility, properly a pathological condition.

Intermediate Conditions.—A certain number of the involution psychoses continue over into the senile period when the patients undergo the mental and physical changes of senile involution. This is in part, at least, the reason for the termination in dementia of a certain group of involution cases, aside from the added obvious fact that vascular degeneration is also an important etiological moment. The cases that show this outcome in dementia are more especially the paranoid types. This group of cases shows, therefore, that there is a gradual transition from the psychoses of the distinctly involution period to those of the senile period, a perfectly understandable condition if we conceive of the psychotic manifestation as being expressions of mental conflicts that are at the basis of the individual character and which express themselves in the later years of life when efficient reaction is becoming progressively less possible, and which later on become fixed, chronic and disintegrating at a time when the physical changes of the senium coöperate to this end.

Normal Senile Involution.—The more usual symptoms of senile involution occurring after sixty are in the main a loss of memory for recent events, due to lack of impressibility to the extent even that events of only an hour before are completely forgotten, lack of ability to recognize faces, marked egotism, so that others' wants and comforts are not considered, which may be associated with some irritability on interference. There is developed more and more as the years go on a true misoneism, so that the patient will positively not tolerate any change in the usual order of things, everything must be done the same from day to day, the same seat is preëmpted, a particular kind of food demanded and the like with other comforts. With this misoneism and the lack of memory for recent events goes a marked tendency to reminiscence. The events of youth and the years long past, unlike those of recent occurrence, are vividly recalled and the patient thus really lives in a world of former days, constantly recalling

[1] Le cerveau sénile, Prog. méd., 1921, vol. 48.

and reiterating things that occurred long ago. This condition becomes progressively worse, the patient leading a vegetative existence almost wholly, no mental initiative, failure of judgment and a progressive loss of comprehension of the environment, so that there is no adequate grasp of the present at all.

With this mental failure goes a corresponding change on the physical side. The signs of age are evident in the wasted muscles, the wrinkled, inelastic skin, gray hair, the raucous voice, arcus senilis, senile cutaneous affections and signs of arteriosclerosis in the superficial arteries. In this condition it should be remembered that the condition of the palpable arteries may not indicate at all the condition of the cerebral vessels. The superficial vessels may show marked arteriosclerotic changes, while the cerebral vessels are in relatively good condition, or, on the contrary, the cerebral vessels may be seriously affected in a person whose radials are comparatively soft and whose temporals are not noticeably tortuous. Similarly with other vascular areas.

Upon this background of dementia there may appear the usual pictures—excitements, depressions, paranoid states, stuporous states and confusions. All of these conditions, however, must be reconstructed in psychological terms to receive any explanation at all, and it is not difficult oftentimes to make out some logical reason for the particular type of delusional formation.

In the senile dement and in the delusions of this period there is noticed an apparent indifference in the emotional sphere, an emotional poverty. Persecutory delusions, delusions of infidelity, hypochondriacal delusions take on grotesque forms and their expression is not accompanied by an adequate affect. A patient tells of severe injuries, of having been shot, while perhaps smiling; another patient tells a long and pitiful story about her affairs and about a claim she has against the government, but tells it in a stereotyped, matter-of-fact way without the expression of any feeling. This condition is usually described as one of emotional blunting, emotional deterioration. Bleuler,[1] however, lays great stress upon what he believes to be the fact, namely, that there is no lack of capacity for feeling, but that the affectivity is only disturbed secondarily, that patients with organic brain disease fail to get a sufficiently clear idea and therefore do not react adequately in the emotional sphere. Wherever it is possible to get a sufficient comprehension of the situation the emotion of the proper quality is manifested and with commensurate intensity.

Simple Senile Deterioration.—This condition of gradually progressing dementia without marked psychotic disturbances is designated as *simple senile deterioration.*

The senile dement is apt to be restless and suffer from insomnia, reversing the time of day and sleeping perhaps in the daytime and lying awake at night, perhaps wandering about the house at night

[1] Affectivity, Suggestibility, Paranoia. Translated by Chas. Ricksher, New York State Hospital Bulletin, February 15, 1912.

in a more or less disoriented condition. This tendency to disorientation, without the development of delusions occurs in the course of the progressive deterioration, and it is where disturbances of the sensorium are marked and confusion becomes very much more in evidence that we have the condition of *senile delirium.* This confusion, however, may be only transitory and is quite characteristically in evidence in the intervals between waking and sleeping.

Senile Delirium.—This is merely a form of senile deterioration in which confusion dominates the picture and in which usually disorders of the sensorium are also markedly in evidence. Patients are highly disoriented, they do not know where they are, or what time of day it is, do not know whether they have just had their dinner, or whether it is time to go to bed or get up, are frequently quite active and irritable and are apt to die from exhaustion. The delirium may take the form of an occupation delirium.

Presbyophrenia.—A certain proportion of these patients resemble very closely the Korsakow's psychosis, being disoriented as to time and place and supplying memory defects by fabrications. One old man, for example, who was so feeble he could hardly stand, relates that he had been working for a man, making some sort of a wire affair for the past seven months.

Course.—The course of senile dementia in its various forms is a progressive one. The patients tend to become profoundly demented, wholly disoriented and die naturally of marasmus. Where the change is somewhat more acute, delirious or confusional episodes may be in evidence from time to time, and of course if the excitement is at all prolonged exhaustion is the result. The patients are naturally susceptible to intercurrent affections, particularly pneumonia, nephritis and cystitis and many of them die in delirium the result of a terminal infection.

Diagnosis.—Paranoid conditions may resemble paranoid states of earlier life, but occurring in the senium, usually show evidences of organic brain disease and marked mental deterioration.

Some of the patients also show similarities to dementia precox, especially those that develop negativistic reactions. Just what relation these cases have to the precox of earlier life is not fully known. The marked mental dilapidation and the period of life at which they develop usually give the clue to the diagnosis.

A few cases show a mixed symptomatology, presenting the picture of senile deterioration on the one hand, together with the various symptoms of arteriosclerotic disease with focal lesions on the other hand. Here are found all sorts of mixtures of pupillary disturbances, disturbances of speech and various forms of paralysis.

Certain borderland conditions show marked emotional states of either depression or excitement which dominate the picture for the time being. These cases show beneath the emotional state the presence of a defect which shows the basal disorder.

The similarity between presbyophrenia and Korsakow's disease is

very considerable. Nouet[1] has made a careful study of the two conditions and has set down the following differential criteria:

Korsakow's psychosis affects persons particularly of adult age; presbyophrenia, on the contrary, belongs to the period of old age, the age of predilection oscillating about seventy years. Presbyophrenia, it is known, is quite uniquely an affection of women, while Korsakow's psychosis is far from being rare among men. The humor of the presbyophrenic is always gay, euphoric and satisfied, quite different from the psychopolyneuritic, who is ordinarily depressed or apathetic and whose face preserves an invariable immobility. The traits of the presbyophrenic are extreme mobility, they laugh, make grimaces in which the mimic muscles participate. The facies of the psychopolyneuritic, however, are always dull and without expression. Loquacity is a symptom scarcely ever lacking in presbyophrenia; the patients talk without stopping about everything, with equal volubility. This symptom is lacking in Korsakow's psychosis. The presbyophrenic is polite, amiable and cordial, characteristics which



Fig. 454.—Presbyophrenic facies.

one seeks in vain among the psychopolyneuritics. Disorders of consciousness are much more marked in the presbyophrenic. These patients have no understanding of their state of illness and content themselves with laughing when one asks a question relative to their physical or mental health. On the contrary the psychopolyneuritic gives a fairly good account of himself and his position, and is the first to lament his situation. The judgment of these patients, even in the chronic forms, is less noticeably affected, and their degree of intellectual enfeeblement less marked. The amnesia, finally, is more profound in the presbyophrenic and besides in this affection the patient does not possess at all a knowledge of this amnesia—the inverse of what one observes in the chronic forms of Korsakow's disease, where the subjects speak spontaneously to their interlocutors of the profound troubles of their memory. Presbyophrenia is rare in its typical forms, but common in the *formes frustes*. Where the symptoms only approach or where certain of them are lacking, the presbyophrenia has, perhaps,

[1] Presbyophrénie de Wernicke et les Psychopolynévrites, L'Encephale, February 10, 1911.

the distinctive characteristics of arteriosclerosis, which are seen much more among these patients than among simple senile dements. Patients die nearly always of cerebral hemorrhage and their nervous centers present at autopsy pronounced atheromatous lesions.

Pathology.—Grossly the brain shows signs of atrophy and is decreased in weight, the bones of the skull are thinned, sometimes in well defined regions, particularly the temporal regions. There is a compensatory external hydrocephalus as a result of the atrophy of the brain. The convolutions are shrunken and the fissures correspondingly widened. The atrophy is not uniformly distributed and may be very much more marked in some areas than others. Arteriosclerosis is a frequent finding, but is not a necessary part of the picture. In fact the two processes are quite distinct in every way, although frequently associated. When arteriosclerosis is present its results may, of course, be found in such lesions as softenings.

The architechtonic of the cortex is greatly disturbed. The nerve cells show advanced degeneration with large quantities of degenerative products of a lipoid nature within them, amounting to a severe grade of fatty degeneration. A very characteristic picture are the basket formations about the nerve cells. The neurofibrils appear to be thickened and produce whirls and loops about the nerve cell. It is thought by some that these basket formations are due to incrustations upon the neurofibrils of perhaps neuroglia origin. The condition is particularly well seen in presbyophrenia.

The miliary plaques are perhaps the most distinctly pathological findings in senile dementia. In fact they are distinctive as much as any pathological finding can be distinctive of any one condition. Their presence in considerable numbers practically makes the diagnosis of senile dementia, while their absence or extreme rarity would negative such a diagnosis. They are small spots of necrosis which are not dependent upon softenings and are in no way associated with the bloodvessels.

There is a great deal of neuroglia proliferation throughout the brain and marked evidences of disintegration and the presence of scavenger cells and the like.

Aside from these changes the usual changes of this period of life are found, particularly in the heart, the kidneys and other internal organs.

Treatment.—The mild cases, especially those that maintain their orientation fairly well, can be cared for at home. Those with marked confusion, especially with a tendency to wandering, need an attendant to be with them. There is danger of their becoming lost and coming to grief, or if they wander about the house at night they are apt to meet with some accident, more often to fall down stairs and sustain fractures. Patients who are very resistive, present surgical troubles, are filthy in habits, or show a tendency to commit sexual offences, should be cared for in an institution.

As regards the more special treatment, little is to be said. Hygienic surroundings, a simple diet, looking after the emunctories, and if

insomnia is present the occasional exhibition of a hypnotic constitutes about all there is to be done. In this class of cases, more perhaps than in any other, is the use of alcohol as a hypnotic indicated. A little whisky and hot water, or a glass of beer or ale acts very nicely. It should be given, however, strictly under medical authority and supervision, as these patients are apt to be susceptible to its influences. In the earlier stages of the disease potassium iodide is the drug par excellence for its general alterative properties and its effect on the arterial tension.

As soon as evidences of mental deterioration appear relief from business worries, cares, responsibilities and mental stresses of all sorts is indicated, with the general hope of limiting disintegration as far as possible. Whether this is of value or not is pretty difficult to state, for it must be remembered that there appear to be certain hereditary tendencies involved even in this condition and that arteriosclerotic disease and senile dementia both appear to be more prevalent in certain families.

Alzheimer's Disease (*Presenile Dementia*).—This disease was first described by Alzheimer in 1906 and since that time a number of cases have been reported. The disease occurs usually in the fifth decade, although cases have been reported in the early part of the fourth decade and as late as the beginning of the seventh. The symptomatology is one of a gradually, often of a rather rapidly progressing dementia, interrupted perhaps with episodes of a certain amount of excitement and anxiety. A rather rapidly progressing dementia in a man of about forty, or a little over, is in itself a rather unusual picture, and in the absence of signs of brain tumor or syphilis, Alzheimer's disease should be thought of. The dementia is marked by a considerable degree of disorientation. The symptoms otherwise are very largely neurological and particularly focalized about the function of speech. Various aphasic and apraxic symptoms are prominently in evidence. Paraphasia and asymbolia are quite frequent. There may also be spasticity and convulsions, although there are never well marked paralyses.

The pathology of this condition throws almost the only light upon its nature. There is marked and extensive degeneration of the nerve cells with disturbances of cortical architechtonic and the presence of large quantities of disintegration products, while there are found numbers of basket formations and typical miliary plaques. The condition of the bloodvessels is quite normal. The pathological picture thus resembles closely that found in senile dementia. In general, the disease is considered as a presenile dementia, although some believe it to be a distinctive disease.

Thus to express the situation as does Stertz:[1] The histological findings are not the expression of a disease process which forms its own laws in the sense of paresis, for instance, but they are the signs of cortical involution which follows its own laws. The differences

[1] Zur Frage der Alzheimerschen Krankheit, Dtsch. Verf. f. Psychiatr. Jahresvers., Dresden, 1921, **4**, 25 u. 26.

between senile and presenile dementia are dependent upon the difference in the functional system which gives out. There is no difference in the histopathology perceptible. Presenile dementia presents a clinical picture indicating a distinct tendency to the localization of the disease process while in senile dementia the picture indicates a much more general failure. In presenile dementia the impressive and expressive functions which mediate the contacts with the external world are affected from the beginning; in senile dementia these faculties are preserved up to the very last stages.

The presence of this disease, if it be considered a presenile dementia, offers another one of those warnings to us not to be too dogmatic. It would appear that the senium is by no means a clearly defined period of life, and that the pathological changes which are usually thought of as dependent upon old age, may occur within wide limits and perhaps represent failure of special tissue resistances.

Arteriosclerotic Psychoses.—These psychoses are dependent, on their physical side, upon arteriosclerotic changes in the cerebral blood-vessels, and this condition is due in turn to the general causes which produce arteriosclerosis. In the main the two characteristics present are certain mental changes combined with cerebral defects. It must be borne in mind, however, that an advanced degree of arteriosclerosis may exist, particularly in the peripheral vessels, and the cerebral vessels retain their elasticity, while on the contrary the cerebral vessels may be severely sclerosed while the peripheral, palpable vessels show little change. Arteriosclerosis is essentially a regional disease. Combined with the arteriosclerosis may be extensive somatic disease, alcoholism and syphilis modifying and complicating the picture.

The psychoses of arteriosclerosis form another one of the connecting links which join the periods of involution and the senium. Many of the involution psychoses merge into arteriosclerotic deterioration, and arteriosclerosis is frequently combined with the changes incident to the senium.

Aside from the usual causes of arteriosclerosis there are undoubtedly psychological causes at work. The hypertension which is conceived as so important an element in the causation of arteriosclerosis has to be thought of as a heightened visceral tonus and as such dependent, to some extent at least, upon psychological factors. There would seem also to be certain hereditary factors at play. Certain families show a high incidence of death dependent upon arteriosclerotic disease.

There are four fairly well defined varieties of this disease based upon both clinical and pathological findings as follows:

1. **Arteriosclerotic Brain Atrophy.**—This occurs in two forms: a mild form with severe arterial sclerosis but an absence of focal brain lesions. The symptoms are easy fatigue, slight failure of memory, dizziness and headache. The severe type may resemble the mild at first but is progressive, leads to profound dementia and presents in its course apoplectiform and epileptiform attacks and focal symptoms.

2. **Subcortical Encephalitis** (Binswanger).—In this condition the white matter is largely involved as a result of disease of the long medullary arteries. Apoplectiform and epileptiform attacks occur and also transitory attacks of confusion, aphasia and paresis, disturbances suggesting focal lesions. Focal lesions are not found extensively but areas of softening often occur in the basal ganglia.

3. **Perivascular Gliosis.**—In this condition there is a disappearance of nervous elements about the diseased vessels and replacement by neuroglia.

4. **Senile Cortical Devastation.**—Here extensive destruction of cortical areas in the vascular territories of the diseased vessels is found. In this condition the arteriosclerosis is localized largely in the small cortical vessels which come off from the pia. The basal vessels remain relatively normal.

Pick has very thoroughly described certain large atrophies involving whole lobes or portions of lobes. The occipital lobes may be involved, producing blindness, or the temporal, producing deafness, for example. The atrophy, however, does not always follow a vascular area, and so, while it is generally supposed to be due to arteriosclerotic disturbances in the irrigation of these territories, the cause is not always altogether clear.

Symptoms.—In the main the symptomatology of cerebral arteriosclerosis is one of gradually progressive mental deterioration, to which are added the evidences of focal lesions which are the results of thrombotic softenings. (See Chapter on Hemorrhage.)

The prodromal disturbances of the arteriosclerotic psychoses are very apt to extend over a considerable period of time and manifest themselves in the main as nervousness and irritability, with headache, dizziness, insomnia, associated of course with the special signs of the vascular disease, more particularly, as a rule, high blood-pressure. With this series of symptoms there may be, of course, associated a certain amount of deafness, with sclerosis of the drum membrane, cardiac attacks with the Stokes-Adams syndrome and evidences of interstitial nephritis. Quite frequently, too, these patients show the signs of arteriosclerosis of the vessels of the spinal cord, with perhaps some sclerosis of the palpable vessels. On the mental side the patient may have a feeling of growing inefficiency, at least his work shows a falling off in efficiency, which characteristically manifests itself at first in a failure to do the creative things, easy fatigability, beginning memory defect, and attention disorders. There is apt to be insight and a certain amount of depression. Pick has called particular attention on the emotional side to a lack in the finer modulations of the emotions. Bleuler's suggestion must be remembered, that patients with organic brain disease fail to show a natural emotional reaction, not because of any defect of emotion but because of a lack of grasp of the situation, and when the situation is fully appreciated an adequate emotional response issues.

These prodromal symptoms gradually merge into symptoms of

greater severity and may be punctuated from time to time by attacks of excitement or of depression and delusions may develop which are characteristically of the paranoid type. The following case illustrates this paranoid trend very well: A man who had been a successful business man in his younger days began to fall off in efficiency as he entered the arteriosclerotic period of life and finally entered one of the Soldiers' Homes as he was unable to adequately support himself. Here, because of his training as an accountant, he was employed in the office. While employed here the characteristic series of paranoid ideas developed. In the first place he developed exalted ideas of his own ability which were distinctly defensive in character, compensating him for the real fact, his failing ability. Alongside of these exaggerated ideas he had delusions of a persecutory character. He was interfered with by those about him who were envious of him because he had secured such a good position in the Governor's office. This is another defense reaction in the opposite direction and serves to explain to him how a really efficient man after all can turn out such poor work. It is not his fault, but the fault of those about him, and so he is again saved from the realization of his failings. Removed from this situation and taken to a hospital where he could no longer indulge in alcohol, which had been a factor in bringing about this condition, he recovered from these distinct psychotic manifestations, but without, however, a full insight into what his condition had been. This lack of insight probably had its basis in the organic changes. The man was no longer capable of adjustment, except within narrow limits and while removal from the painful conditions relieved the situation, he was unable to fully understand it.

This case shows very well how even in a psychosis dependent upon organic brain disease the mental symptoms as such must receive a psychological interpretation.

From time to time these patients show periods of confusion and bewilderment with disorientation. These periods may be of considerable duration and some of them at least are dependent upon thrombotic attacks which, when they do not occur in the motor area, are easily overlooked. Even though they do occur in the motor area the disturbance of consciousness may not be very great, or at least there is nothing comparable to the unconsciousness and coma of cerebral hemorrhage; there is, perhaps, only a slight confusion, and the paralysis, if it exists, is only very slight and is often explained by the family as the result of some inconsequential cause. Then, again, the patient may be so blunted mentally as not to complain of a slight impairment of function.

These patients are especially susceptible to alcohol and become easily very badly confused from small quantities.

One of the characteristic features with regard to this group of psychoses is that the so-called "nucleus of the personality" is well preserved. The patient preserves all of the outward appearances of his old self until the mental dilapidation, owing to a progressive nar-

rowing of the field of interests, has reached an extreme degree. There is not, as a rule, that marked "change" in the individual that is seen in some of the psychoses.

The areas of softening produce focal lesions and these focal lesions are most prominent in the motor areas, producing various upper motor neuron paralyses, and in the speech areas producing various forms of aphasia and apraxia. The marked focal lesions, particularly those which lead to well defined speech disturbances, hasten very greatly the dementia. An aphasia which puts the patient out of actual touch with his fellows hastens the tendency to mental deterio-

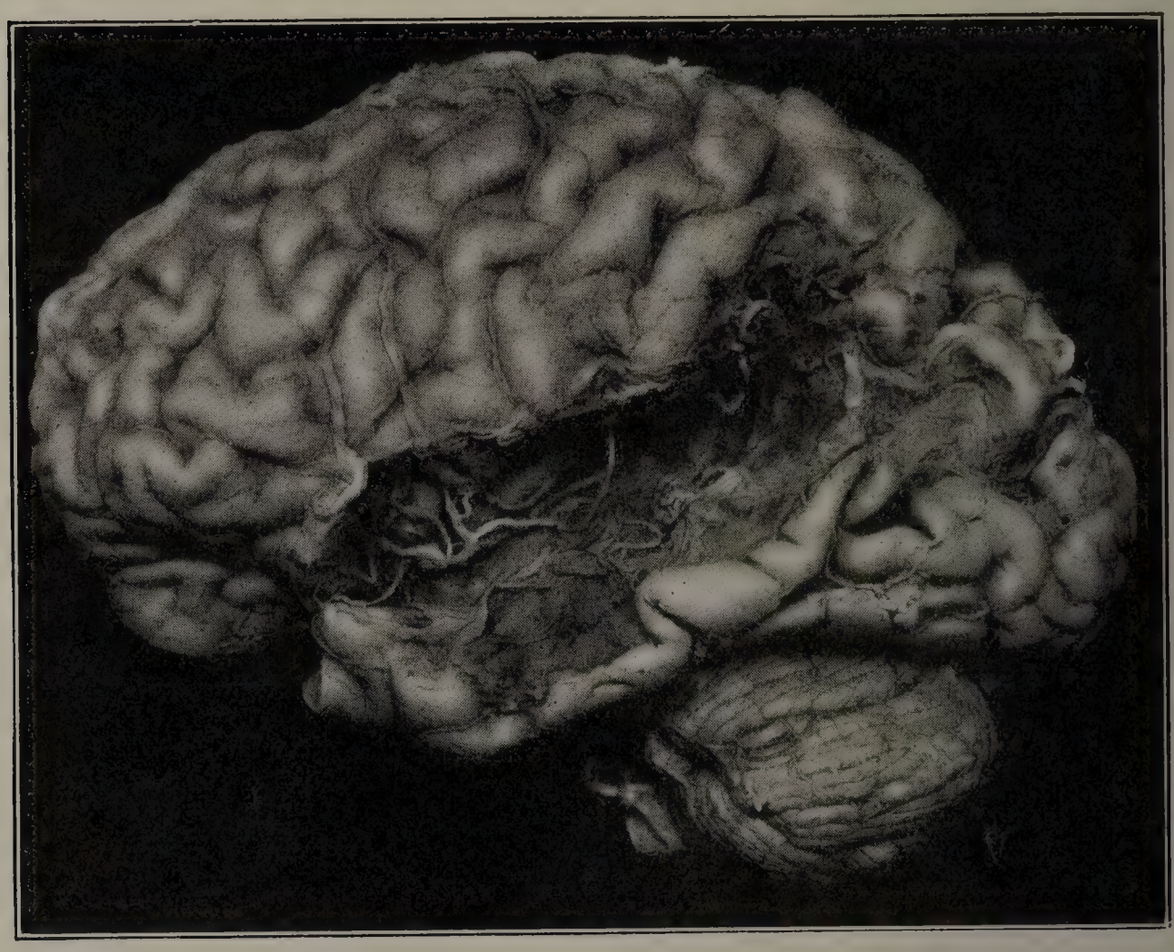


FIG. 455.—Extensive thrombotic softening, the result of arteriosclerosis. Patient aged eighty-three years. There were also softenings on the right side in the lower portion of the cuneus and upper portions of the lingual and fusiform lobules. There is general atrophy of the convolutions.

ration in this class of patients who are no longer fluid, but are well along on the downhill path of life. They need every stimulus of mental activity to even hold their own, and as soon as an important function like speech is destroyed they are quite apt to lapse promptly into a serious deterioration.

It is in this class of patients that, as a result of the focal lesions, epileptiform attacks develop late in life—the so-called "late epilepsies." Their significance in the main is in pointing to the focal disorder and as an indication that the patient needs careful oversight.

Pathology.—The pathology shows the arteriosclerotic process in the cerebral vessels in various stages of progress. There may be miliary

aneurisms of the smaller vessels and other vessels may be completely occluded with resulting areas of softening. These areas of softening usually show entire disintegration of the nervous elements with large numbers of scavenger elements about. The nerve cells show various grades of degenerative change dependent upon deficient nutrition as a result of decrease in size of the lumen of the vessels. There is neuroglia overgrowth about the vessels and in the degenerated territories.

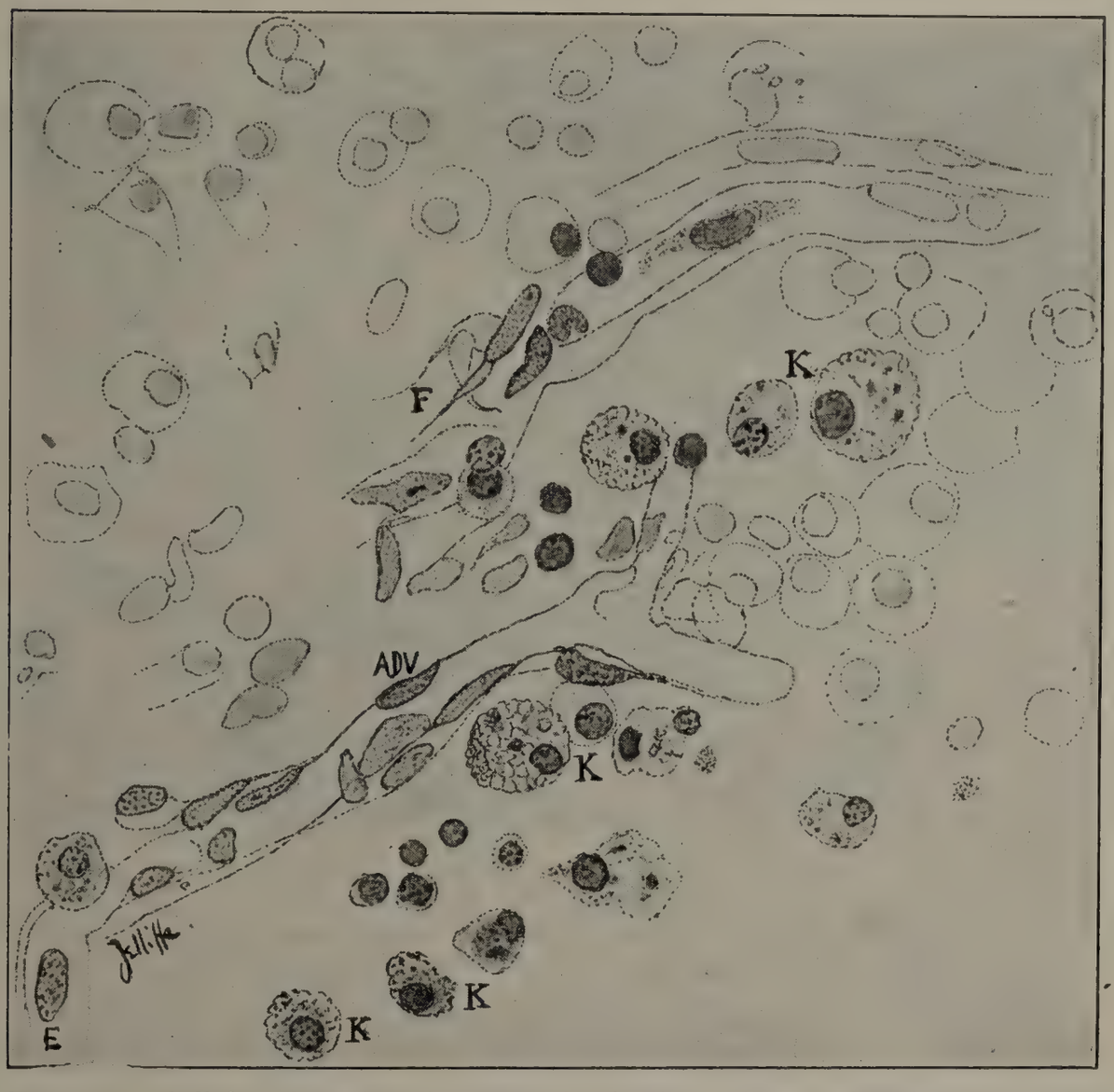


FIG. 456.—Cellular alterations in areas of softening. Arteriosclerosis, acute softening, lacunar softening, senile deterioration, etc.: *K*, Körnchenzellen; *E*, endothelial cells; *ADV*, adventitia cells; *F*, new fibroblasts.

Diagnosis.—The diseases most apt to be mistaken for the arteriosclerotic psychoses are paresis and the psychoses of cerebral syphilis, which should be considered together now that the etiology of paresis has been so thoroughly established. The reason for the possibilities of mistake is dependent upon the fact that both processes are more or less diffuse, producing a progressive mental deterioration, and that both processes tend to be more severe at certain points and thus produce focal symptoms. Syphilitic and metasyphilitic disturbances occur, as a rule, at a much earlier age than the arteriosclerotic, gen-

erally not later than the fourth decade, while the arteriosclerotic period is not entered until the fifth decade. When an arteriosclerotic has had syphilis also the differentiation becomes more difficult. The Wassermann of the cerebrospinal fluid, however, would be negative, while with large areas of softening on the surface of the cerebrum there would be considerable evidence of disintegration products in the cerebrospinal fluid. On the mental side the preservation of the "nucleus of the personality" is much more in evidence than in paresis.

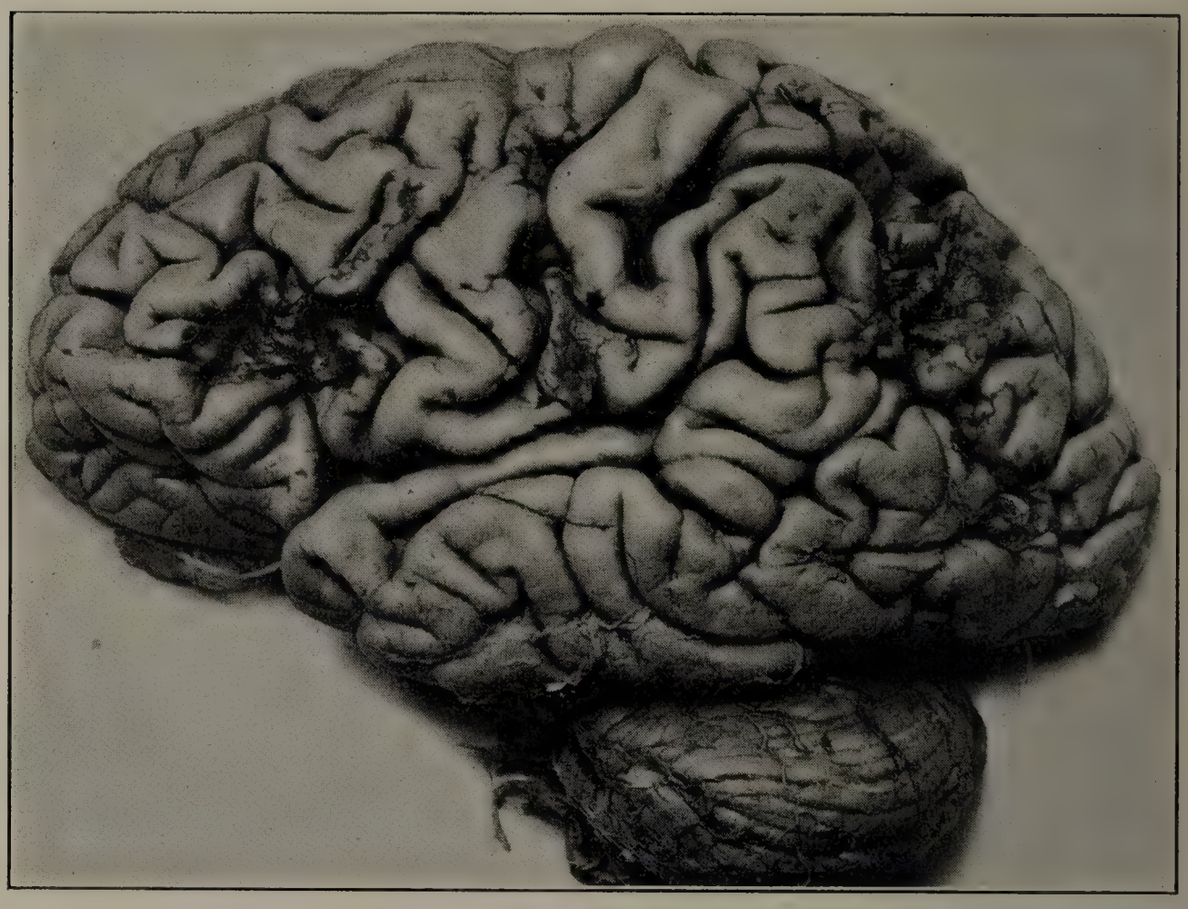


FIG. 457.—Widely distributed arteriosclerotic softenings; patient aged seventy-two years. There were also numerous softenings in the basal ganglia. Brain greatly shrunken. It can be seen from the distribution of these lesions how the clinical picture might simulate paresis.

Treatment.—In a general way the treatment should be prophylactic as far as possible. With the first symptoms of prolonged and intractable high blood-pressure the individual should be removed from the influences of physical and mental stress, and the usual means should be employed to keep the blood-pressure down and to reduce or do away with any toxemic condition present. A carefully regulated diet from which alcohol and tobacco are excluded, hydrotherapy intelligently applied, possibly a visit to some watering place, and in some instances a visit to a somewhat higher altitude, with moderate outdoor exercise and the exhibition from time to time of drugs to reduce the pressure and, especially where there are kidney complications, the drinking of considerable quantities of mildly alkaline water are in general the things to be depended upon. Insomnia has to be dealt with, and in cases where the patient is under absolute control

a small amount of alcohol at night will sometimes produce the desired result. Unless the patient is under absolute control, however, it is best to give hot milk, or such simple hypnotics as veronal.

Senile Epilepsy.—So long as epilepsy continues to be such a vague concept, continues to include so many essentially different things which are only grouped together because of the apparently common factor, the convulsion, just so long will the classifications and designations continue to rest upon convenience rather than upon any surer footing. In this way *senile epilepsy* comes commonly to be spoken of. Redlich[1] would regard all cases as senile which occur after the sixtieth year. A more detailed analysis will show that some of them are to be classed in the group of the essential epilepsies; others must be regarded as of autotoxic origin, such as diabetes; in others the seizures are the first manifestations of a progressive senile paralysis; in still others the etiology may be luetic. In some cases the seizures may be due to tumors or cysticercus. The dominant factor in senile epilepsy, however, is arteriosclerosis. Redlich believes the senile plaques have a special connection with the appearance of epilepsy at advanced age and that it is necessary to assume an atypical form of senile dementia which is related to the usual form as Lissauer's type of paresis is related to the more usual, ordinary type of the disease. It is found that the cases of dementia in which seizures occur show plaques and fibrillary degeneration in Ammon's horn as the place of predilection. Redlich suspects that in cases of senile epilepsy without dementia the characteristic changes are confined to the cornu Ammonis.

[1] Ueber senile Epilepsie, insbesondere deren Beziehung zur senilen Demenz und der sog. Alzheimerschen Krankheit. Allg. Zeitschr. f. Psych., 1920, vol. **76.**

CHAPTER XXIV.

IDIOCY, IMBECILITY, FEEBLE-MINDEDNESS, AND CHARACTEROLOGICAL DEFECT GROUPS.

WITHIN the past few years the defective classes have assumed enormous sociological significance, so that this chapter has easily become perhaps the most important in the whole group of disorders at the psychological level, considered from the social angle. Of course only a brief outline of the various types of defect can be given in a text-book such as this.

In drawing a distinction between dementia and idiocy Esquirol said: "The demented man is deprived of the good that he formerly enjoyed; he is a rich man become poor; the idiot has always lived in misfortune and poverty." In other words the idiot, the imbecile, and the feeble-minded lack something; the psychotics are suffering from a disorder of that which they possess.

This definition, so admirably worded by Esquirol, adequately expressed the distinction between the insane and the idiot and imbecile according to the knowledge of his day, and has been used continuously since to express such distinction. It can no longer be said to be a valuable formula except it be used with many reservations and explanations.

In the first place, from the standpoint of this book, there is no such thing as insanity, at least in a medical sense, as Esquirol used that term and as other psychiatrists in the past and the present have used it. Insanity is purely a legal and sociological concept, and as such does not imply anything more than the judgment of a man's fellows on the desirability of having him live in the community. Defectiveness, under which term the various grades of idiocy, imbecility, and feeble-mindedness are included, is quite as generic a term, and while it refers to a condition the fundamental characteristic of which is lack of development, it includes a great variety of states, and in its application is a relative term only.

It is quite as illogical to group all defectives together and endeavor to draw a conclusion from their study as a whole, as it is to group all of the so-called insane together and endeavor to draw a conclusion from the study of all of them. There is obviously very little similarity between the cretin and the defective as a result of cerebral hemorrhage during a prolonged and difficult labor, and therefore any principles or conclusions which are reached as regards the whole defective class without an adequate appreciation of the multitude of different conditions that are found under this generic caption must be avoided.

It must be remembered, too, that with any defective patient it is possible to have associated a psychosis, so that the picture may be still more complicated. Therefore in other parts of this book various forms of what are grouped in this chapter are treated of from an etiological standpoint.

In considering the etiology of various defect states the important general principle that should be borne in mind is the location of the defect; (1) as to whether it is in the germ plasm or acquired, and (2) as to the time of development of the individual when it became operative. It is only those defects which are the result of a lack of some element in the germ plasm that are truly of a hereditary nature.

The process of development may be interrupted at any time from the beginning of the growth of the child in the uterus to the attainment of its complete adult development, and as this interruption may take place during intra-uterine life it is in such instances that it is important to make the distinction between a truly inherited and an acquired defect. In both instances the defect is congenital, that is, exists from birth.

Disease or injury may affect the child during intra-uterine life or during the process of birth or after birth and during the processes of extra-uterine development. Previous to birth, illness or injury to the mother are the common etiological factors; during birth asphyxiation and injury by the forceps are common causes, while after birth the infectious diseases and direct injuries enter largely into the etiology. Finally, there are relative conditions of defect which are due to lack of the opportunity to develop, such defect, for example, as results from the deprivation of the important sense organs, as the eyes and ears. If the patient be born blind and deaf, under ordinary circumstances he will be very defective mentally, because he has not the opportunities for learning which the ordinary child has. On a still different plane relative defect due to sordid and unsanitary conditions and lack of educational facilities is found. Children are ignorant who have no opportunity to go to school and learn, and may even lack the ordinary brightness that comes about spontaneously if they have lived under insanitary conditions that impaired their general health and energies, especially if these conditions be complicated by the presence in the child of some such debilitating factor as infected tonsils, adenoids, high degree of myopia, otitis media with deafness, and such other like things which impair the general health and vigor of the child and interfere with the patency of the avenues through which he gains his information of the outer world. This general enfeeblement of the child may also be the result of debilitating habits, such as masturbation and the use of narcotics and alcoholic drinks, the latter of which especially are an extreme expression of a vicious environment.

Classification.—The general group of defectives is a very wide one and includes not only the idiots and the imbeciles and the frankly feeble-minded, but the still higher grades of backward children, and

then a whole borderland group of ne'er-do-wells in whom the defect is much less clearly defined than in the lower grades.

David Starr Jordan has said that "A good citizen is one who can take care of himself and has something left over for the common welfare." This saying represents in a general way the basis on which the classification of this group may be based. The defectives as a class may be said not only not to have anything left over for the common welfare, but only in the highest grades, and then under the most favorable circumstances, can they care for themselves. A general basis of classification has been their educability and their capacity to care for themselves, according to some such scheme as follows, for example:

Feeble-mindedness.—A condition of mental defectiveness capable of much improvement by educational methods. The afflicted individual may ultimately take a place in the world and be self-supporting under favorable circumstances.

Imbecility.—A condition of mental deficiency which can, however, be materially improved by training, but not sufficiently for the subject to take a place in the world.

Moral Imbecility.—A condition of mental defectiveness which is shown predominantly by the absence of the highest functions, particularly the moral; capable of training to a considerable degree, but always a menace to society.

Idio-imbecility.—A condition midway between idiocy and imbecility.

Idiocy.—A condition of profound mental defectiveness. The lower grades are unteachable, while the higher may be trained slightly in self-help, *i. e.*, to attend to the calls of nature.

More recently an attempt has been made to define with greater accuracy the different grades of defect, and this effort has taken the form of an attempt to correlate the psychological development of the defective with the psychological development of the average child, so that the defective as a result of this correlation is said to correspond to the development of the average child at such and such an age. In other words, age has come to have a psychological rather than a chronological significance, and an individual who may be forty years old, but who is only developed mentally to the extent that an average child is developed at the age of seven, is spoken of as having the psychological age of seven years.

Until the use of this scheme of classification all people were classified among other ways, in accordance with their chronological age. For example, in this country a person attains his majority and can cast his vote at the age of twenty-one. This refers of course entirely to the chronological age. It can easily be seen that if there are a material percentage of persons in the community who are defectives, although they may have acquired the chronological age of twenty-one they have not the mental development and the judgment that it is expected goes with such an age, and therefore from the psychological standpoint

are not twenty-one and ought not on that basis be permitted to vote. The psychological classification of the age as outlined by the Binet-Simon test is a far more accurate way to standardize the mental development of the individual than the chronological age method, and although there are objections which may be urged against it and although it is concededly not a perfect method and is at present undergoing gradual modifications, still it is so much better than the chronological method that it should be used in its place, and is by far the best standard which we have for designating the development of the defective. The classification, according to this method which has been adopted by the American Association for the Study of the Feeble-minded, is as follows:

Mental age.	Capabilities.	Class.	
Under one year	Helpless	Low	Idiot.
1 year	Feeds self. Eats everything	Middle	
2 years	Eats discriminatingly	High	
3 years	No work. Plays little	Low	Imbecile.
4 years	Tries to help		
5 years	Only the simplest tasks	Middle	
6 years	Tasks of short duration. Washes dishes		
7 years	Little errands in house. Dusts	High	
8 years	Errands. Light work. Makes beds	Low	Moron.
9 years	Heavier work. Scrubs, mends, lays bricks, cares for room with simple furniture		
10 years	Good institution helpers. Routine work	Middle	
11 years	Fairly complicated work with only occasional oversight		
12 years	Uses machinery. Cares for animals. No supervision. Cannot plan	High	

The classification, according to the psychological age, while it is practically useful for defining the stage of development which the individual has reached, is also of some value in differentiating hereditary and acquired conditions. Somewhere from 65 to 80 per cent. of defectives have feeble-minded parents, and therefore in most of them the condition is hereditary. In these hereditary cases it would seem as if the child developed quite normally up to a certain point and then stopped rather suddenly, and that this stopping was a pretty general one, so that the development ceases at a certain level without many irregularities. A patient, for example, will test to seven years of age, will do practically all of the tests for seven years, and fail completely with all of the tests above that. In other words, the age is very accurately seven. The defect is a clean-cut one. When this condition of affairs is found there are reasons to suppose that the trouble lies in the germ plasm.

In acquired conditions the stoppage is not so abrupt as a rule. For example, with the deterioration that goes along with epilepsy in childhood, the tests show much greater irregularity, a much more uneven development, and this might be expected of any condition in which deterioration was going on. In numerous deteriorating conditions such results are found and therefore it can be seen that one is dealing

not only with a stoppage of development, but with a disease process which, while it is in operation, is producing symptoms of its own.

The various schemes of determining the psychological age are all, largely if not altogether, measures of intelligence. The affective aspect of the mental life is not taken into consideration hardly at all in any of the plans. The way of expressing the results of any plan of testing are (*a*) mental age (M. A.). This means the results of the tests as set over against the normal child. If a child performs the test that a ten year old normal child performs, then it is said to be psychologically ten years old no matter what its age chronologically. (*b*) Age Difference. This is the difference between the mental age as determined by the tests and the chronological age. (*c*) Intelligence Quotient (I. Q.). The intelligence quotient is the ratio of mental age to chronological age. (*d*) Co-efficient of Intelligence (C. I.). The ratio of the point scale score to the expected or normal score. (*e*) Index of Brightness (I. B.). Expressed by the number of points over the normal for that age. (*f*) Percentile Rank (P. R.). The methods used most frequently for testing are the Point scale[1] and the Stanford Revision[2] of the Binet-Simon scale.

The most useful classification of the feeble-minded is a clinical one. It would be quite as absurd to enter into a discussion of the general psychology or the general pathology of defectives as it would be of the so-called insane, and therefore in this chapter the different well defined forms will be briefly and separately described.

The following is Weygandt's[3] classification based upon an etiological-clinical grouping.

1. Mental defect as the result of sensory deprivation.
2. Fundamental defect due to injury to the germ plasm including microcephaly, anencephaly, hydranencephaly, hemicephaly.
3. Mental weakness due to encephalitis including lobar sclerosis, porencephaly, cerebral infantile palsy.
4. Mental weakness due to meningitis including defect states following tuberculosis and endemic cerebrospinal meningitis.
5. Hydrocephaly.
6. Amaurotic family idiocy.
7. Tuberous hypertrophic sclerosis.
8. Mongolism.
9. Infantilism.
 - (*a*) Dystrophic infantilism resulting from cardiopathy, nutritional disturbances, tuberculosis, lues, malaria, effects of toxins such as alcohol.
 - (*b*) Dysglandular infantilism.
 - (*aa*) Status thymicolymphaticus and thymic idiocy.

[1] Yerkes, Bridges and Hardwick: Point Scale for Measuring Mental Ability, Pub. by Warwick and York, Baltimore, Md.

[2] Terman, L. M.: The Measurement of Intelligence, Pub. by Houghton Mifflin Company, Boston.

[3] Erkennung der Geistesstörungen, München, 1920.

(*bb*) Dysthyreodism due to defective development of the thyroid including myxedema, cretinism, cachexia strumipriva. Also is included hyperthyroidism, Basedow's disease.

(*cc*) Dysgenitalism.

(*dd*) Dyspituitarism, hypophyseal dysfunction rarely with acromegaly and giantism when the disease begins in youth, dystrophia adiposo-genitalis. Also certain other forms of hypophyseal giantism.

(*ee*) Epiphyseal disorders.

(*ff*) Pluriglandular diseases.

10. Idiocy from hereditary lues.
11. Mental defect due to abuse of alcohol by parents.
12. Athetotic idiocy usually with symptoms of encephalitis. Little's disease may also be associated with mental defect.
13. Choreatic weak-mindedness.
14. Spasmophilia.
15. Idiocy with rachitis.
16. Chondrodystrophic weak-mindedness.
17. Oxycephaly.
18. Early forms of dementia precox.
19. Dementia infantilis.
20. Early forms of manic-depressive psychosis.
21. Hysterical degeneration.
22. Neurasthenia.
23. Traumatic idiocy.
24. Minor groups: mental disease and interferences in development in childhood due to tuberculosis, multiple sclerosis, familial spastic spinal paralysis, Westphal-Strümpell's pseudosclerosis or Wilson's disease, Friedreich's ataxia, etc.

It will be noted that groups 18 to 22 are transition forms to the more definite types of psychoses as they are found in adults.

Clinical Varieties.—Amaurotic Family Types (*Tay-Sachs Disease*).—This is a disease which generally affects more than one child in the family and appears to be confined in its incidence to the Jewish race. Its etiology is unknown. Its pathology is in general a degeneration of certain elements of the brain, more particularly the cortical elements and pyramidal tracts, including particularly the optic nerves. It has been described in three stages:

First Stage.—The infant is usually all right at birth and the disease does not make itself manifest for some few months thereafter, usually at about the fourth month. At this time the first symptoms observed are some weakness in the neck muscles and indications of dimness of vision. If the fundus is examined during the fourth or fifth month there will be found a whitish-gray symmetrical patch, oval in shape, with a horizontal axis accupying the macula lutea. In the center of this patch is seen the fovea centralis which appears as a dark cherry-

red spot. Optic atrophy follows, and later total amaurosis. *Second stage:* In this stage the weakness of the neck muscles is more marked and the head falls backward if unsupported, and while lying on the back the infant is unable to turn over to either side. The handgrasp is noted to be feeble, objects are dropped and the infant is generally apathetic. The vision is materially reduced in this stage, but the senses of taste and hearing are preserved, the sense of hearing appearing to be unusually acute. *Third stage:* In this stage the affected muscles are atrophied, and later the atrophy extends to all of the muscles of the body, emaciation becomes marked, the reflexes exaggerated, and late in the course of the disease the extremities become rigid, and there is retraction of the head.

Spasmodic contractions and convulsions have been noted. There is at no time any rise in temperature, and the thoracic and abdominal viscera remain normal. Death usually occurs in less than two years.

These cases of early blindness, inbecility and paralysis constitute the infantile type of the disease. A juvenile type has been described which, aside from occurring later in life may differ in other particulars, as, for example, in the absence of paralysis, the preservation of the macula, and in not apparently being confined to the Jewish race.[1]

Sclerotic Types.—Certain types of mental defect are seen associated with a condition of the brain which in general may be said to be due to an overproduction of neuroglia tissue and corresponding atrophy and disorientation of nerve elements. The exact nature of this process is not definitely known. It is not improbable that there are a number of different conditions comprised in this general picture.

The sclerosis may be diffused pretty generally or it may be localized in patches and the affected portions may be atrophic or they may be hypertrophic. When the process is hypertrophic and involves a large portion of the brain one finds what has been called a hypertrophied brain. The brain is much larger and heavier than the normal, and is much firmer in consistence after being hardened. It does not look like a normal brain, but the surface has a cauliflower appearance. The condition is usually associated with grave degrees of mental defect and with epilepsy. The localized varieties are more apt to be associated with convulsions than the diffuse, while there may also be noted marked tremors.

This disease has been recently correlated with other changes than the local cerebral changes. Kufs[2] in a valuable extensive article, including autopsy material, has accumulated the evidence for a distinct disease entity which comprises various manifestations. In the first place, besides these changes in the brain, which have briefly been referred

[1] Feldman, J.: Der jetzige Stand der Lehre von der Tay-Sachsschen familiären amaurotischen Idiotie, Klin. Monatsbl. f. Augenheilk, 1919, vol. **63.**

[2] Beiträge zur Diagnostik und pathologischen Anatomie der tuberösen Hirnsklerose und der mit ihr kombinierten Nierenmischtumoren und Hautaffektionen und über den Befund einer akzessorischen Nebenniere in einen Ovarium bei derselben. Ztschr. f. die gesamte Neurologie und Psychiatrie, Band xviii, Heft 3.

to, there is a distinct cutaneous affection in the form of adenoma sebaceum, which affects more particularly the face and the back. Along with this condition is frequently found associated mixed tumors of the kidney—tumors made up of various elements of which the smooth unstriped muscle tissue is the most prominent.

These three symptoms, then, adenoma sebaceum, mixed tumor of the kidney, tuberous sclerosis of the brain, are the triad which constitute the most important evidences of this disease entity. In addition to this pathology it is to be noted first, about the sclerosis that it involves sometimes the cerebellum, that tumors of the ventricles are quite characteristic, and that relatively commonly there is associated with this triad of symptoms rhabdomyoma of the heart. Myoma of other organs, such as the stomach and the uterus have also been observed.

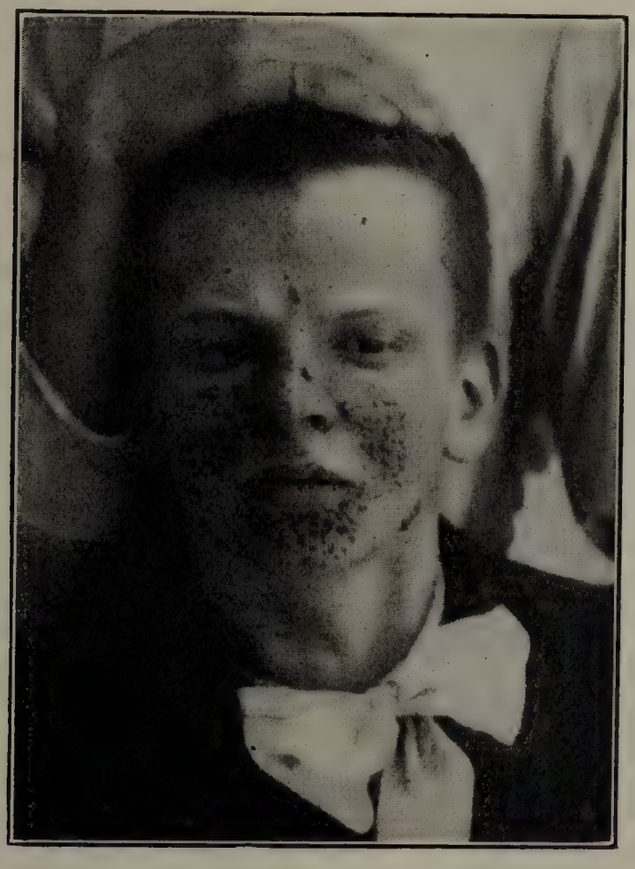


FIG. 458.—Adenoma sebaceum. The skin affection associated with tuberous sclerosis of the brain, a profound degree of mental defect and various tumors of the viscera, especially the kidneys. (Courtesy of Dr. Martin W. Barr.)

In the blood relations of these patients suffering from tuberous sclerosis remarkable skin anomalies are often met with. Atypical nevi and other atypical skin conditions occur, while skin malformations that belong in the nevi class are found in patients with epilepsy on a tuberous sclerosis basis. The distribution is mostly on the back. The upper skin alone is essentially affected, usually in the form of a circumscribed hyperkeratosis.[1]

While on the mental side, along with this condition there usually is associated a marked degree of mental defect with epilepsy, still

[1] Weygandt, W.: Hautveränderungen bei tuberösen Sklerose, Arch. f. Dermatol. u. Syphilis, Orig., 1921, Bd. **132.**

Kirpicziks has reported a case occurring in a man twenty-eight years old without mental impairment.

Cretinism.—This disease is endemic in certain parts of Europe, but so far as we come in contact with it in this country is sporadic.

The disease is due to a defect in the secretion of the thyroid gland. All degrees of defect may be present, from athyroidism through the various degrees of hypothyroidism. The several degrees of cretinism which are described and which correspond with different degrees of defect are three, namely, the lowest grade, in which the defect is greatest, the cretins; the middle grades, the semicretins; and the highest grades, the cretinoids.



FIG. 459



FIG. 460

FIGS. 459 and 460.—Cretinism before and after treatment. (Josefson.)

The disease usually begins quite early in the life of the child, sometimes during the first year, although it may be delayed for several years. The general symptoms are those, *first*, of retarded development; the child appears less bright than he should be, walking is learned slowly, speech is delayed in development, the anterior fontanelle is late in closing; and *second*, the characteristic cretinous appearance. The body is dwarfed, the head relatively large, and the legs short and bowed, hands and feet stumpy, and the ossification of the bones is delayed. The appearance of the face is typical, the nose is broad and

flattened, the lips are thick, the tongue thick and often protruding from the partly opened mouth, the eyes widely separated, the eyelids often heavy and swollen, and the hair coarse and scanty. The skin is cachectic in appearance and dry and thick, resembling the skin of the myxedematous patient, the neck is short, the abdomen protuberant, sometimes with umbilical hernia. The signs of puberty are late in making their appearance, there is often a failure in the complete development of the genital organs, and many of these patients are sterile. The pulse and respiration are slow, the temperature may be subnormal, and the movements of the patient are usually very deliberate. Mentally the cases show various grades of defect from the



FIG. 461



FIG. 462

FIGS. 461 and 462.—Showing bony changes before and after treatment. (Josefson.)

lowest grades of idiocy through the various degrees of imbecility. In general cretins are quite good-natured, pliable individuals who are easy to get along with and care for.

Diagnosis.—In the matter of diagnosis the principal conditions which have to be differentiated are rickets, achondroplasia, and mongolism.

Rickets can usually be differentiated by the characteristic beading of the ribs, the symmetrical enlargement of the epiphyses, and the absence of the typical signs of cretinism.

Achondroplasia should not be mistaken for cretinism. There is an imperfect development of the long bones, but none of the signs of cretinism, particularly none of the impairment of mental development.

Mongolism is at times extremely difficult to differentiate. It is very important that this condition be not mistaken for cretinism, particularly with reference to the matter of treatment, as treatment is capable of modifying the cretin, but not the mongol. The following table of differential signs taken from Shuttleworth and Potts,[1] will serve to point out the detailed characteristics between these two conditions:

Mongolism.	Cretinism.
1. Characteristics noticeable from birth.	1. Characteristics often not noticeable until sixth or seventh month.
2. Skull brachycephalic: contour rounded or short oval; longitudinal and transverse diameters nearly correspond.	2. Skull dolichocephalic: flat at top (fontanelles close late), expanded laterally; broad behind, often asymmetrical
3. Forehead usually smooth.	3. Forehead usually wrinkled.
4. Palpebral fissures "almond-shaped," and more or less oblique upward and outward. Frequent epicanthus. Strabismus common. Ciliary blepharitis frequent.	4. Palpebral fissures horizontal, but appear small, owing to pseudo-edema of eyelids. Strabismus and ciliary blepharitis less common.
5. Cheeks chubby, often florid. Complexion mottled.	5. Often circumscribed malar flush; complexion ashy or waxy.
6. Lips often transversely fissured. Lower lip may be pursed up over upper.	6. Lower lip often everted. Mouth open. Drivelling common.
7. Tongue large and coarsely papillated if not fissured. Tongue frequently protruded and drawn back.	7. Tongue large, but not coarsely papillated or fissured. Tip of tongue thickened, and constantly protruding.
8. Skin smooth in infancy, but furfuraceous later; not redundant or "baggy."	8. Skin dry and scaly; forms folds here and there, being redundant and "baggy."
9. Hair "wiry," often "mouse color," but sometimes blonde. Downy growth common on forehead and cheeks.	9. Hair harsh, coarse, and scanty. Usually of darkish tint (Bourneville says brown); scalp often eczematous.
10. Thyroid gland palpable to greater or less extent.	10. Thyroid gland impalpable to most thorough examination.
11. No fatty tumors (pseudolipomata) in posterior triangle of neck.	11. Fatty tumors (pseudolipomata) frequently found in posterior triangle of neck, etc.
12. Long bones somewhat shorter than usual, but slender.	12. Long bones shortened and thickened, in some cases bowed.
13. Hands broad; thumb and little finger short, the latter often curved toward ring finger. Fingers taper at ends.	13. Hands broad, thick, and stumpy, with wrinkled skin. Fingers square at tips.
14. Feet large and flat. Fissure between great and next toe often seen.	14. Feet squat; skin redundant about ankles and dorsum of foot.
15. Abdomen often distended; occasional umbilical hernia; often inguinal hernia.	15. Abdomen very bulky and prominent with folds of skin; umbilical hernia common.
16. Expression more or less vivacious and mobile, observant and imitative.	16. Expression dull and immobile; unobservant and apathetic.

Deficient stature, flattened bridge of nose, with expanded alæ, late and irregular dentition, deferred closure of fontanelles and retarded puberty are similar in each variety.

Recently cataract has been described as frequently developing in these cases—an indication of a possible endocrine dysfunction.

Treatment.—This has been taken up in Chapter IV on Diseases of the Internal Secretions, or Endocrinopathies.

Mongolism.—The Mongolian or Kalmuc type of defectiveness is so-called because of the resemblance of the patient to the Mongolian race, particularly the slant of the eyes and the general facial expression.

[1] Mentally Deficient Children, London, 1910.

The etiology is obscure. The condition has been believed to be the result of some abnormal state of the mother which may be produced by injury or shock, but which in general is believed to be the result of a worn-out reproductive capacity, the mother being unable to bring the child to complete development in the uterus. Corresponding with this assumption the Mongol is generally the last child born in the family, and not infrequently comes from good stock. In an examination of 42 Mongol children Thursfield[1] failed to corroborate the theory that the Mongol is apt to be the last of a large family.



FIG. 463.—Mongolian type, aged eighteen years, apathetic idiot, semimute, speaks only a few words, enunciation imperfect. Learned to feed himself and is cleanly. Dwarfish, brachycephalic, forehead flat and wrinkled transversely. Eyes oblique, photophobia, chronic conjunctivitis. Tongue very large, filling mouth completely; deeply fissured, papillæ enlarged. Hands broad. Fingers short and thick. (Courtesy of Dr. Martin W. Barr.)

They were not either members of a large family or were they the last born. In several instances healthy children were born following the advent of a Mongol. Among the mothers were not only apparently healthy women but comparatively young ones (below thirty). There was no evidence to connect the occurrence of Mongolism with ill-health of the mother during pregnancy, nor did there appear to be evidences of syphilis or tuberculosis.

McLean[2] has reported twins, one of which was a Mongolian idiot.

[1] Mongolism, British Jour. of Children's Dis., January–March, 1921.

[2] Mongolian Idiocy in One of Twins, Jour. Am. Med. Assn., January 7, 1922.

This argues strongly against syphilis or exhausted reproductive powers as exclusive factors but, as the children were of opposite sexes, and therefore dizygotic, that is, from separate ova, Mendelian segregation could therefore have occurred and so this case makes it appear that the condition is truly hereditary, due to germ plasm determiners.

There are three prominent physical signs of this condition which are sufficient when present to make a diagnosis. They are the form of the skull and the peculiarities of the palpebral fissures and of the tongue. The skull is brachycephalic, diminished in its antero-posterior diameter, flattened on the face and occiput, but without recession of the frontal and supraoccipital regions, as in the microcephalic. The tongue is large, the circumvallate papillæ are hypertrophied, and there are marked irregular transverse fissures. This condition of the tongue is characteristic of this type, and is not found in any other variety. Thompson has suggested that the fissuration is dependent upon two factors: an extreme vulnerability of the mucous membrane, and the habit of sucking the tongue, commonly present in these children. The hands and feet are broad, clumsy and spatulate, while an incurving of the little finger has also been described as of frequent occurrence in these children. In addition to these quite characteristic symptoms the children are clumsy, joints loose, the skin rough, the abdomen protuberant and there is a tendency to chronic inflammatory conditions of the mucous surfaces. The circulation is generally poor, the blood picture corresponds to that of the normal child under ten years of age.[1] Congenital cardiac anomalies may be present, such as imperfect closure of the foramen ovale, vital resistance is very low, and these patients are quite apt to die relatively early in life from tuberculosis.

Mentally these children are usually at a very low grade of development, generally gravitating about four years of age. They may be less or they may possibly reach the seven-year limit, but rarely go beyond it. As a rule they are good-natured and easy to care for by those who are understanding and sympathetic.

A special form of cortical aplasia has been described as belonging to this condition.

Thyroid does no good in these conditions. It is therefore necessary to carefully separate them diagnostically from cretinism, a condition which resembles it very closely on casual observation. For differential diagnosis see Cretinism.

Hydrocephalic Types.—Various degrees of mental defect may be associated with hydrocephalus. Hydrocephalus may of course occur previous to birth, but is rarely congenital, as a child with any marked degree of hydrocephalus could not be born alive. The condition may be relatively acute, in which case it leads rapidly to death, but it may come to an arrest or be extremely slow in progress. In this latter group of cases we find patients sometimes who live to a fairly advanced age, although as a rule this disease terminates life before the patient has passed middle life and generally much earlier. The symptoms in

[1] Nadohy, G. and Weinberg, M.: The Blood Picture in Mongoloid Idiocy, Jour. Am. Med. Assn., vol. **77**, p. 163.

the slowly progressive cases are the symptoms of gradual obliteration of the mental faculties, and are undoubtedly dependent upon pressure effects. These symptoms are in general loss of intelligence, gradual loss of vision, hearing, the function of language, and gradually the sinking into a semistuporous condition, and death. The causes of hydrocephalus are probably numerous, syphilis, tuberculosis, brain tumor, and meningitis are among them.

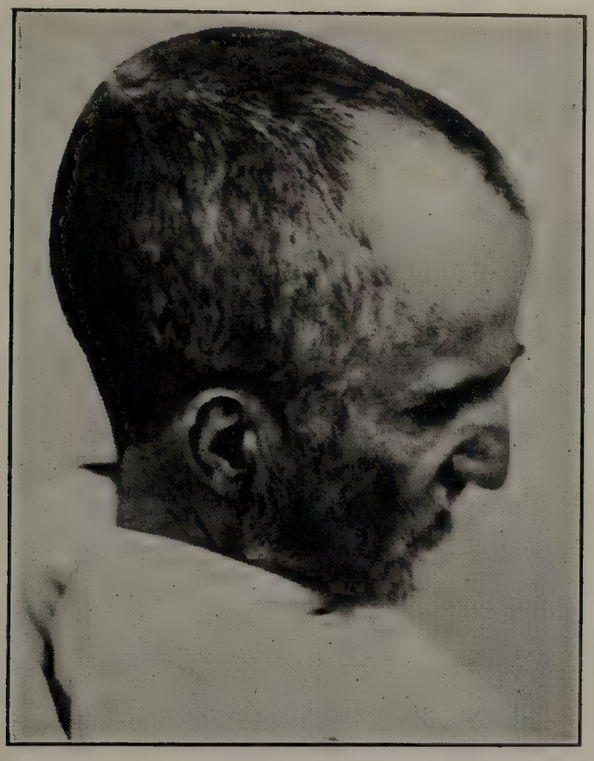


FIG. 464.—Hydrocephalus. Anteroposterior diameter 9¾ inches; transverse diameter 7⅞ inches. This man for many years made a living as a bell-ringer. Previous to death he became profoundly demented and his head so heavy that he could not lift it from the pillow. Autopsy showed the brain to be literally little else than a bag of fluid.

Microcephalic Types.—Mental defect associated with extreme smallness of the head. No definite rule can be laid down as to what size of head should be considered microcephalic. Ireland gives the general rule that heads below seventeen inches in circumference (431 millimeters) may be so considered. This rule is not absolute, and when it is considered that these small-headed varieties may probably be the result of various etiological factors it will be appreciated that the term microcephalic had better be used purely as a descriptive term rather than as a term to apply to a definite class of defectives even though that class be considered solely from a morphological standpoint.

The old theory that the condition was due to premature synotosis has long since been discarded, as has also the operation of craniectomy based upon that theory.

The characteristic condition of the microcephalic brain is its extreme smallness, more particularly pronounced in the temporosphenoidal, parietal and occipital regions. The posterior lobes of the cerebrum do not cover the cerebellum. The convolutions of the cerebrum are more simple in pattern than in the normal brain, and in addition there

may be localized ageneses with resulting microgyria. There may also be associated morbid processes such as encephalitis. The hypoplasia usually also involves the spinal cord.

The general appearance of these patients is quite characteristic. The conformation of the skull is "sugar loaf," or as it is technically termed "oxycephalic." This is characterized by a rapidly receding forehead with a flat occiput. Along with the receding forehead there is usually also a receding chin which gives a pointed aspect to the face, which, associated with a small stature gives a quite characteristic general appearance to these patients and has led to their being characterized as "bird-like" in appearance.

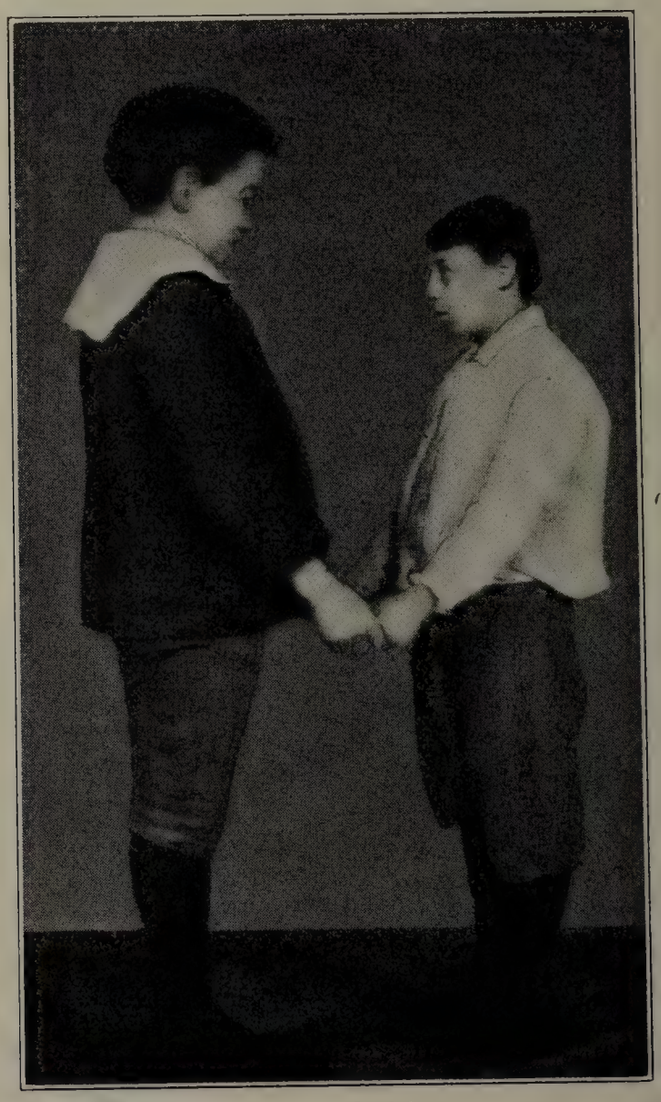


FIG. 465.—On left, high-grade imbecile, aged thirteen years; mentally, six years. On right, microcephalic imbecile, aged nine years; mentally, three years.

The mental development of these patients varies between wide limits, although the tendency is for them to belong to the lower grades of defect. They are usually well disposed, good-natured, affectionate, and not difficult to care for. They generally do not live to advanced years, but die at rather an early age.

Oxycephalus.[1]—Oxycephalus, acrocephalus, the steeple-shaped or sugar loaf skull while it is characteristic of microcephaly occurs

[1] Weygandt: Der Geisteszustand bei Turmschädel, Dtsch. Zeitschr. f. Nervenheilk., Bd. **68** and **69**.

without such marked reduction in skull size. Osteologically there is premature ossification of the sutures, shortening of the skull base, extraordinarily strong bony structure, and peculiarities of the inner surface which presents a strongly marked juga cerebralis and strongly



FIG. 466.—Erma. Front view.



FIG. 467.—Erma. Side view.



FIG. 468.—Mary. Front view.
Microcephalic twins, aged eight years.



FIG. 469.—Mary. Side view.
(Courtesy of Dr. A. C. Rogers, Faribault, Minnesota.)

marked impressions which can be observed during life by the roentgen rays.

The most important neurological signs are optic atrophy, frequent protrusion of the bulbi, and pressure on the olfactory nerve causing disturbances in the sense of smell even to complete destruction.

In most cases there are no psychic disturbances, indeed the mental endowment may be beyond the average. On a basis of eight cases Weygandt divides the disturbances into three groups: Feeble-mindedness of various degrees, degenerative traits, and psychotic symptoms. The degenerate types with average intelligence were unstable, spendthrifts, liars, swindlers. The third group showed, besides intellectual inferiority, symptoms of a profound psychosis with irritability, ill humor, delusions—a syndrome which could not be classified in any of the recognized groups.

Having in mind Meltzer's view concerning the involvement of the meninges together with the increased production of liquor and the analogy of these conditions to chondrodystrophy and micromegaly, Weygandt believes the conditions is due to a disturbance in the development of the meninges, dura and pia, arising from hereditary causes. The brain is, therefore, not to be regarded as the seat of the primary disturbances, and the epileptic appearances and psychic defects are to be referred to a secondary involvement.



Fig. 470.—Paralytic type. Athetoid movements of hands and arms.

Paralytic Types.—There are a large variety of cases in this group. The paralyses may involve any portion of the body or be of almost any extent. Monoplegias and diplegias are common, but localized palsies and hemiplegias are also not infrequent. They depend either upon lack of development of certain portions of the brain or more frequently upon injuries and sometimes new growths. Hemorrhage is the most frequent injury that produces the various palsies. This may occur as the result of prolonged labor or injury by forceps delivery, or may be the result of injury during the early months of life. The amount and the character of the mental defect varies within wide limits and no description which would apply to this whole class would be possible, principally because the class is not homogeneous.

Porencephalus.—Within this group of paralytic types one of the conditions which is found and which has been frequently described is porencephalus. This condition is consequent upon gross cerebral lesions such as a lack of cerebral substance resulting in a cyst connected with the ventricle—*true porencephalus,* or due to cysts not connecting with the ventricles and resulting from softening, hemorrhage, or inflammation—*false porencephalus.*

Traumatic Types.—Arrest of mental development may occur as the result of an injury to the brain during the developmental period. The most frequent type of injury producing this result is prolonged labor with instrumental delivery. Where the injury produces a lesion in the motor pathway with a resulting paralysis the patient is generally included within the paralytic group.

Epileptic Types.—Epilepsy occurring early in life is one of the causes of lack of mental development. Just how this result is brought about by the epilepsy is not altogether clear. In part it is due to the fact that the child has to be differently treated from normal children, is often deprived of the same educational advantages because of its disease, but apart from this there seems to be a direct relation between the lack of development and mental deterioration, and the epilepsy itself. This is probably dependent upon the fact that the epilepsy is a disease which strikes very deep, close to the foundation of the neurological mechanisms. Its manifestations are evidently, in part at least, dependent upon disturbances at the physico-chemical level. Disturbances which are as fundamental as this necessarily are difficult to deal with and also necessarily impair the superposed levels. In other words, the mental defect is the symptom at the psychological level of the same underlying factor that also expresses itself as the convulsion. (See Chapter on Epilepsy.)



FIG. 471.—Hemiplegic, aged eleven years. (Courtesy of Dr. A. C. Rogers, Faribault, Minnesota.)

Epileptic attacks are found in many of the other forms of defect, more particularly in those defects associated with gross cerebral lesions such as are found in the paralytic and traumatic types. It has generally been assumed that the localized lesion was the cause of the epileptic manifestations. While this may be so in certain cases, still in a general way it must be borne in mind that classical epilepsy has, presumably, a hereditary background dependent upon a defect in the germ plasm and that perhaps only those

children develop convulsions as a result of injury or localized cerebral lesions in whom hereditary conditions are favorable. At least the hereditary factor should not be neglected in the study of the patient simply because a localized lesion has been found. Perhaps the localized lesion could not have produced such a result without the hereditary factor—the spasmophilic tendency.

The epileptic type of defective, besides the symptoms of his defect, characteristically manifests the symptoms of the epileptic character and is therefore quite a difficult problem with which to deal.

Inflammatory Types.—This group includes those conditions resulting from inflammation of the meninges and of the brain, found most frequently as a result of acute infectious fevers such as pneumonia, typhoid, and the exanthemata. Local areas of meningitis or meningo-encephalitis are not infrequent in connection with the specific fevers, and when extensive or severe and occurring early in life produce an arrest of development, to a certain degree, of the mental faculties together, not infrequently, with epilepsy. Strümpell's polioencephalitis superior belongs in this group.

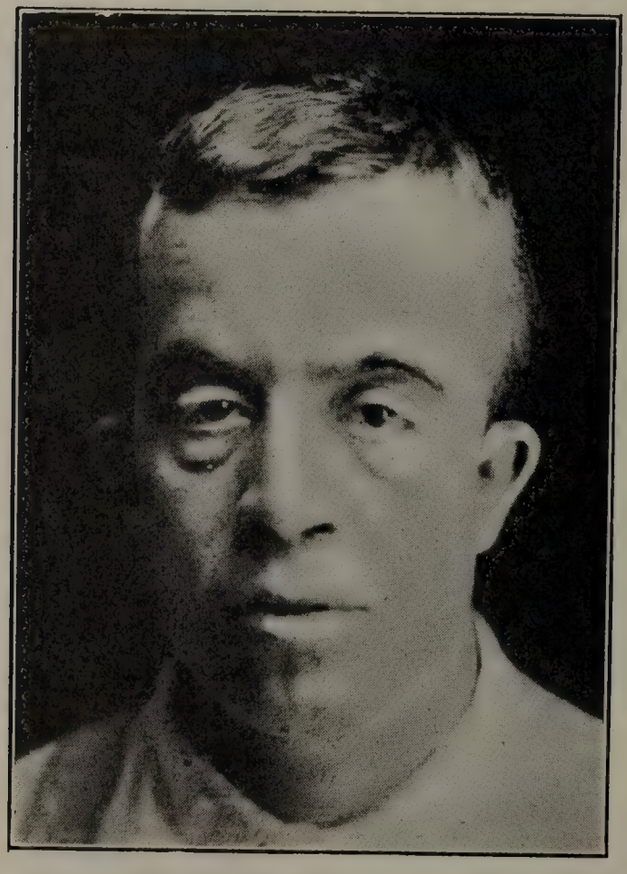


FIG. 472.—Sensorial type, imbecile by deprivation, middle grade. Male, aged about thirty years, deaf-mute. Taught himself to talk and to read and write in a fashion. Often cruel to children but devoted to animals. Appropriates things not his own. Very keen and observant. (Courtesy of Dr. Martin W. Barr.)



FIG. 473.—Sensorial type, imbecile by deprivation, middle grade. Boy, aged ten years, deaf-mute, no ears. (Courtesy of Dr. Martin W. Barr.)

Sensorial Types.—The defect in this group is the result of deprivation. Here there is no defect of the germ plasm or no defect in the structure

of the brain, but owing to injury or disease which has destroyed the patient's vision or hearing or both the child is cut off from communication with the outside world to such an extent that mental development is impaired thereby. Theoretically these patients are of course educable, but it is only very rarely that one is found with the capacity, the patience, and the ingenuity to develop such a child by educational methods. The cases of Helen Keller and Laura Bridgman stand out as illustrative of what can be done.

Milder grades of defect due to deprivation of the ordinary educational advantages occur on the same principle.

Syphilitic Types.—Syphilitic types of defect have always been known in that group of cases presenting distinct evidences of hereditary syphilis, particularly the Hutchinson teeth, linear scars about the mouth and nose, and keratitis. Until, however, the discovery of the *Treponema pallidum* and the elaboration of the Wassermann reaction there was no comprehension of the frequency with which syphilis entered into the etiology of the various types of mental defect. One of the best known authors, who just antedated this period, says that the number of cases of defectives due to syphilis is quite insignificant, probably not more than 1 or 2 per cent., and comments upon this in the face of the frequency of syphilitic disease in the fully developed brain and the frequency of so-called inherited syphilis. The Wassermann reaction was positive in 6 per cent of the children in institutions for the feeble-minded as compared with 5.2 per cent. in hospital children.[1]

There is one distinct type of disease which only recently has come to be recognized with any degree of accuracy which used to be classed among the defective states and which is due to syphilis, namely, *juvenile general paresis.* It is probable that still in many places this disease is not recognized, but is put down probably as some form of progressive defect. The number of juvenile paretics is not very large. (See Paresis.)

The application of the Wassermann test to the defectives as a class shows that not far from 20 per cent. of the patients taken indiscriminately show a positive Wassermann reaction. Of course it is quite another question as to just what the relationship is in these patients between the syphilis and the mental defect. In one case it may be that the syphilis has produced vascular disease and the defect is due to a vascular lesion. In another case it is the typical effect of a general paresis, or perhaps a meningoencephalitis, and still further probably syphilis has a decidedly deleterious effect upon development in ways that we do not at present understand, so that it may well be that certain of the types of defect which do not present characteristic syphilitic lesions may be due to subtle nutritional changes, which, in the last analysis, have their origin in syphilis. The great part that this disease plays in this class of cases is yearly becoming more and more evident.

[1] Solomon, H. C. and M. H.: Syphilis of the Innocent, Washington, D. C., 1922.

Inflammatory conditions of all sorts and their results are dependent upon this cause—meningitis, hemiplegia, porencephalus, hydrocephalus, which may be associated with epileptic convulsions.

Idiot-savants.—These are rare cases, who, although often deeply defective, still have some special ability wonderfully developed. It may be music, calculation, or memory for some special class of facts, etc. The calculators can name the answer to mathematical problems almost instantly; the musical prodigies often play well and may even improvise; one patient under the observation of one of the authors would instantly name the day of the week for any date for years back. Many of these patients have a capacity for mimicry and buffoonery, and from this class undoubtedly were recruited in the old days many of the court fools.

The psychology of these individuals is not understood and they themselves are quite unable to give any explanation of their special abilities. Their abilities, however, are really not so great as they appear. They appear exaggerated because they stand out upon a background of pronounced defect, also because they are unusual in the sense of not being the common possession of mankind. The calculators, for example, do very wonderful calculations in the way of adding up long series of figures very rapidly. There are, however, well-known devices for increasing the rapidity of the ordinary old-fashioned method of adding, and many of our experts at figures today could compete with some degree of success with these calculators. The patient, for example, who could name the day of the week for years back upon merely being given the date, and do it instantly, spent most of his time in studying calendars. It would not be surprising if almost anyone could accomplish such a feat if he spent any such commensurate degree of effort upon it.

Other descriptive terms are used to describe certain types of defectives, for instance in addition to the so-called Mongol or Kalmuc types there are described American Indian and Negroid types because of the obvious resemblances. Then again, for purposes of practical classification the lower grades of defectives are spoken of as either *apathetic* or *excitable*, and certain of these latter who keep up certain definite and characteristic movements almost continuously are described as *rhythmic idiots*. Other motor disturbances of course also occur. Aside from paralysis and epilepsy, probably one of the most common is athetosis.

Mild Grades of Defect.—A systematic examination of large numbers of children has disclosed the fact that a great number of the disorders of conduct and types of inefficiency which are manifested among them are dependent upon some degree of feeble-mindedness. Systematic examinations of school children, for example, have resulted in showing a not inconsiderable percentage of the general school population so far behind in their mental development as only to be described by a term indicating an inherent defect. These defects range all the way from well marked imbecility among the younger children up through the

grades of the so-called backward children; for the most part, the different grades of the moron, of the feeble-minded. The measuring rod which has been used for determining these conditions has been some form of intelligence rating tests, and the treatment which has been applied has been the segregation of these defective children from the general school population into classes and sometimes whole schools devoted particularly to them, thereby gaining both the advantage of the application of special educational efforts to these children and the relief of the normal child from the dragback to which he was subjected by having the defective in the same class with him.

Many of the mental defects, it must be borne in mind, are only relative affairs and are dependent upon general conditions of ill health, and poor nutrition, cardiac disease, chronic poisoning (alcohol, lead)

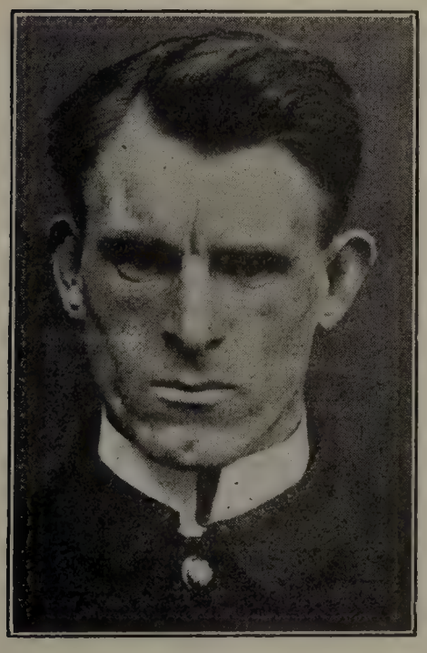


FIG. 474.—American Indian type. (Courtesy of Dr. Martin W. Barr.)



FIG. 475.—Negroid type. (Courtesy of Dr. Martin W. Barr.)

and infections (malaria, tuberculosis). An important group are due to adenoid vegetations in the posterior pharynx. Under such conditions of ill health development is impaired and does not proceed at a normal rate. With anemia, impaired digestion, and infected tonsils which produce a constant toxemia, the child cannot be expected to proceed in his development with normal rapidity.

In addition to such conditions as this it is found that the defect is often due to high grades of myopia which make it impossible for the child to learn, because he cannot see to read or even see the blackboard. In the same way deafness and other quite gross lesions have been found to account for many of these conditions.

In addition to these types there are the usually milder grades of defect—*infantilism*—dependent upon the dysfunction of the various endocrine glands. Here are found the thymic types, status thymico-

lymphaticus, hyper- and hypothyroidism, dysgenitalism (hypergenitalism), dyspituitarism (including dystrophia adiposogenitalis), dysadrenalism, and pluriglandular imbalances.

Chondrodystrophy and micromelia may be associated with mental defect, although many of these deformed individuals are bright and witty, for example the court fools of olden times.

There are many other conditions which may have some grade of mental defect associated such as cerebral tumors, spinal paralysis, Westphal-Strümpell pseudosclerosis, Friedreich's ataxia, facial hemidystrophy, etc.

It should be remembered, too, that the child may have a neurosis or a psychoneurosis, or even a psychosis and these conditions may produce conduct disorders which show a moral obliquity which, if they are not carefully investigated, may too easily be classified as due to defective conditions. Such psychic states are susceptible of psychoanalysis, and often of cure.



FIG. 476.—In center a moron, aged twenty-four years; mentally, ten years. At right moron, aged eleven years; mentally, eight years. At left, imbecile, aged nine years; mentally, six years.

Psychopathic Constitution.—There are many anomalies of character which, because normal or usual to the individual, cannot be said to properly constitute a psychosis, but because they lead to an inefficient type of adjustment of the individual to his environment, and because persons exhibiting these peculiarities often become actively disordered, may be considered as borderland conditions.

Something of the *hysteric* and *epileptic* characters has already been discussed. The general type of inefficiency of the *neurasthenic* and the *psychasthenic* character has been described, and attention has been called to the *unresistive* and the *post-traumatic* types with their intolerance of alcohol and fever. There also may be said to exist a *manic-depressive* type and lately attention has been directed to the "*shut-in*" type as the type par excellence found in those cases which develop dementia precox.

Besides these there are the "*cranks*," who with some pet scheme closely approach the paranoiac type and that host of *ill-balanced, eccentric* individuals who may be superficially brilliant but lack continuity of purpose and capacity for the continuous expenditure of effort in any one direction. Their life, to use the well chosen words of Régis, is one "long contradiction between the apparent wealth of means and poverty of results."

There are also the *pathological liar*, or *pseudologia phantastica*, and certain types of *swindlers*.

Constitutional anomalies of mood are seen, those who are always depressed—*psychopathic depression*—and the opposite state—*psychopathic exaltation*. Others never seem to be quite able to successfully cope with conditions; they are the failures of life, the cases of *constitutional inferiority*.

Conditions of *psychogenic depression* occur quite often in psychopathic individuals. Many weak characters who are led into crime develop a symptomatic depression when caught and sentenced. More pronounced defects of character are seen in the *criminal* classes, many of whom lack the ordinary moral inhibitions and are properly classed as *moral imbeciles*.

There are many psychogenic states that occur in psychopathic individuals—*deviates*. The so-called *prison psychoses* are types and come about as the patient's reaction to the difficulties in which he finds himself. They may be hysterical, catatonic, paranoid, or manic-depressive in type, according to the type of individual. They clear up when the stress is removed—pardon, expiration or commutation of sentence, etc.

These are the types to which Siemerling has given the name "*situation psychoses*" meaning that the psychosis is a result of the situation in which the patient finds himself. The imprisonment or perhaps the death sentence are intolerable facts which can neither be escaped nor permitted to enter consciousness and the various types of reaction —hysterical, catatonic, etc., are the methods employed to try and square with reality according to the make-up of the individual. Many "shell shock" neuroses develop in this group.

This whole group of reactions is found in psychopathic individuals, the types, of course, from which the criminal classes are recruited and which present to society some of the most difficult of its problems. The solution of these problems will only be in sight when the make-up of the individual is appreciated as a factor.

The question of *constitutional inferiority* involves many live issues. This term, like other clinical designations, does not apply to a well defined class, but in the main it may be said to cover two groups, those that are inferior, more especially from the intellectual angle, and those that are inferior, more particularly from the emotional angle. The latter group are naturally more important, at least from a sociological standpoint, because they include those ill-balanced individuals who so frequently run counter to the established order of things and therefore come within the purview of the criminal law for more or less serious offences. A discussion of this group is quite impossible in this place, both because of its great extent and its present lack of classification. It includes a considerable number of the juvenile delinquents, of the recidivist type of criminal, of the paupers and prostitutes, of the ne'er-do-wells, the black sheep of the family, and at the higher levels of erratic, half-genius, half-crazy persons with brilliant spots here and there, but without continuity, whose efficiency is materially impaired and who live often a more or less wandering existence. This type not infrequently suffer psychotic episodes when for any reason, such as conscription or imprisonment they are prevented from slipping from under responsibility or running away from reality. Often at the slightest stress such as a strong effort to make good, business reverses or loss of a loved object they develop paranoid symptoms, deep depression, or take to alcohol or drugs. Many of the inferior are less driven by their emotions and a great deal of the drudgery of the world's work is probably done by these dull and relatively stupid persons.

Theoretically these cases do not improve. The intellectually inferior can only be improved up to the point of their capacity to learn. The emotionally uncontrolled, however, tend spontaneously to a certain degree of clinical improvement when they have passed the fourth decade and the drive of the emotions begins to quiet.

Simulation.—Simulation is not an adequate diagnosis. It is necessary to know what lies back of the simulated symptoms and then it will be recognized as a reaction which may or may not be the best the individual is capable of under the circumstances. If it is the best then manifestly we are dealing with a defective personality, in most cases at least. If it is not the best then perhaps the better tendencies can be reinforced by encouragement on the one hand or, on the other, by the fear of punishment or by being made to actually suffer pain. If the punishment method is used it should be intelligently calculated to effect the ends sought along the same lines that a conditioned reflex is produced. If it is used as a species of self-indulgence on the part of those inflicting it it cannot be expected to produce good results.

In diagnosing simulation care must be used to avoid concluding that because a symptom is useful it is therefore simulated. All symptoms are useful and this criterion would indict phagocytosis. The diagnosis of malingering should never be made except it is clear that the symptom was consciously and intentionally created with the clear

conscious intent to deceive and even when such a diagnosis is made it may not be evident that the individual could have done better. Even if it is evident that the individual could have done better the object should not be punishment but to make the material (individual) available for the purposes in hand if possible—useful as a soldier, as a citizen—and to this end punishment should only be considered as a means.

ANOMALIES OF EROTIC EXPRESSION.

Quantitative Anomalies.—These are *frigidity* or lack of desire for sexual congress—*sexual anesthesia*—or *eroticism—sexual hyperesthesia.*

Qualitative Anomalies.—These are *inversions* and *perversions.* Inversion consists of a lack of harmony between the physical and the psychical sex and leads to *homosexuality* or desire for persons of the same sex. Various physical anomalies are often found in these persons. For example, the general conformation of the body, pilosity, etc., may indicate one sex, while the genitalia are of the other.

Sex inheritance is *alternative.* That is, both male and female characters are present in the germ and only one normally develops. Sometimes there seems to be an uncertainty as to which will develop and the result is a certain mixture which may take place either in the bodily or psychic sphere alone or in both.

The perversions are many and include the various anomalous means of gratifying the sexual appetite.

With respect both to inversion and perversion it must be remembered that in the young child the sexual instinct has not developed and later as it develops and comes into prominence it differentiates and tends to specialize by centering its aims in a special direction, *i. e.*, toward the opposite sex and normal coitus. The child, before this takes place is, to use a term of Freud's *polymorphous-perverse.* From this point on development taken place in the direction of the establishment of the primacy of the genital zone and the corresponding recession in relative importance of the other zones (mouth and anus more particularly but also the skin zone and in fact any other area or segment which may have come to have undue erotic significance). He may be developed in any direction by appropriate influences or he may stay in the undeveloped, infantile stage. The dominance of any other than the sex zone is the basic factor in many of the sexual perversions.

The most important of the perversions are:

Masturbation.—Masturbation is very frequent among psychopaths and very often a result rather than a cause of mental anomalies, though undoubtedly an important factor in some cases of acute psychosis. A transient period of onanism in infancy is probably normal and serves to focalize the sexual sensations on the genital zone and thus establish the hierarchy of the erogenous zones with the primacy of the genital zone.

Active Algolagnia (*Sadism*).—The gratification of the sexual feeling by the infliction or sight of pain—real or simulated. In the latter case

the sadism is *symbolic*. As the male is normally the more active and aggressive in the sexual relation, as might be expected, this anomaly is more frequently found in men.

Passive Algolagnia (*Masochism*).—The gratification of the sexual feeling by suffering pain—real or simulated. In the latter case it is *symbolic*. The female, being the more passive of the two sexes in the sexual relation, so an exaggeration of this passivity is more frequently found among women.

Homosexuality.—Every one passes through a period in development when he tends to fall in love or be especially interested in persons like himself, that is, of the same sex. This is the homosexual state and is the natural transition between self love and the state of object love in which the object is of the opposite sex. A homosexual residual of this stage remains as a normal component of every individual which, adequately sublimated is of great social value as the basic factor in friendships between persons of the same sex, but inadequately sublimated becomes the basis of homosexuality in its various perverse forms. Sexual desire for the same sex which seeks either to dominate or be dominated by the sexual object produces two types, the object homo-erotics and the subject homo-erotics.

Narcissism.—A form of auto-erotic sexuality in which a person is in love with himself—his own body or someone like himself. The love interest of the individual has never been adequately projected outside himself upon objects which are satisfying and so remains, including its libidinous component, attached to self.

Adequately sublimated the narcissistic libido functions to create a self that is worthy of love, it is the basic factor in the formulation of a personal ideal. Unsublimated it is the basis of selfishness and a factor in severe neuroses and in the psychoses, especially some of the more malignant such as paraphrenia.

Fetichism.—Sexual excitement and gratification by the sight, contact or possession of some object or part of the body. The object is usually some wearing apparel, such as shoes, handkerchief, petticoat, or a part of the body other than the sexual organs.

Bestiality.—Sexual relation with animals.

Exhibitionism.—Sexual gratification by exposing the genital organs.

Scoptophilia.—Pleasure derived from looking. The ambivalent opposite of exhibitionism (pleasure in being looked at). One who indulges the pleasure is a *voyeur*.

Necrophilia.—The desire to have sexual congress with a dead body.

Most of these conditions stand for what was normal at a certain stage in development but should have been left behind in progress; or else they are the result of aberrant development from these lower points when there has been a stagnation of the developmental process and so are included in this chapter. The higher psychic ramifications are discussed in the chapters on the neuroses, psychoneuroses, epilepsy, and certain psychoses, notably dementia precox and manic-

depressive psychosis, in all of which disturbances of psychosexual development are present.

This list of the anomalies of the sexual instinct as defined refers to the actual expression of this instinct in outward activity. All of these various manifestations, however, may appear in the fantasies of the patient, expressed symbolically and without any appreciable tendency to carry them into action. In fact, the analysis of practically any one would show a majority at least of these tendencies in symbolic expression in the unconscious. It can easily be understood why this should be so, because such tendencies as homosexuality, narcissism exhibitionism, etc., represent stages in normal development, the records of which are preserved in the unconscious. In their crude manifestations they belong to the symptoms of mental defect, in their higher symbolic expressions in the neuroses and psychoses they indicate the tendencies of the individual which have not been adequately socialized.

All of these instinctive tendencies may be adequately sublimated and socialized. The sadist may become a surgeon, a butcher, a military commander; masochistic types accomplish their best under the control, direction and domination of others; homosexuality is sublimated in very valuable forms of social activity—such as welfare work and ordinarily gets expression in social groups (clubs) of the same sex; narcissism is at the basis of personal ideals; exhibitionism is seen socialized in the public speaker and the actor. These instincts produce pathological results only when sublimation has failed.

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